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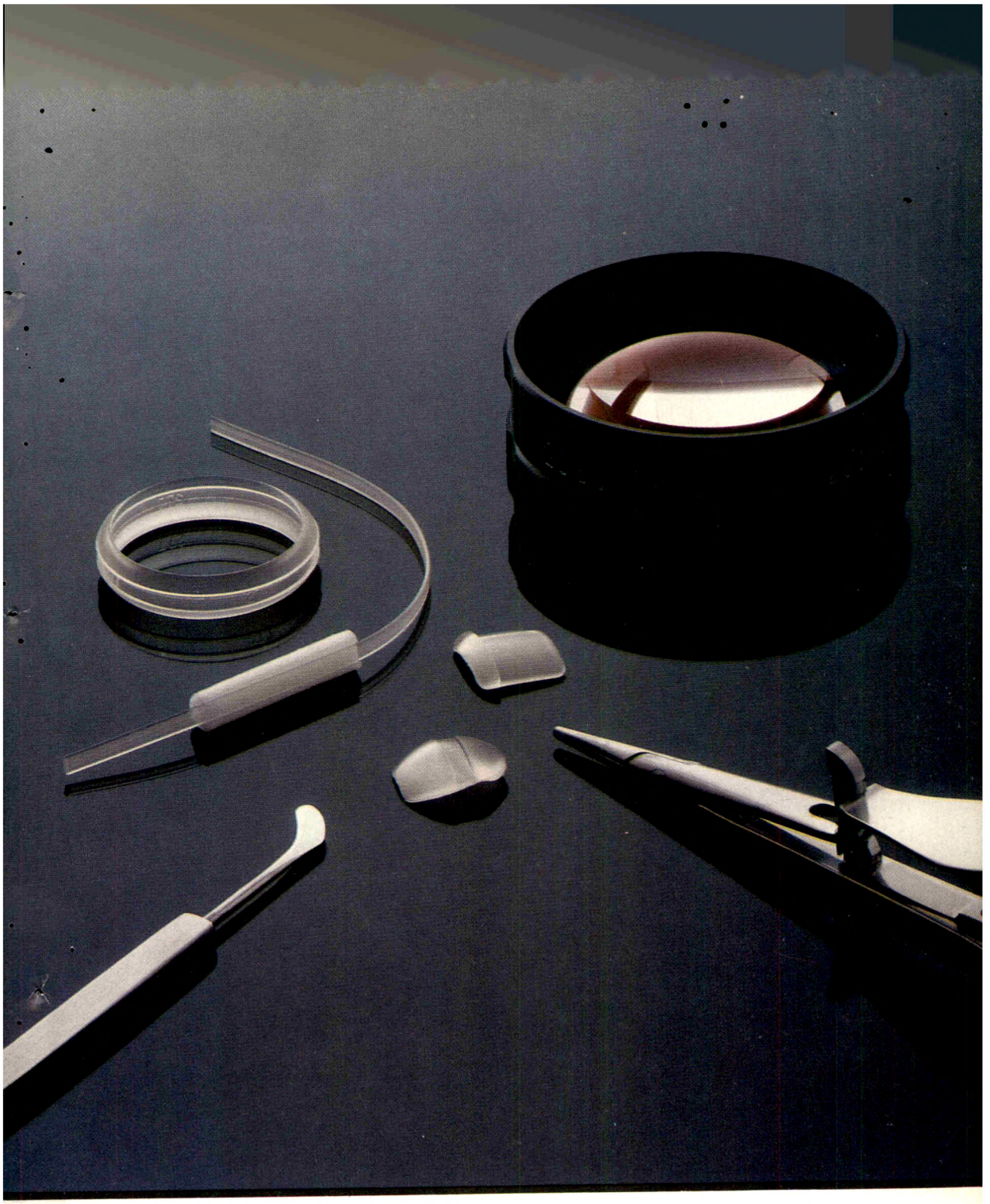
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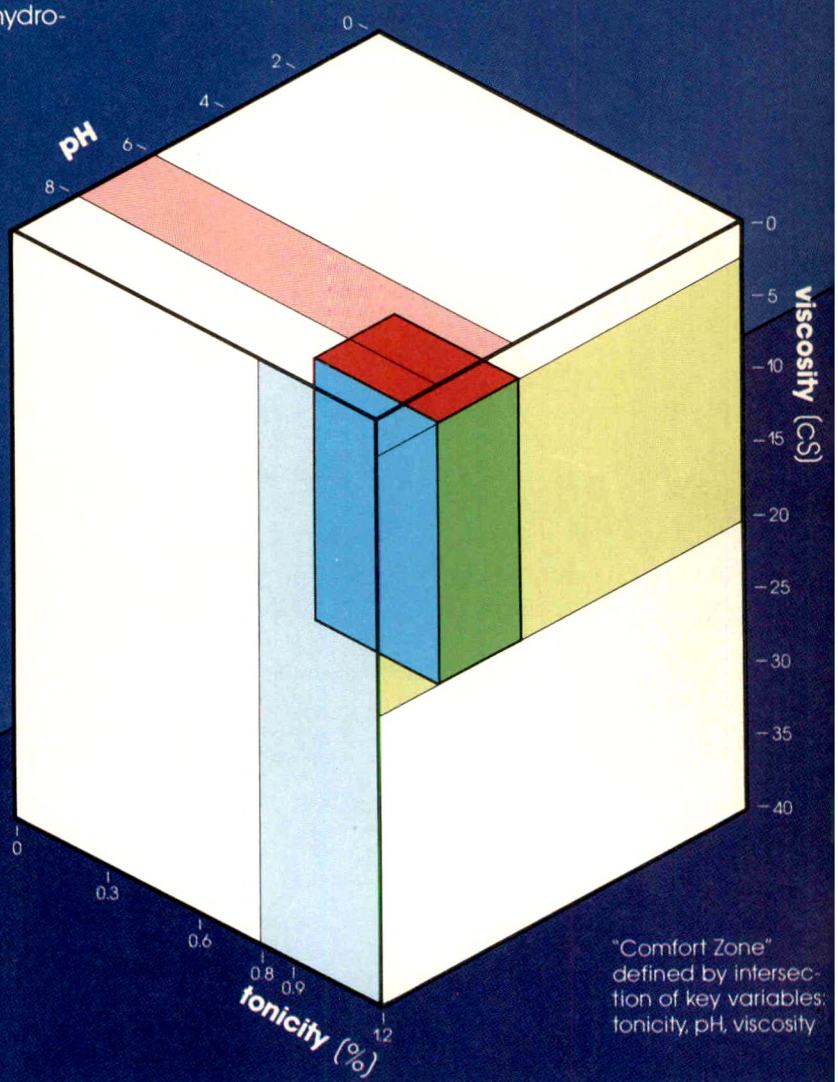
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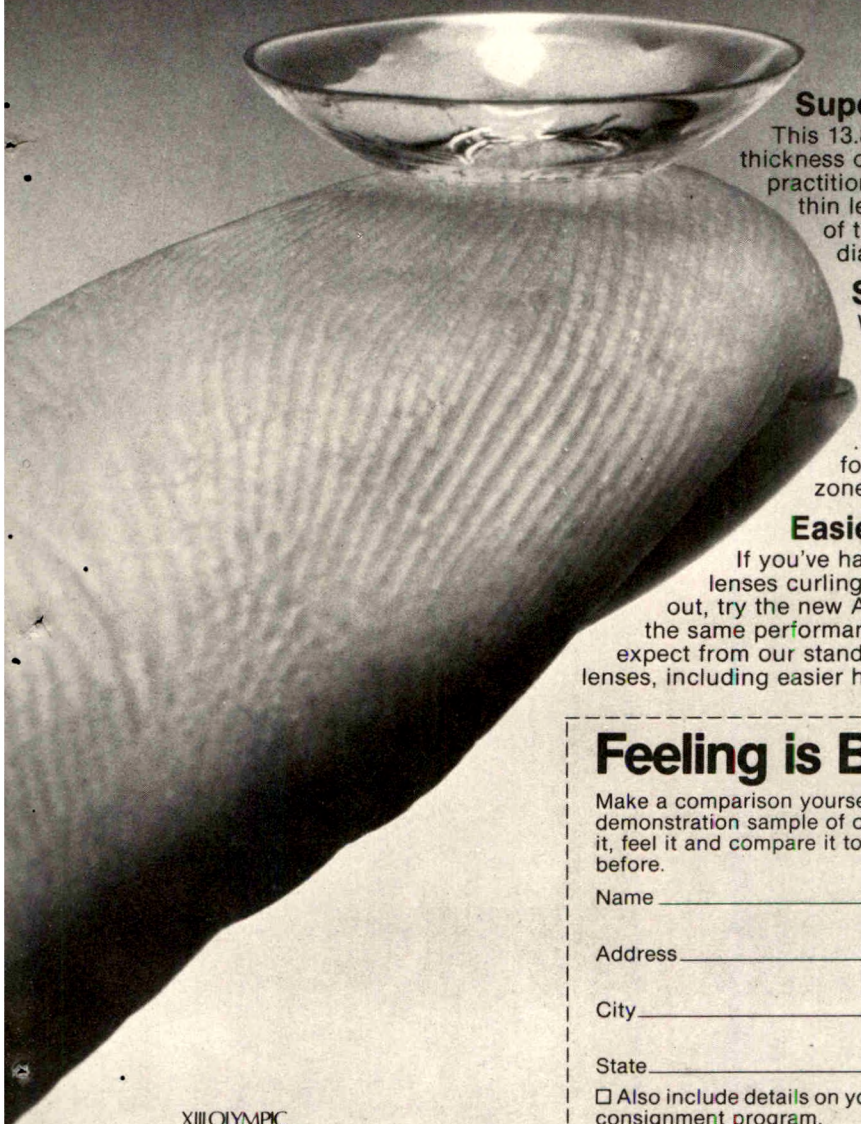
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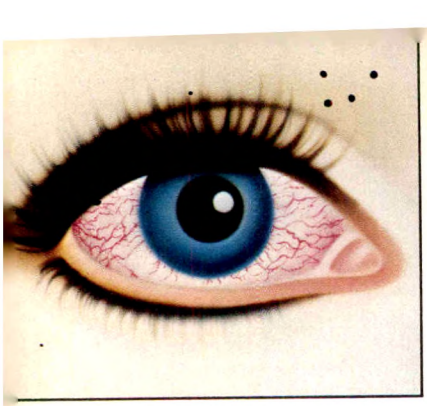
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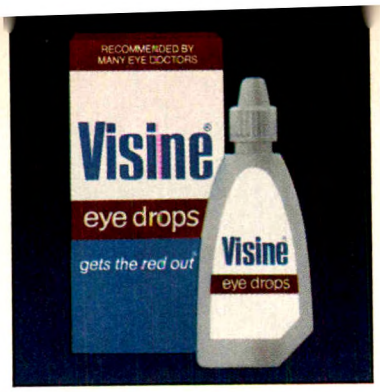
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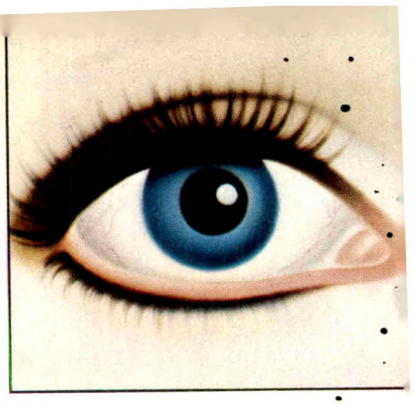
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Further confirmation of Visine's safety is provided by the low number of adverse reaction complaints received. In each of the last five years, such complaints have averaged fewer than 2 out of every million bottles sold.

Quickly provides effective decongestant action to bring lasting relief.

A double-blind test conducted among 100 patients indicated that 87 patients had hyperemia-free eyes in 5 minutes or less—59 of them in 1 minute or less. Other tests suggest a prolonged duration of effect unaccompanied by rebound hyperemia.
Double-blind evaluation of Visine—relief of symptoms of eye fatigue and irritction. Study #0152-8/72, February 1973.

Ideal isotonic formulation provides maximum comfort by maintaining normal hydration and thickness of cornea.

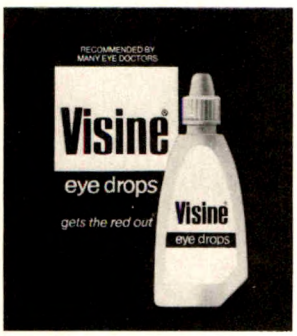
Visine ophthalmic solution—with a tonicity of about 290 mOsm/kg – matches that of 0.9 percent sodium chloride solution which is normal for tears. Thus Visine is formulated to avoid adversely affecting proper corneal thickness or causing discomfort.

Proper combination of both pH and buffering capacity assures comfortable instillation.

Visine ophthalmic solution has been formulated to have a pH of 6.2-6.5 and a low buffering capacity. This combination provides better stability of Tetrahydrozoline HCl and minimizes stinging and irritation by allowing the product to attain the normal pH of tears (7.4) very quickly after instillation into the eye.

©1979, Pfizer Inc.

Sine (Tetrahydrozoline HCl) ...an excellent vasoconstrictor among ophthalmic solutions



Dispensing Information

Composition: Tetrahydrozoline hydrochloride 0.05%; sodium chloride; boric acid; sodium borate; with benzalkonium chloride 0.01% and disodium ethylene diamine tetraacetate 0.1% added as preservatives.

Uses: For fast, lasting relief from minor irritation of eyes due to Dust-Smoke-Smog-Contact Lens adaptation (after removal of lenses).

Plant Allergies such as hay fever and rose fever. Minor eye irritation from or due to night driving, reading, T.V., sewing, swimming, wind and sun glare.

Refreshes, soothes and helps relieve redness caused by minor irritations.

Warning: Use only in the eyes. If relief is not obtained within 48 hours, or if irritation or redness persists or increases, discontinue use and consult your physician. Infection, pus, draining, foreign body in the eye, any mechanical, chemical, heat or other injury to the eye require the immediate attention of your physician. As with other eye medications, Visine should not be used by individuals with glaucoma or other serious eye diseases.

Parents: Keep this and all other medications out of the reach of children. Before using with children, consult your physician.

Directions: Place 1 or 2 drops in each eye two or three times a day as directed by physician. Visine is produced under sterile conditions.

Visine® professional samples on request

To receive free samples (twenty-four ¼-oz. Visine samples) for your patients, just fill out the coupon below: AJO-7-0

Name _____
Please print

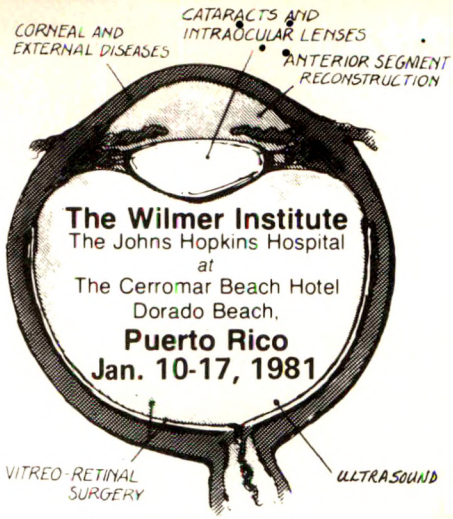
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Mail to: Visine Redemption Center
Leeming Division Pfizer Inc.



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Current Concepts in Ophthalmology

A course designed for the ophthalmologist in clinical practice with particular emphasis on an up-to-date review of the medical and surgical management of selected topics in corneal, external, and vitreo-retinal diseases.

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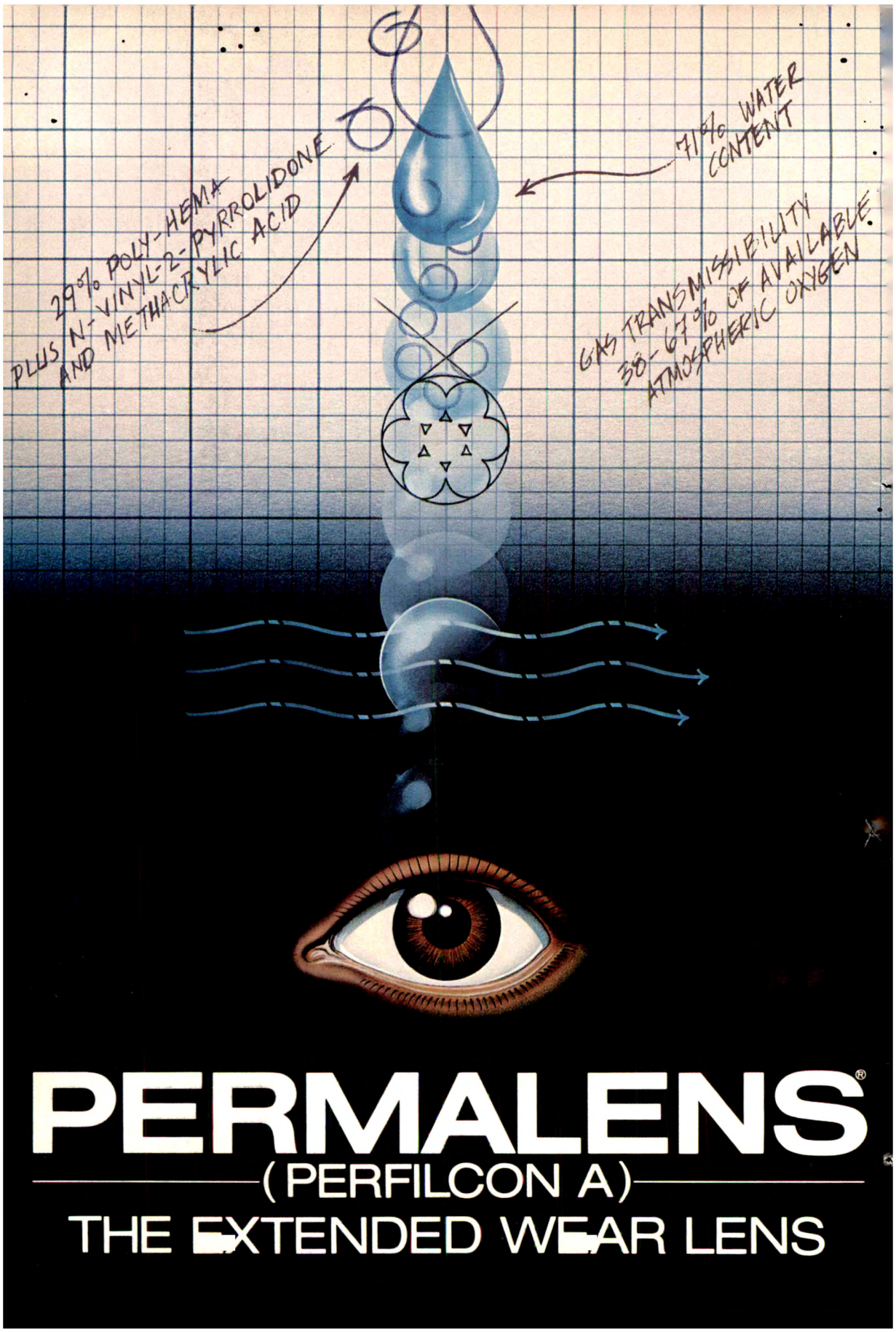
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PERMALENS®

(PERFILCON A)

THE EXTENDED WEAR LENS

Permalens® Contact Lenses: not just extended wear of a lens, but a lens **designed specifically for extended wear.**

That's why Permalens® Contact Lenses are rapidly changing the field of vision correction. Now your aphakic patients can again experience near-normal vision from the moment they wake up in the morning. Without the bother of daily insertion and removal.

And because of their advanced design, Permalens® Contact Lenses exhibit a physiological response comparable to daily-wear soft lenses. Even though your patients wear them 24 hours a day, for weeks at a time. The long-term acceptance of Permalens® Contact Lenses has been proven through extensive clinical trials.

This response is partly due to the Permalens® material, perfilcon A, which combines high water content, high oxygen permeability, and dimensional stability. These properties were designed into the lens to facilitate oxygen transfer directly through the lens, so the cornea can receive an oxygen supply adequate to maintain itself in a healthy state.

These properties also make Permalens® Contact Lenses more supple, and therefore comfortable on the patient's eyes. Patients have reported, in fact, they're the next best thing to wearing no lens at all.

Easy to fit, Permalens® Contact Lenses are available in a range of powers from +11.00 to +17.00 diopters. And with just four parameters, the lens fits most corneal sizes and curvatures.

Maintenance, too, is uncomplicated. Since Permalens® Contact Lenses usually need only be removed once a month, cleaning and disinfection are simplified. The preferred regimen is Preflex®/Flexsol®/Normol.® (Thermal regimens can also be used.) For re-hydration during lens wear, Clērz® or Adapettes® eye drops are available.

Patient comfort and convenience . . . clinically proven safety . . . easy fitting . . . simple maintenance. Good reasons why thousands of patients and practitioners alike have accepted Permalens® as the extended-wear lens.

Permalens®
(perfilcon A)

Hydrophilic Contact Lens
For Extended Wear
Post-Cataract (Aphakic) Use

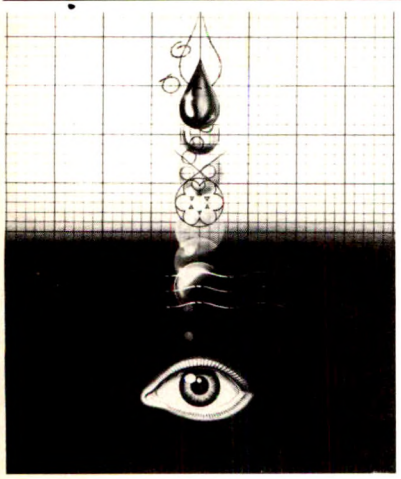
For full prescribing information, see next page.

Now available nation-wide. For details, contact your representative or call:

CooperVision® Inc.
OPTICS DIVISION

265 N. Whisman Road
Mountain View, CA 94043
(800) 227-8170
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Permalens®
(perfilcon A)

Hydrophilic Contact Lens For Extended Wear Post-Cataract (Aphakic) Use

DESCRIPTION

The **Permalens**® (perfilcon A) Hydrophilic Contact Lens is a hemispherical shell which covers the cornea and may cover a portion of the adjacent sclera. The lens material, perfilcon A, is a terpolymer of 2-hydroxyethyl methacrylate, N-vinyl-2-pyrrolidone, and methacrylic acid, with ethyleneglycol dimethacrylate as a crosslinking agent. The hydrated lens consists of 29% perfilcon A and 71% water by weight. The gas permeability of **Permalens**® Hydrophilic Contact Lenses, using the Fatt method[1] has been determined to be: 42.0×10^{-11} $cm^2 \cdot ml\ O_2/sec \cdot ml \cdot mm\ Hg$ at 36°C.

ACTIONS

When placed on the human cornea, the hydrated **Permalens**® Hydrophilic Contact Lens acts as a refracting medium to compensate spherical ametropias.

INDICATIONS AND USAGE

Aphakia

Permalens® (perfilcon A) Hydrophilic Contact Lenses are indicated for extended-wear aphakic vision correction in persons with non-diseased eyes.

Note: Supplemental spectacles may be required, in some conditions, to provide near vision or to compensate for uncorrected refractive astigmatism.

CONTRAINDICATIONS

Permalens® Hydrophilic Contact Lenses are contraindicated by the presence of any of the following conditions:

1. Acute and subacute inflammations of the anterior segment of the eye.
2. Any eye disease which affects the cornea or conjunctiva.
3. Insufficiency of lacrimal secretion.
4. Corneal hypoesthesia.
5. Any systemic disease which may affect the eye or be exaggerated by wearing contact lenses.

WARNINGS

Abrasions and Infections

If a lens becomes less comfortable than it was when it was first placed on the wearer's cornea, the lens should be removed and inspected for the presence of a foreign body, lens coating, deposits, or lens damage. If examination of the patient's eye reveals any eye abrasion, ulceration, irritation or infection, or any other abnormal eye condition is observed concurrently with lens wear, the lens should be removed immediately and a physician consulted.

Patients should not be fitted with Permalens® (perfilcon A) Hydrophilic Contact Lenses during the post-operative period until, in the opinion of the surgeon, the eye has healed completely.

Medications and Eye Drops

Permalens® Hydrophilic Contact Lenses must be stored in an appropriate solution when off the eyes. The solution used is dependent on the system selected for disinfection. When the lenses are disinfected with the thermal disinfection system, they may be stored in PERMASOL™ Rinsing and Storage Solution (sterile buffered balanced salt solution preserved with edetate disodium 0.1% and thimerosal 0.001%) or BOILnSOAK® Sterile Saline Solution (sterile buffered isotonic solution containing boric acid, sodium borate, sodium chloride 0.7%, preserved with Thimerosal (Lilly) 0.001% and edetate disodium 0.1%). When lenses are disinfected with the chemical disinfection system, they may be stored in FLEXSOL® Sterile Disinfection and Storage Solution (sterile buffered isotonic solution of sodium chloride, sodium borate, boric acid, polyvinylpyrrolidone, polyoxyethylene and polyoxypropylene, preserved with Thimerosal (Lilly) 0.001%, chlorhexidine 0.005% and edetate disodium 0.1%).

Only the above solutions and the following may be used with **Permalens**® Hydrophilic Contact Lenses:

1. PREFLEX® Sterile Cleaning Solution (sterile solution of a nonionic cleaner preserved with Thimerosal (Lilly) 0.004% and edetate disodium 0.2%).
2. NORMOL® Sterile Rinsing Solution (sterile buffered isotonic aqueous solution of sodium chloride, sodium borate and boric acid, preserved with Thimerosal (Lilly) 0.001%, edetate disodium 0.1% and chlorhexidine 0.005%).
3. CLERZ® Lubricating and Rewetting Eye Drops (sterile solution preserved with edetate disodium 0.1% and Thimerosal (Lilly) 0.001%).
4. ADAPETTES® Sterile Lubricating Solution (buffered isotonic aqueous solution containing Adsorbobase® (polyvinylpyrrolidone with other water-soluble polymers), Thimerosal (Lilly) 0.004%, and edetate disodium 0.1%).

No conventional (hard) contact lens solution should be used. Only those solutions specifically recommended by your eye-care practitioner should be used during wear.

WEARING RESTRICTIONS

Permalens® (perfilcon A) Hydrophilic Contact Lenses should not be worn while swimming or in the presence of noxious or irritating vapors.

Lens-Care Regimen

Patients must adhere to the recommended lens-care regimen of **Permalens**® Hydrophilic Contact Lenses. Failure to follow this procedure may result in the development of serious ocular infections which might result in corneal ulcers.

PRECAUTIONS

Storage

Permalens® Hydrophilic Contact Lenses must be stored in the appropriate storage solution: PERMASOL™ Rinsing and Storage Solution, BOILnSOAK® Sterile Saline Solution, or FLEXSOL® Sterile Disinfection and Storage Solution, depending on disinfecting methods used. If left exposed to air, the lenses will dehydrate, become brittle and break readily. If a lens dehydrates, it should be soaked in one of the following until it returns to a hydrated (soft) pliable state:

1. PERMASOL™ Rinsing and Storage Solution
2. BOILnSOAK® Sterile Saline Solution
3. FLEXSOL® Sterile Disinfection and Storage Solution

PERMA-case™ Contact Lens Storage and Carrying Case should be used for storing lenses.

Hygiene

Hands must be washed and rinsed thoroughly, and dried before handling the lenses. Cosmetics, lotions, soaps and creams must not come in contact with the lenses since eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until the spray has settled.

Cleaning and Disinfecting

Prior to wearing, **Permalens**® (perfilcon A) Hydrophilic Contact Lenses must be both cleaned and disinfected.

Cleaning: Monthly removal for cleaning is recommended. However, at the discretion of the eye-care practitioner, the lenses may be removed at a frequency adjusted to the needs of each patient. Each time the lenses are removed from the wearer's eyes, both surfaces of the lenses must be cleaned using several drops of PREFLEX® Sterile Cleaning Solution. Between regular cleaning periods, CLERZ® Lubricating and Rewetting Eye Drops or ADAPETTES® Sterile Lubricating Solution may be used to maintain lens hydration while being worn.

Routine Thermal Disinfection Method: Permalens® Hydrophilic Contact Lenses may be effectively disinfected using a low-heat disinfecting unit approved for use with hydrophilic contact lenses. After cleaning with PREFLEX® Sterile Cleaning Solution, rinse lenses with PERMASOL™ Rinsing and Storage Solution or BOILnSOAK® Sterile Saline Solution, and disinfect in a RINCON® low-heat Thermal Disinfection Unit or other approved unit. The lens storage containers must be emptied and filled with fresh PERMASOL™ Rinsing and Storage Solution or BOILnSOAK® Sterile Saline Solution just prior to disinfecting the lenses.

Alternate Thermal Disinfection Method: If a thermal unit is not available, the following disinfecting system can be used temporarily. After cleaning the lenses according to instructions, fill each chamber of the PERMA-case™ with PERMASOL™ or BOILnSOAK®. Bring a pan of water to a rolling boil. Remove the pan from heat. Place the sealed carrying case into hot water and allow to cool. When the water is cool, lenses have been disinfected.

NOTE: USE OF THE RINCON® THERMAL DISINFECTION UNIT SHOULD BE RESUMED AS SOON AS POSSIBLE.

Chemical Disinfection Method: Disinfection with PREFLEX® Sterile Cleaning Solution, FLEXSOL® Sterile Disinfection and Storage Solution and NORMOL® Sterile Rinsing Solution has been shown to be an effective disinfection system. Following each removal, **Permalens**® Hydrophilic Contact Lenses must be cleaned with PREFLEX® Sterile Cleaning Solution and rinsed with NORMOL® Sterile Rinsing Solution. Each time the lens is stored, the PERMA-case™ Contact Lens Storage and Carrying Case must be emptied and refilled with fresh FLEXSOL® Sterile Disinfection and Storage Solution. Fresh FLEXSOL® Sterile Disinfection and Storage Solution must also be used when disinfecting a lens.

WARNING: FLEXSOL® STERILE DISINFECTION AND STORAGE SOLUTION SHOULD *NEVER* BE USED WITH H

LENS EJECTION

Occasionally, a lens may become partially dehydrated during sleep. This may result in the lens being dislodged from the eye by lid action or rubbing the eyelids. Dislodged or routinely removed lenses should be thoroughly rehydrated in one of the following solutions:

1. PERMASOL™ Rinsing and Storage Solution
2. NORMOL® Sterile Rinsing Solution
3. BOILnSOAK® Sterile Saline Solution

After the lens returns to a soft, supple state it should be cleaned and disinfected prior to replacement on the eye.

ADVERSE REACTIONS

Serious corneal damage may result from wearing lenses which have been soaked in conventional hard contact lens solutions. Eye irritation may occur within a short time after putting on a lens stored in a solution of improper pH or tonicity. Removal of the lens will relieve the irritation.

Excessive tearing or redness, unusual eye secretions, reduced visual acuity, and photophobia are not normal. If these symptoms occur, the patient should be examined by an eye-care practitioner to determine the cause.

DOSAGE AND ADMINISTRATION

Fitting

Conventional methods of fitting contact lenses do not apply to **Permalens**® (perfilcon A) Hydrophilic Contact Lenses. For a detailed description of the fitting technique, refer to the **Permalens**® Hydrophilic Contact Lens Professional Fitting Guide, copies of which are available from Cooper Laboratories, Inc., 2 N. Whisman Road, Mountain View, California 94043.

WEARING SCHEDULE

Permalens® Hydrophilic Contact Lenses are indicated for extended-wear (24-hour) use, without a gradual daily increase wearing time required. Cleaning is recommended at monthly intervals. However, at the discretion of the eye-care practitioner the lenses may be removed at a frequency adjusted to the needs of each patient.

Lens Care and Handling

Care must be taken on the initial visit to ensure that the patient is supplied with all necessary accessory products and fully understands all care and handling instructions for the lenses. Regular post-fitting visits are necessary to assure patient health and compliance with instructions.

HOW SUPPLIED

Each lens is supplied sterile in a sealed glass vial containing a buffered isotonic salt solution. The vial is marked with the dioptric power, base curve, diameter, lot number, and expiration date of the lens.

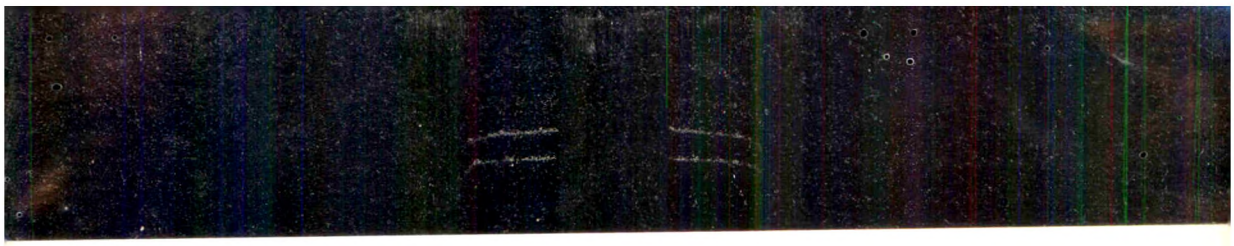
Accessories	
Patient Care Kit	Cat. #802
Wearer's Manual	Cat. #802
PERMA-case™ Contact Lens Storage and Carrying Case	Cat. #802
FLEXSOL® Sterile Disinfection and Storage Solution*	Cat. #901
NORMOL® Sterile Rinsing Solution*	Cat. #901
PREFLEX® Sterile Cleaning Solution*	Cat. #901
CLERZ® Lubricating and Rewetting Eye Drops	Cat. #801
PERMASOL™ Rinsing and Storage Solution	Cat. #801
RINCON® Thermal Disinfection Unit**	Cat. #902
BOILnSOAK® Sterile Saline Solution*	Cat. #901
ADAPETTES® Sterile Lubricating Solution	Cat. #901

CAUTION: Federal Law Prohibits Dispensing Without Prescription.

[1] Fatt I, Morris JA: A survey of gas-permeable contact lenses. *The Optician*. 27-36 (November 4, 1977)

*A product of Burton, Parsons & Company, Inc.
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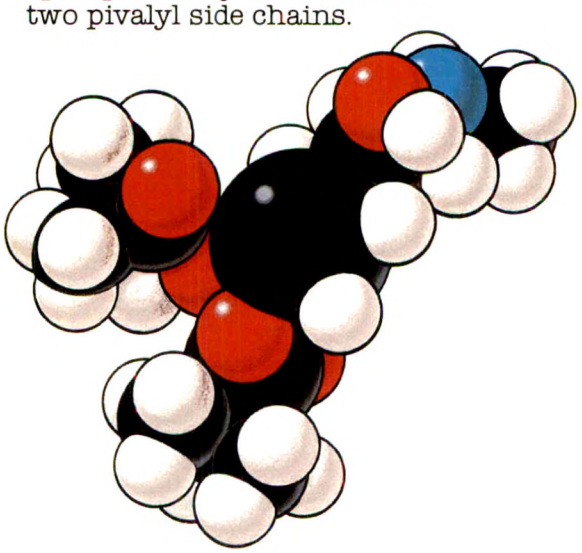
The future begins with a change in the right direction.

New Propine
(dipivefrin HCl)

The right direction to effective control of glaucoma with fewer side effects.

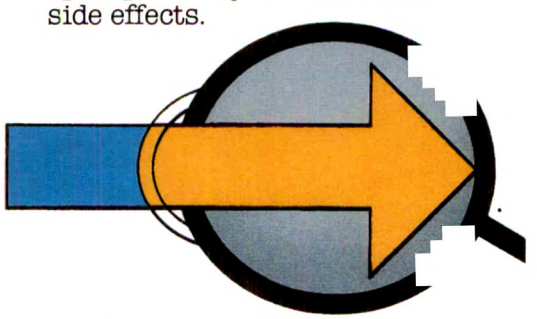
The prodrug Propine (dipivefrin HCl): the confidence of an established base with the new benefit of fewer side effects.

Prodrugs are agents which enter the body as one compound and then are altered into the active drug through enzymatic activity. Propine is a prodrug of epinephrine that differs from epinephrine by the addition of two pivalyl side chains.

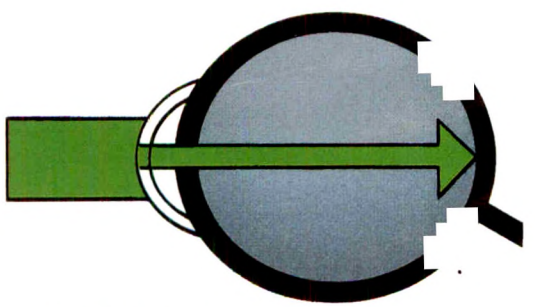


Propine molecule depicting addition of the two pivalyl side chains.

This addition creates a more lipophilic compound which is absorbed better into the eye — 17 times better penetration.[1] Once inside, it is converted to epinephrine by enzymes within the eye. This action makes Propine more potent than comparable concentrations of epinephrine, yet with fewer side effects.



Propine (dipivefrin HCl): better penetration with minimal side effects.



Epinephrine: less efficient penetration with greater chance of side effects.

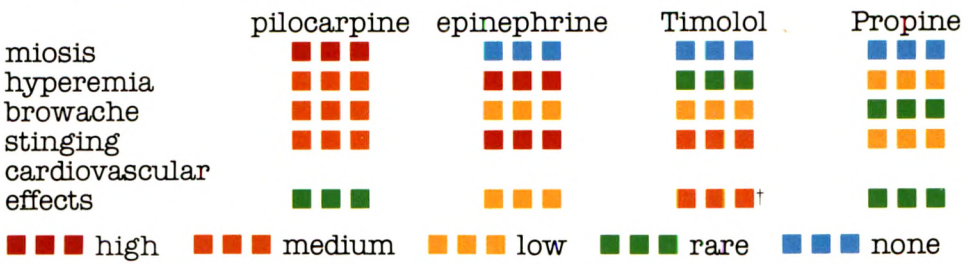
The prodrug for glaucoma therapy.

Concomitantly with Timolol or pilocarpine, or alone, Propine™ (dipivefrin HCl) provides:

Effective control of IOP

- Therapeutic response comparable to 2% pilocarpine q.i.d.
- Reduces mean pressure 20 to 24%
- Facilitates outflow and decreases aqueous production

Fewer side effects



†potential additive effect when used with systemic beta-blockers.

Convenient b.i.d. dosage

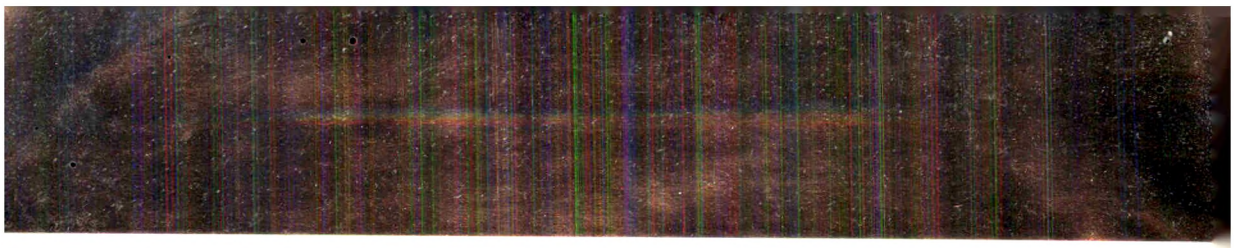
- Greater likelihood of patient compliance
- Easier concomitant use with Timolol b.i.d. dosage

Economy

- Nearly half the price of equivalent amounts of Timolol... an important consideration for fixed income patients.

Propine (dipivefrin HCl): the right direction for use

- Concomitantly with Timolol or pilocarpine
- Switch epinephrine and pilocarpine users to Propine
- Initial therapy: effective and safe choice for the new glaucoma patient.



Propine™
(dipivefrin HCl)

...the right direction in glaucoma therapy.

Propine™ (dipivefrin hydrochloride) sterile ophthalmic solution
Contains: Dipivefrin HCl* 0.1% with: benzalkonium chloride 0.004%; edetate disodium; mannitol; sodium chloride; sodium metabisulfite; hydrochloric acid to adjust pH and purified water. *Licensed under U.S. Patent Nos. 3,839,584 and 3,809,714. **CLINICAL PHARMACOLOGY:** Propine (dipivefrin HCl) is a member of a new class of drugs known as prodrugs. Prodrugs are usually not active in themselves and require biotransformation to the parent compound before therapeutic activity is seen. These modifications are undertaken to enhance absorption, decrease side effects and enhance stability and comfort, thus making the parent compound a more useful drug. Enhanced absorption makes the prodrug a more efficient delivery system for the parent drug because less drug will be needed to produce the desired therapeutic response. Propine is a prodrug of epinephrine formed by the diesterification of epinephrine and pivalic acid. The addition of pivaloyl groups to the epinephrine molecule enhances its lipophilic character and as a consequence, its penetration into the anterior chamber. Propine is converted to epinephrine inside the human eye by enzyme hydrolysis. The liberated epinephrine, an adrenergic agonist, appears to exert its action by decreasing aqueous production and by enhancing outflow facility. The Propine prodrug delivery system is a more efficient way of delivering the therapeutic effects of epinephrine, with fewer side effects than are associated with conventional epinephrine therapy. The onset of action with one drop of Propine occurs about 30 minutes after treatment, with maximum effect seen at about one hour. Using a prodrug means that less drug is needed for therapeutic effect since absorption is enhanced with the prodrug. Propine at 0.1% dipivefrin was judged less irritating than a 1% solution of epinephrine hydrochloride or bitartrate. In addition, only 8 of 455 patients (1.8%) treated with Propine reported discomfort due to photophobia, glare or light sensitivity. **INDICATIONS:** Propine™ (dipivefrin HCl) is indicated as initial therapy for the control of intraocular pressure in chronic open-angle glaucoma. Patients responding inadequately to other antiglaucoma therapy may respond to addition of Propine. In controlled and open label studies of glaucoma, Propine demonstrated a statistically significant intraocular pressure-lowering effect. Patients using Propine twice daily in studies with mean durations of 76-146 days experienced mean pressure reductions ranging from 20-24%. Therapeutic response to Propine twice daily is somewhat less than 2% epinephrine twice daily. Controlled studies showed statistically significant differences in lowering of intraocular pressure between Propine and 2% epinephrine. In controlled studies in patients with a history of epinephrine intolerance, only 3% of patients treated with Propine exhibited intolerance, while 55% of those treated with epinephrine again developed an intolerance. Therapeutic response to Propine twice daily therapy is comparable to 2% pilocarpine 4 times daily. In controlled clinical studies comparing Propine and 2% pilocarpine, there were no statistically significant differences in the maintenance of IOP levels for the two medications. Propine does not produce miosis or accommodative spasm which cholinergic agents are known to produce. The blurred vision and night blindness often associated with miotic agents are not present with Propine therapy. Patients with cataracts avoid the inability to see around lenticular opacities caused by constricted pupil. **CONTRAINDICATIONS:** Propine™ should not be used in patients with narrow angles since any dilation of the pupil may predispose the patient to an attack of angle-closure glaucoma. This product is contraindicated in patients who are hypersensitive to any of its components. **PRECAUTIONS: Aphakic Patients.** Macular edema has been shown to occur in up to 30% of aphakic patients treated with epinephrine. Discontinuation of epinephrine generally results in reversal of the maculopathy. **Pregnancy Category B.** Reproduction studies have been performed in rats and rabbits at daily oral doses up to 10 mg/kg body weight (5 mg/kg in teratogenicity studies), and have revealed no evidence of impaired fertility or harm to the fetus due to dipivephrine HCl. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed. **Nursing Mothers.** It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when Propine is administered to a nursing woman. **Usage in Children.** Clinical studies for safety and efficacy in children have not been done. **Animal Studies.** Rabbit studies indicated a dose-related incidence of meibomian gland retention cysts following topical administration of both dipivefrin hydrochloride and epinephrine. **ADVERSE REACTIONS: Cardiovascular Effects.** Tachycardia, arrhythmias and hypertension have been reported with ocular administration of epinephrine. **Local Effects.** Burning and stinging was the most frequent side effect reported (6%) with Propine alone. Injection was observed in 6.5% of the patients. Epinephrine therapy can lead to adrenochrome deposits in the conjunctiva and cornea. **DOSAGE AND ADMINISTRATION: Initial Glaucoma Therapy.** The usual dosage of Propine is one drop in the eye(s) every 12 hours. **Replacement with Propine.** When patients are being transferred to Propine from antiglaucoma agents other than epinephrine, on the first day continue the previous medication and add one drop of Propine in each eye every 12 hours. On the following day, discontinue the previously used antiglaucoma agent and continue with Propine. In transferring patients from conventional epinephrine therapy to Propine, simply discontinue the epinephrine medication and institute the Propine regimen. **Addition of Propine.** When patients on other antiglaucoma agents require additional therapy, add one drop of Propine every 12 hours. **Concomitant Therapy.** For difficult to control patients, the addition of Propine to other agents such as pilocarpine, carbachol, echothiophate iodide or acetazolamide has been shown to be effective. **Note:** Not for injection. **HOW SUPPLIED:** Propine (dipivefrin HCl) is supplied on prescription only as a 0.1% sterile solution in plastic dropper bottles in the following size: 10 ml — NDC 0023-0260-10.

References: 1. Mandell AI. et al. Dipivalyl epinephrine: A new prodrug in the treatment of glaucoma. Ophthalmology 1978 Mar;85(3):268-275.

Propine™...another advancement in research and development by the leader in ophthalmics

ALLERGAN Pharmaceuticals, Inc. Irvine, CA 92713

THE BLUE FIELD ENTOPTIC PHENOMENON. INSIGHT INTO SIGHT.



This is an artist's rendition of the blue field entoptic phenomenon, the perception of one's own leukocytes (flying corpuscles) flowing in the perifoveal capillaries.

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[1] Sinclar, S., Riva, E., Loebl, M.: Blue field entoptic phenomenon in cataract patients. Archives of Ophthalmology 97: 1092, 1979.

[2] Ibid.

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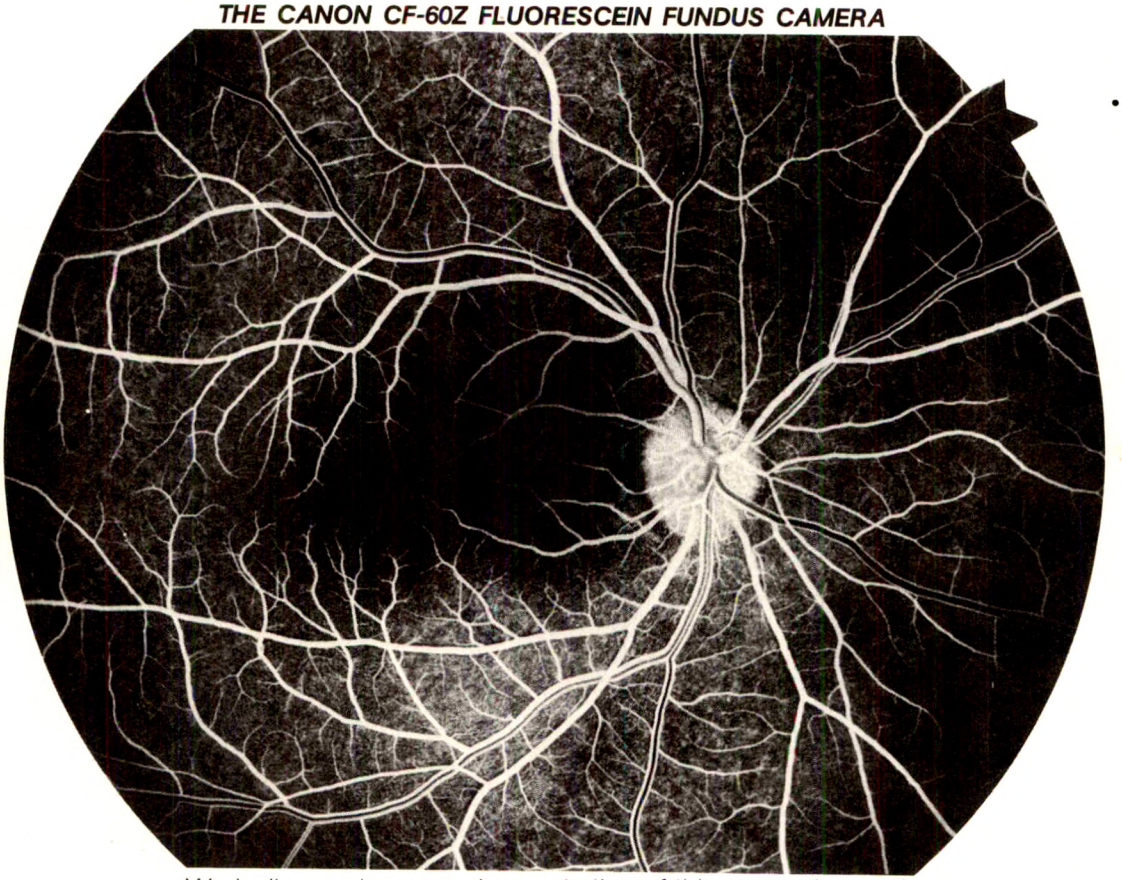
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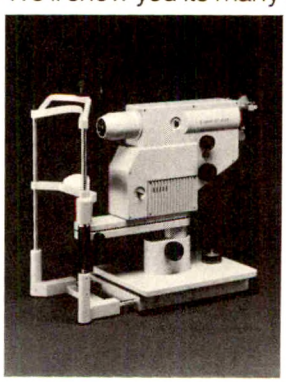
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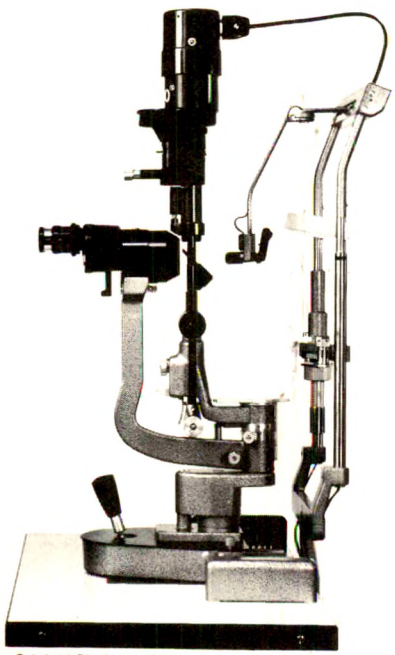
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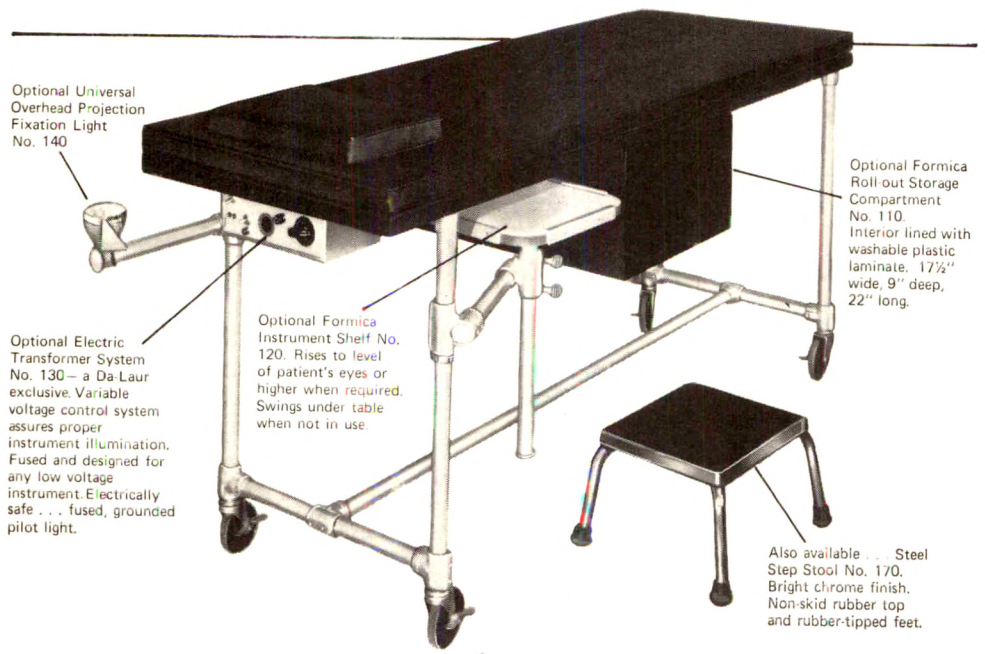
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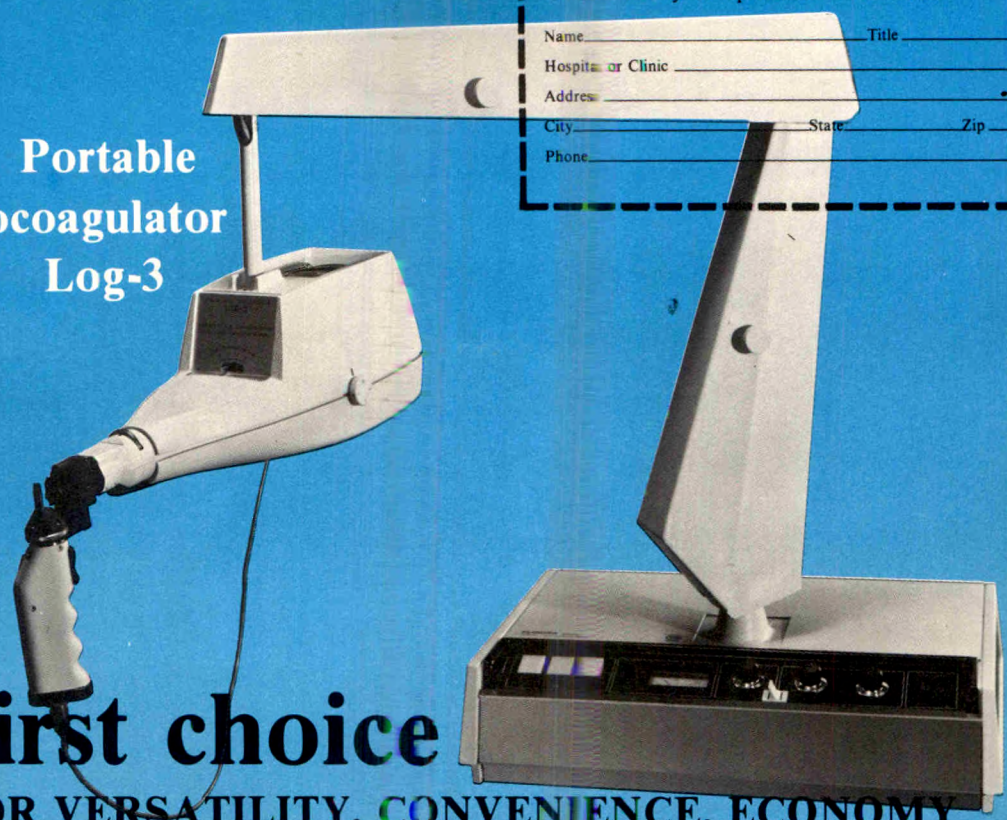
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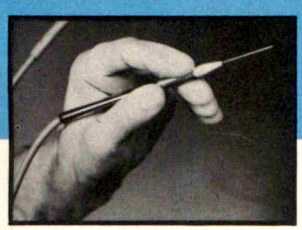
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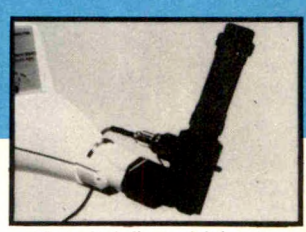
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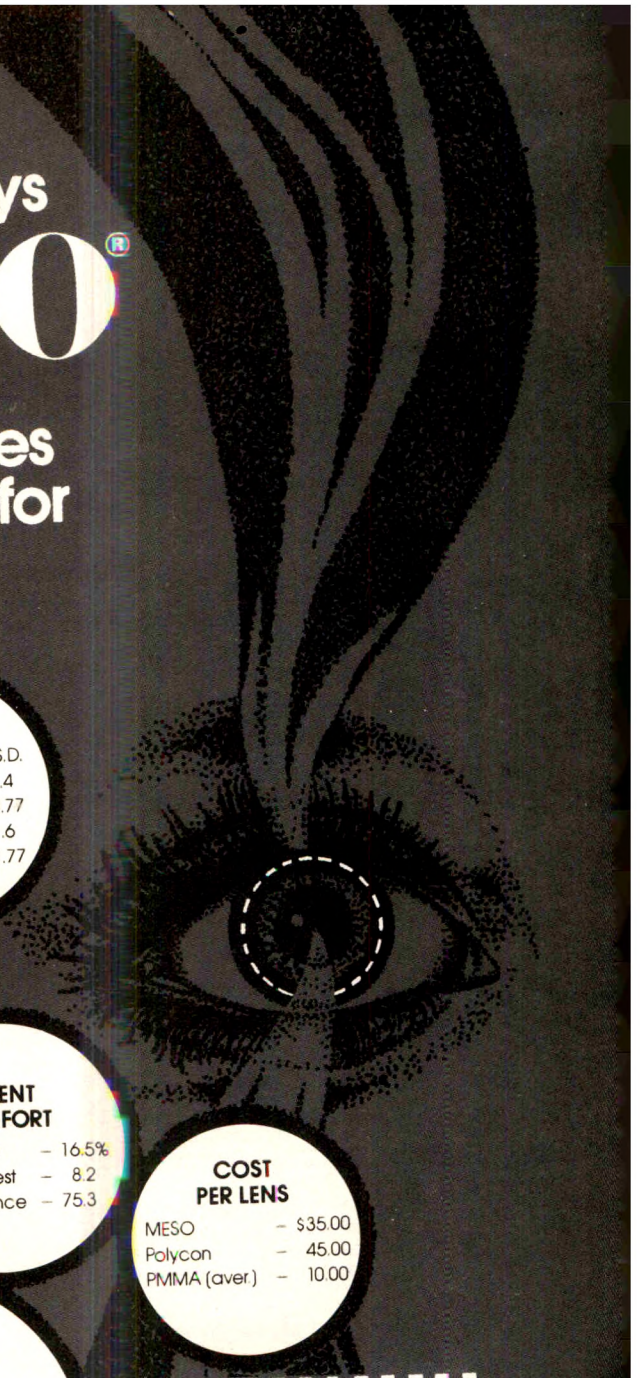
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Description: The MESO (cabufocon) contact lens is composed of the polymer cellulose acetate butyrate.

It is manufactured in a range of base curves (6.0-9.0mm) and diameters (6.0-12.5mm) to permit custom fitting of the cornea.

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- Water absorption: 1.8% by weight (ASTM D570)

The water content of the MESO (cabufocon) material when fully hydrated is 1.8% due to the polymer's free hydroxyl and carbonyl content.

Action: The MESO (cabufocon) contact lens, when placed on the human cornea, acts as a refracting medium to correct myopia, hyperopia, astigmatism and aphakia.

Indication: The MESO (cabufocon) contact lens is indicated for the correction of visual acuity in non-diseased eyes and aphakia.

Contra-indication: MESO (cabufocon) contact lenses are contraindicated in the presence of any of the following conditions:

1. Active conjunctivitis.
2. Active or chronic trachoma infection.
3. Active herpes infection.
4. Any condition which has led to extensive corneal neovascularization.
5. Corneal hypothesia except that resulting from aphakia.
6. Any situation in which the wearer is unable to maintain good lens care regimen.

Warnings: The MESO (cabufocon) contact lens must be stored only in Flex-Care or Flexsol solution.* No other storage solutions are recommended. Patients must adhere to the recommended daily care regimen. Failure to do so may result in ocular infection.

If the lens becomes more uncomfortable to the wearer than when first placed on the eye the lens should be removed, checked for dirt or scratches and cleaned. If this condition persists, or if the eye becomes red or painful, the lens should be removed immediately. If symptoms persist, your eye-care practitioner should be consulted.

The MESO (cabufocon) contact lens should be removed before sleeping or swimming and should not be worn in the presence of noxious fumes or vapors. If unusual blurring occurs, the lens should not be worn until the condition subsides.

Precautions:

Lens care – Storage: When the MESO (cabufocon) contact lens is removed from the eye, it should be stored only in Flex-Care or Flexsol sterile soaking solution after cleaning with Preflex* and rinsing with Flex-Care or Normol solution.* The lens should be completely covered with the storage solution. Fill the lens case receptacle till just below the rim.

Before placing the MESO (cabufocon) lens on the eye, rinse the lens with Flex-Care or Normol solution. Discard the old rinsing solution in the case, rinse the case with Flex-Care or Normol, and dry the case. Refill with fresh storage solution each time the lens is stored.*

The MESO (cabufocon) lens, unlike the hydrophilic soft lens, does not dry out or become brittle if exposed to air. If the lens does become dry, it should be soaked in storage solution for a minimum of four hours before reinsertion on the eye.

Fluorescein may be used with the lens.

Lens cleaning: Ordinary mucous and oily deposits are removed with daily cleaning of the lens with Preflex as described in your patient booklet.

Adverse reactions: As with any contact lens, corneal edema and corneal abrasions may occur if the lens is not properly fit, or if the wearing time is excessive.

Dose and administration: A gradual build-up of wearing time is recommended. A recommended schedule is as follows:

A) On the day of the initial fitting, two to four hours continuous wear.

B) Increase one hour each day for one week until eight continuous hours have been achieved.

C) If checkup is normal, wearing time is again increased one hour a day until full time (12 hours or more) is achieved.

Wearing schedule should be determined by the eye care practitioner.

Studies have not been performed to support safety for extended wear of this lens.

How supplied: Each lens is supplied in a sealed sterile packet. The packet is identified with the dioptric power, base curve, center thickness and manufacturer's lens number.

When the lens is removed from the packet, it should be stored in Flex-Care or Flexsol solution for at least four hours prior to dispensing to ensure maximum patient comfort.

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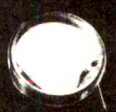
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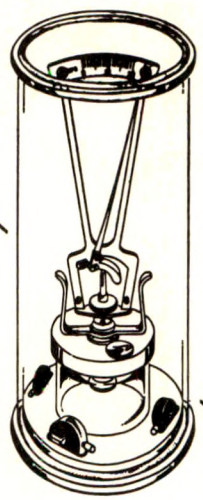
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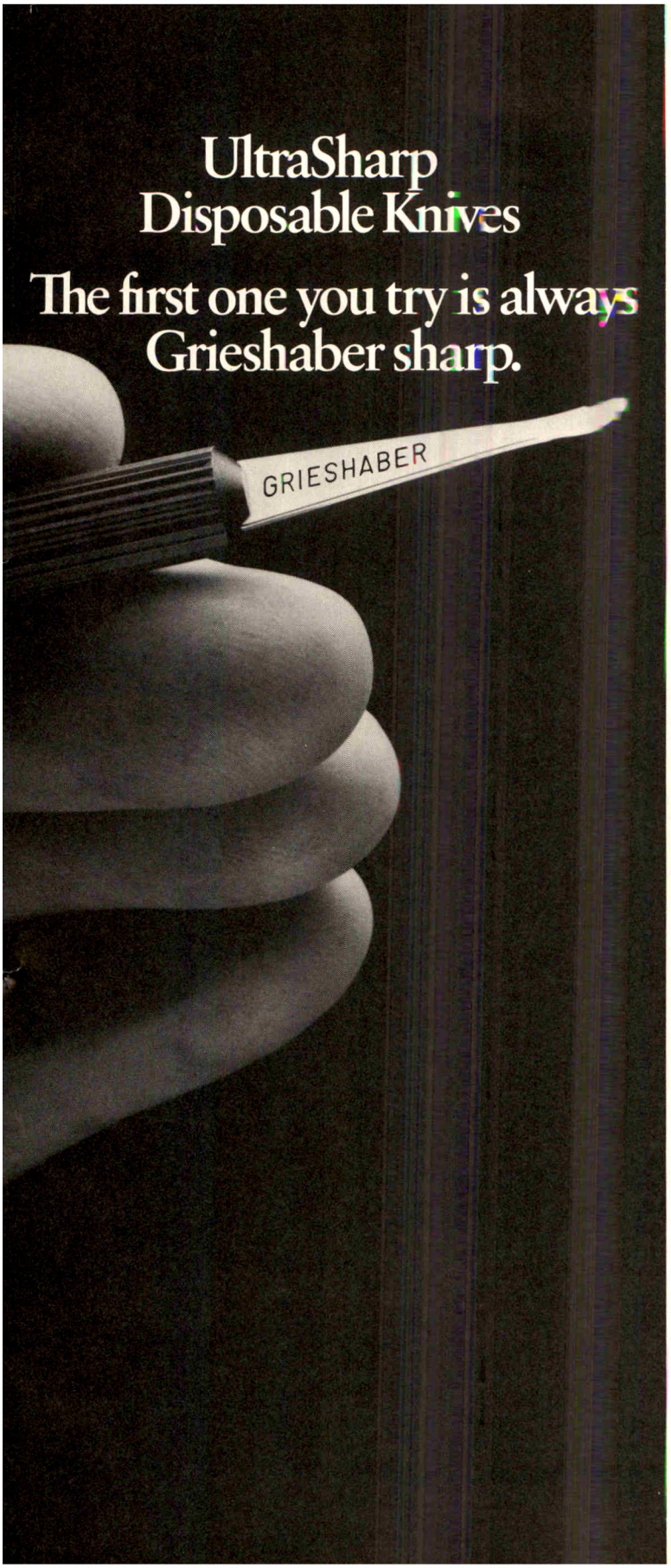
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Eleventh Eye Seminar

NEURO-OPHTHALMOLOGY FOR THE CLINICAL OPHTHALMOLOGIST

Sunday, October 5, 1980, 8:00 A.M.—5:00 P.M.

JONATHAN D. TROBE, M.D., *Gainesville, Florida*
Optic disc—pseudopapilledema, drusen, disc edema, papilledema
Diplopia

NORMAN J. SCHATZ, M.D., *Philadelphia, Pennsylvania*
Retrochiasm, lateral geniculate nucleus, occipital lobe
"How to count from 1-12", Painful Ophthalmolplegia

RONALD M. BURDE, M.D., *St. Louis, Missouri*
Afferent visual system—optic nerve to chiasm
Pupil , Pituitary Syndromes

7 hours AMA Category I credit, Registration: $80.00

Write: EYE SEMINARS
621 S. New Ballas Rd.—Suite 343 • St. Louis, Missouri 63141

Jack Hartstein, M.D., *Co-chairman,* Richard B. Oglesby, M.D., *Co-chairman*

Mark your calendar for the Spring Seminar also, Sunday, May 17, 1981:
REFRACTIVE SURGERY.

THE POSTGRADUATE INSTITUTE OF THE NEW YORK EYE AND EAR INFIRMARY ANNOUNCES THE SIXTEENTH ANNUAL REVIEW COURSE "BASIC SUBJECTS IN OPHTHALMOLOGY"

DECEMBER 1 through 6, 1980

SUBJECTS

PHYSIOLOGY • PHARMACOLOGY • OCULAR MOTILITY
NEURO OPHTHALMOLOGY • MEDICAL OPHTHALMOLOGY
DEVELOPMENTAL ANOMALIES • PEDIATRIC OPHTHALMOLOGY
ANATOMY and HISTOPATHOLOGY OF THE EYE
DISEASES of the CORNEA and CONJUNCTIVA
INTERPRETATIONS of POSTERIOR POLE LESIONS
PRINCIPLES of PHYSICAL OPTICS and REFRACTION

COURSE DIRECTOR: JAMES C. NEWTON, M.D.

FACULTY

Ronald Carr, M.D.
D. Jackson Coleman, M.D.
Andrew P. Ferry, M.D.
David S. Friendly, M.D.
John S. Hermann, M.D.
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This six-day intensive course is designed as a comprehensive review of the basic subjects in Ophthalmology and should be especially helpful to those physicians preparing for the American Board Certification Examinations and those having to fulfill requirements for continuing medical education. This course qualifies for 48 credits in Category 1 for the AMA-CME Physicians Recognition Award.

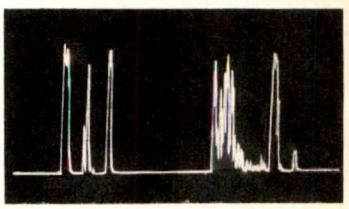
Tuition: $500.00 (Luncheons and Cocktail Party included)

For Application and further information, please write:
Jane Stark, Registrar • Postgraduate Institute
New York Eye and Ear Infirmary 310 East Fourteenth Street New York, New York 10003

There's more to a Biometric Ruler than just a number

For precise IOL power determination consider the New Sonometrics' Model-310





A-scan showing visual axis measurement.

Sonometrics, the proven leader in ophthalmic ultrasound equipment, introduces the new compact Digital Biometric Ruler, Model-310, for precise IOL power determination. A combination of quality, accuracy, and portability makes the DBR-310 an essential diagnostic tool at an affordable price.

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Programmable calculator and printer accessory calculates diopter power of intraocular lens using the advanced Binkhorst calculation module.



DBR-310 SBM focused transducer with internal fixation light.



Non-focused hand-held diognostic transducer.

Highly portable, the DBR-310 can be used in a variety of locations. The unit comes with a protected investment alternative which can be applied when you wish to upgrade to other full capability Sonometrics diagnostic instruments, including the clinically proven Ocuscan-400 and DBR-300 systems.

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AJO-75

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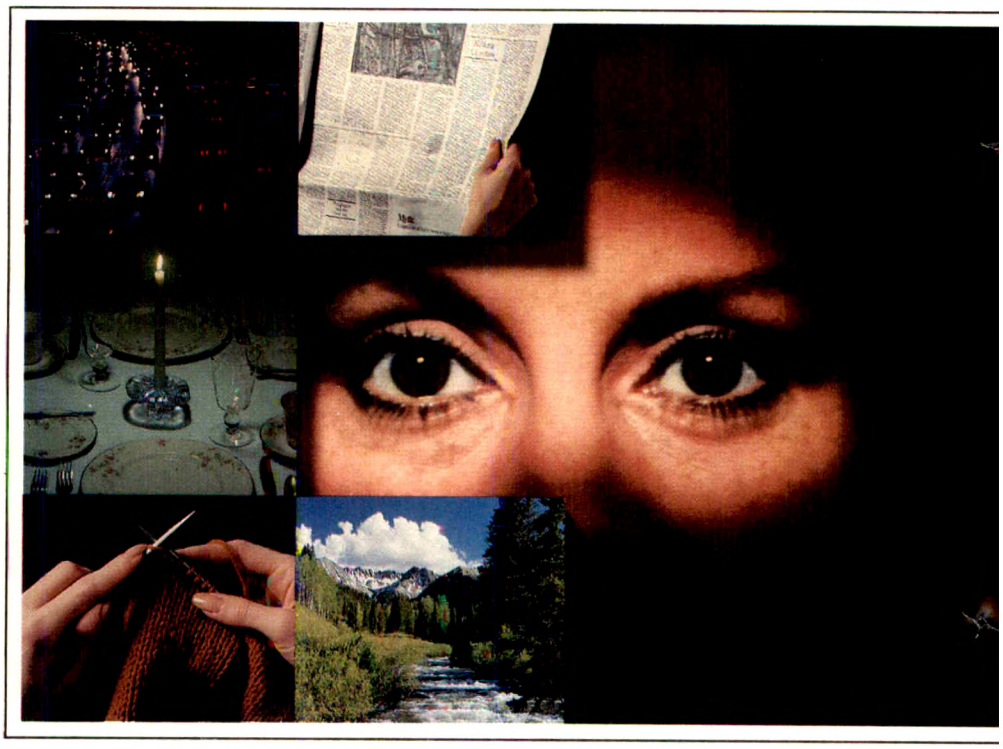
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In chronic open-angle glaucoma therapy

Two good reasons to switch patients to



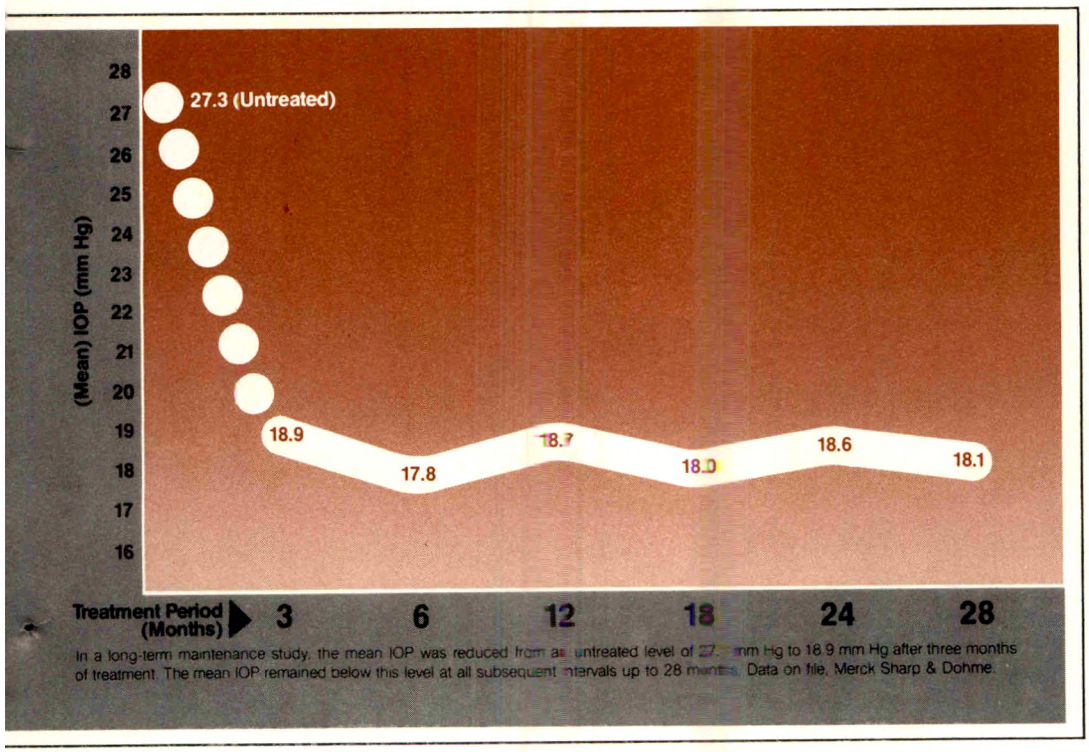
1. A better quality of life for many patients...

Patients on TIMOPTIC® (Timolol Maleate, MSD) generally do *not* experience the following therapy-limiting side effects:

change in pupil size/dimming of vision/night blindness*

TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product. Occasionally, mild ocular irritation has been reported.

Timoptic®
(TIMOLOL MALEATE | MSD)
OPHTHALMIC SOLUTION



In a long-term maintenance study, the mean IOP was reduced from an untreated level of 27.3 mm Hg to 18.9 mm Hg after three months of treatment. The mean IOP remained below this level at all subsequent intervals up to 28 months. Data on file, Merck Sharp & Dohme.

2. Efficacy maintained in long-term therapy...

- No tolerance was observed in 102 patients for 28 months
- TIMOPTIC was significantly more effective than pilocarpine or epinephrine in reducing elevated IOP*
- TIMOPTIC reduced IOP in 3 out of 4 patients*

*Based on controlled multiclinic studies, evaluating over 1000 patients.

MSD MERCK SHARP DOHME

For a brief summary of Prescribing Information, please see following page. Copyright © 1979 by Merck & Co., Inc.

NOW AVAILABLE
NEW
10-ml OCUMETER®



Timoptic®

(TIMOLOL MALEATE | MSD) OPHTHALMIC SOLUTION

a beta-adrenergic blocking agent in ophthalmic solution

a significant advance for the reduction of IOP in:

- chronic open-angle glaucoma
- aphakic glaucoma
- some patients with secondary glaucoma
- patients with elevated intraocular pressure at sufficient risk to require lowering of the ocular pressure
- some patients who respond inadequately to multiple antiglaucoma drug therapy

How to start patients on TIMOPTIC:
Usual starting dosage: one drop 0.25% TIMOPTIC in each eye twice a day.

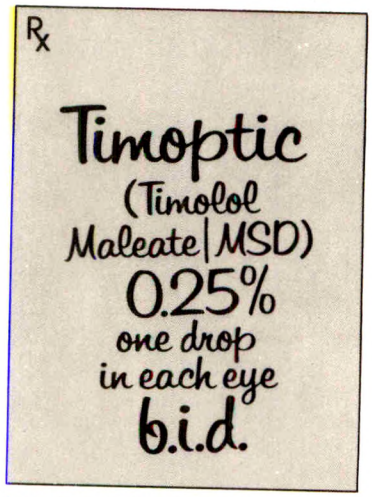
How to transfer from current therapy to TIMOPTIC:
1. On the first day: continue with the agent(s) already being used and add one drop 0.25% TIMOPTIC in each eye b.i.d.

2. On the second day: discontinue the previously used agent(s) and continue with TIMOPTIC in each eye b.i.d.

If clinical response is not adequate:
Dosage may be increased (from the 0.25% solution) by changing to one drop 0.5% TIMOPTIC twice a day in each eye. Dosages above one drop of 0.5% TIMOPTIC twice a day generally have not been shown to produce further reduction of IOP. If the intraocular pressure is maintained at satisfactory levels, the dosage schedule may be changed to one drop once a day in each eye.

Because of diurnal variations in intraocular pressure, satisfactory response to the once-a-day dose is best determined by measuring the intraocular pressure at different times during the day.

In patients with a history of severe cardiac disease, pulse rates should be checked.



CONTRAINDICATIONS: TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product.
PRECAUTIONS: Should be used with caution in patients with known contraindications to systemic use of beta-adrenergic receptor blocking agents. These include bronchial asthma; sinus bradycardia and greater than first-degree block; cardiogenic shock; right ventricular failure secondary to pulmonary hypertension; congestive heart failure; concomitant use with adrenergic-augmenting psychotropic drugs. In patients with a history of severe cardiac disease, pulse rates should be checked. Patients who are already receiving a beta-adrenergic blocking agent orally and who are given TIMOPTIC should be observed for a potential additive effect either on the intraocular pressure or on the known systemic effects of beta blockade.

Sufficient clinical data have not been obtained to show safety or efficacy in narrow-angle or angle-closure glaucoma.
Pregnancy: TIMOPTIC has not been studied in human pregnancy. The use of TIMOPTIC in pregnant women requires that the anticipated benefit be weighed against possible hazards.
Use in Children: Since clinical studies in children have not been done, TIMOPTIC is not recommended for use in children at this time.
ADVERSE REACTIONS: In clinical trials of up to three years duration prior to release in 1978, signs and symptoms of mild ocular irritation were reported occasionally. Local hypersensitivity reactions, including localized and generalized rash, have occurred rarely.

Slight reduction of the resting heart rate (mean reduction 2.9 beats/minute, standard deviation 10.2) has been observed in some patients. Rarely, episodes of acute bronchospasm have been reported in patients with bronchospastic disease (see PRECAUTIONS).
Note: TIMOPTIC has been used in patients with glaucoma wearing conventional (PMMA) hard contact lenses, and has generally been well tolerated. TIMOPTIC has not been studied in patients wearing lenses made with materials other than PMMA.
HOW SUPPLIED: TIMOPTIC Ophthalmic Solution, 0.25% and TIMOPTIC Ophthalmic Solution, 0.5%. Both are available in 5-ml and 10-ml plastic OCUMETER® Ophthalmic Dispensers with a controlled drop tip.
580 (DC7046009)

MSD
MERCK SHARP & DOHME

For more detailed information, consult your MSD representative and the full Prescribing Information.
Merck Sharp & Dohme, Division of Merck & Co., INC., West Point, Pa. 19486

Pure Unadulterated Tears



Daytime Lubricant Liquid

MUROCEL—no derivatives

(methylcellulose 1% 4000 cps sterile)

Murocel gives the feeling of normal eye comfort without the risk of stinging, irritation or sensitivity reactions inherent in the use of liquid alcohol derivatives. Because Murocel is purely methylcellulose, not an analog. When it comes to the eye, a tear should be pure and unadulterated.

Nighttime Lubricant Ointment

DUOLUBE—no additives

(white petrolatum and liquid petrolatum sterile)

Duolube also gives the feeling of normal eye comfort without the risk of irritation or sensitivity reactions inherent in the use of ointments containing preservatives or lanolin. Because Duolube is purely petrolatum, not additive-tainted. When it comes to the eye, a tear should be pure and unadulterated.

Create the natural fluid environment for the moisture-deficient eye

MURO® PHARMACAL LABORATORIES, I

OPHTHALMICS

(10-1711)

VIRA-A®
(vidarabine ophthalmic ointment), 3%



Description. Vira-A (vidarabine, adenine arabinoside, or Ara-A) is an antiviral drug for topical ophthalmic administration. The chemical name is 9-β-D-arabinofuranosyladenine. Each gram of the ophthalmic ointment contains 30 mg of vidarabine monohydrate equivalent to 28.11 mg of vidarabine in a sterile, inert, petrolatum base.

Clinical Pharmacology. Vira-A is a purine nucleoside obtained from fermentation cultures of *Streptomyces antibioticus*. Vira-A possesses *in vitro* and *in vivo* antiviral activity against Herpes simplex types 1 and 2, Varicella-Zoster, and Vaccinia viruses. Except for Rhabdovirus and Oncornavirus, Vira-A does not display *in vitro* antiviral activity against other RNA or DNA viruses, including Adenovirus.

The antiviral mechanism of action has not been established. Vira-A appears to interfere with the early steps of viral DNA synthesis. Vira-A is rapidly deaminated to arabinosylhypoxanthine (Ara-Hx), the principal metabolite. Ara-Hx also possesses *in vitro* antiviral activity but this activity is less than that of Vira-A. Because of the low solubility of Vira-A, trace amounts of both Vira-A and Ara-Hx can be detected in the aqueous humor only if there is an epithelial defect in the cornea. If the cornea is normal, only trace amounts of Ara-Hx can be recovered from the aqueous humor.

Systemic absorption of Vira-A should not be expected to occur following ocular administration and swallowing lacrimal secretions. In laboratory animals, Vira-A is rapidly deaminated in the gastrointestinal tract to Ara-Hx.

In contrast to topical idoxuridine, Vira-A demonstrated less cellular toxicity in the regenerating corneal epithelium of the rabbit.

Indications and Usage. Vira-A Ophthalmic Ointment, 3%, is indicated for the treatment of acute keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus types 1 and 2. Vira-A is also effective in superficial keratitis caused by Herpes simplex virus which has not responded to topical idoxuridine or when toxic or hypersensitivity reactions to idoxuridine have occurred. The effectiveness of Vira-A Ophthalmic Ointment, 3%, against stromal keratitis and uveitis due to Herpes simplex virus has not been established.

The clinical diagnosis of keratitis caused by Herpes simplex virus is usually established by the presence of typical dendritic or geographic lesions on slit-lamp examination. In controlled and uncontrolled clinical trials, an average of seven and nine days of continuous Vira-A Ophthalmic Ointment, 3%, therapy was required to achieve corneal re-epithelialization. In the controlled trials, 70 of 81 subjects (86%) re-epithelialized at the end of three weeks of therapy. In the uncontrolled trials, 101 of 142 subjects (71%) re-epithelialized at the end of three weeks. Seventy-five percent of the subjects in these uncontrolled trials had either not healed previously or had developed hypersensitivity to topical idoxuridine therapy.

The following topical antibiotics: gentamicin, erythromycin, chloramphenicol; or topical steroids: prednisolone or dexamethasone, have been administered concurrently with Vira-A Ophthalmic Ointment, 3%, without an increase in adverse reactions.

Normally, corticosteroids alone are contraindicated in Herpes simplex virus infections of the eye. If Vira-A Ophthalmic Ointment, 3%, is administered concurrently with topical corticosteroid therapy, corticosteroid-induced ocular side effects must be considered. These include corticosteroid-induced glaucoma or cataract formation and progression of a bacterial or viral infection.

Vira-A is not effective against RNA virus or adenoviral ocular infections. Vira-A is also not effective against bacterial, fungal, or chlamydial infections of the cornea or nonviral trophic ulcers.

Contraindication. Vira-A Ophthalmic Ointment, 3%, is contraindicated in patients who develop hypersensitivity reactions to it.

Warnings. ***Use in Pregnancy:*** Vira-A (vidarabine) parenterally is teratogenic in rats and rabbits. Ten percent Vira-A ointment applied to 10% of the body surface during organogenesis induced fetal abnormalities in rabbits. When 10% Vira-A ointment was applied to 2 to 3% of the body surface of rabbits, no fetal abnormalities were found. This dose greatly exceeds the total recommended ophthalmic dose in humans. The possibility of embryonic or fetal damage in pregnant women receiving Vira-A (vidarabine ophthalmic ointment), 3%, is remote. The topical ophthalmic dose is small, and the drug relatively insoluble. Its ocular penetration is very low. However, a safe dose for a human embryo or fetus has not been established. Consequently, Vira-A should be used only when clearly indicated.

It is not known whether Vira-A is excreted in human milk. As a general rule, because many drugs are excreted in human milk, nursing should not be undertaken while a patient is under treatment. However, excretion of vidarabine in breast milk is unlikely because Vira-A is rapidly deaminated in the gastrointestinal tract.

Mutagenic Potential: Results of *in vitro* experiments indicate that vidarabine can be incorporated into mammalian DNA and can induce mutation in mammalian cells (mouse L5178Y cell line). Thus far, *in vivo* studies have not been as conclusive, but there is some evidence (dominant lethal assay in mice) that vidarabine may be capable of producing mutagenic effects in male germ cells.

It has also been reported that vidarabine causes chromosome breaks and gaps when added to human leukocytes *in vitro*. While the significance of these effects in terms of mutagenicity is not fully understood, there is a well-known correlation between the ability of various agents to produce such effects and their ability to produce heritable genetic damage.

Oncogenic Potential: Chronic parenteral (IM) studies of vidarabine have been conducted in mice and rats.

In the mouse study, there was a statistically significant increase in liver tumor incidence among the vidarabine-treated females. In the same study, some vidarabine-treated male mice developed kidney neoplasia. No renal tumors were found in the vehicle-treated control mice or the vidarabine-treated female mice.

In the rat study, intestinal, testicular, and thyroid neoplasia occurred with greater frequency among the vidarabine-treated animals than in the vehicle-treated controls. The increases in thyroid adenoma incidence in the high-dose (50 mg/kg) males and the low-dose (30 mg/kg) females were statistically significant.

Hepatic megalocytosis, associated with vidarabine treatment, has been found in short- and long-term rodent (rat and mouse) studies. It is not clear whether or not this represents a preneoplastic change.

The recommended frequency and duration of administration should not be exceeded (see Dosage and Administration).

Precautions. The diagnosis of keratoconjunctivitis due to Herpes simplex virus should be established clinically prior to prescribing Vira-A Ophthalmic Ointment, 3%.

Patients should be forewarned that Vira-A Ophthalmic Ointment, 3%, like any ophthalmic ointment, may produce a temporary visual haze.

Although viral resistance to Vira-A has not been observed, this possibility may exist.

Adverse Reactions. Lacrimation, foreign-body sensation, conjunctival injection, burning, irritation, superficial punctate keratitis, pain, photophobia, punctal occlusion, and sensitivity have been reported with Vira-A Ophthalmic Ointment, 3%. The following have also been reported but appear disease-related: uveitis, stromal edema, secondary glaucoma, trophic defects, corneal vascularization, and hyphema.

Overdosage. Acute massive overdosage by oral ingestion of the ophthalmic ointment has not occurred. However, the rapid deamination to arabinosylhypoxanthine should preclude any difficulty. The oral LD50 for vidarabine is greater than 5,020 mg/kg in mice and rats. No untoward effects should result from ingestion of the entire contents of a tube.

Overdosage by ocular instillation is unlikely because any excess should be quickly expelled from the conjunctival sac. Too frequent administration should be avoided.

Dosage and Administration. Administer approximately one half inch of Vira-A Ophthalmic Ointment, 3%, into the lower conjunctival sac five times daily at three-hour intervals.

If there are no signs of improvement after 7 days, or complete re-epithelialization has not occurred by 21 days, other forms of therapy should be considered. Some severe cases may require longer treatment.

After re-epithelialization has occurred, treatment for an additional seven days at a reduced dosage (such as twice daily) is recommended in order to prevent recurrence.

How Supplied. N 0071-3677-07 (Stock 18-1677-139)

Vira-A Ophthalmic Ointment, 3%, is supplied sterile in ophthalmic ointment tubes of 3.5 g. The base is a 60:40 mixture of solid and liquid petrolatum.

UK

PD-JA-2320-1-P (5-78)

PARKE-DAVIS
Division of Warner-Lambert Company
Morris Plains, NJ 07950

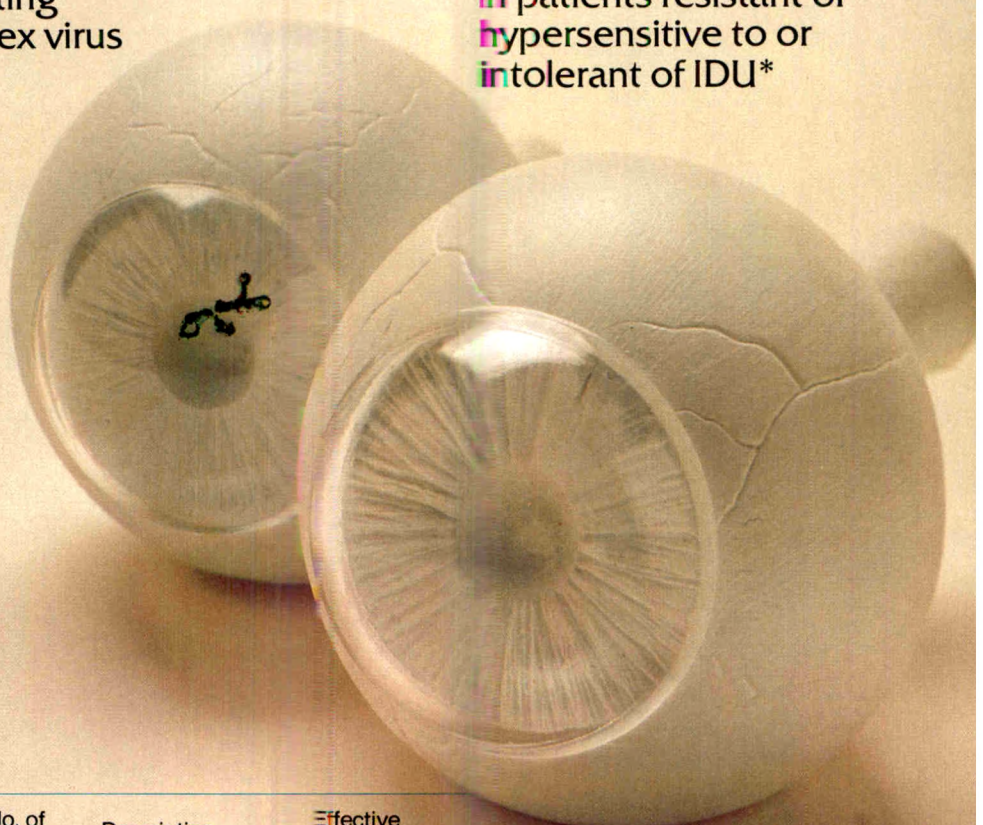
A LOGICAL FIRST CHOICE FOR HERPES SIMPLEX VIRUS KERATITIS

VIRA-A®

(vidarabine ophthalmic ointment), 3%

Not only as effective as IDU in treating Herpes simplex virus keratitis...

but is also effective in patients resistant or hypersensitive to or intolerant of IDU*



Clinical Evidence	No. of Subjects	Description	Effective Response
Vira-A Controlled Trials	81	Previously untreated	70 subjects (86%)
Vira-A Uncontrolled Trials	142	75% had been resistant or hypersensitive to IDU	101 subjects (71%)

NOTE: In the controlled and uncontrolled trials, 70 and 101 subjects, respectively, re-epithelialized at the end of three weeks on Vira-A.

*Data on file, Medical Department, Parke-Davis.

Vira-A is preservative-free.

Please see prescribing information on preceding page.

PARKE-DAVIS

Bausch & Lomb Scientific Update:

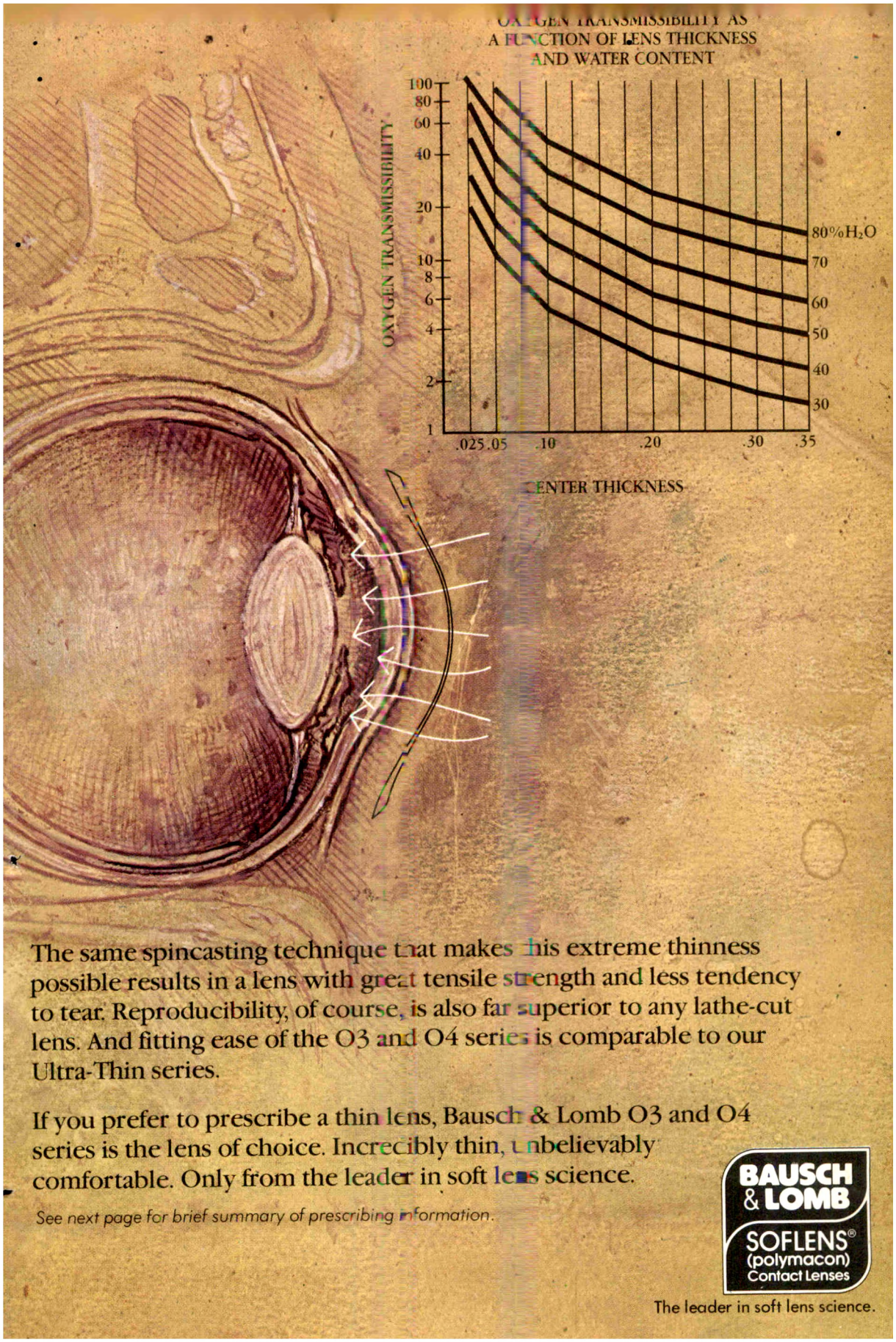
Introducing a lot less lens. And a lot more oxygen.

The new O3 and O4 minus series again demonstrate the merits of Bausch & Lomb's unique spincasting process. At 0.035mm center thickness, the O3 and O4 series are the thinnest soft lenses available in the U.S. The result: truly exceptional oxygen transmissibility and the most comfortable soft lens Bausch & Lomb has ever offered.



Only half the thickness of our Ultra-Thin series, the new O3 and O4 series have at least twice the oxygen transmissibility of any of our other series. Choose the 13.5mm diameter O3 or the 14.5mm diameter O4 in powers from −1.50 to −9.00D. You'll achieve full corneal coverage with the least possible lens mass.

Data on file at Bausch & Lomb.



The same spincasting technique that makes this extreme thinness possible results in a lens with great tensile strength and less tendency to tear. Reproducibility, of course, is also far superior to any lathe-cut lens. And fitting ease of the O3 and O4 series is comparable to our Ultra-Thin series.

If you prefer to prescribe a thin lens, Bausch & Lomb O3 and O4 series is the lens of choice. Incredibly thin, unbelievably comfortable. Only from the leader in soft lens science.

See next page for brief summary of prescribing information.

BAUSCH & LOMB
SOFLENS® (polymacon) Contact Lenses

The leader in soft lens science.

BAUSCH & LOMB SOFLENS® (polymacon) Contact Lenses

BRIEF SUMMARY (For full prescribing information see package insert.)

VISION CORRECTION USE

INDICATIONS: SOFLENS Contact Lenses are indicated for vision correction use in persons with non-diseased eyes and in aphakia. **CONTRAINDICATIONS:** SOFLENS Contact Lenses are contraindicated in the presence of any active untreated infection or abnormality of the anterior segment of the eye. **WARNINGS: Abrasions and Infections**—If a lens becomes less comfortable than it was when first placed on the wearer's cornea, the lens should be removed immediately and the wearer's eye and the lens examined for the possible presence of a foreign body. If any eye abrasion, ulceration, irritation or infection is present or any abnormal eye condition is observed concurrent with lens wear, the lens should be removed immediately and a physician consulted. **Aphakic Patients:** Aphakic patients should not be fitted with SOFLENS Contact Lenses during the postoperative period until, in the opinion of the surgeon, the eye has healed completely. **Lens Care and Handling:** Persons who require only vision correction and who would not, or could not adhere to the recommended daily care of SOFLENS Contact Lenses or who are unable to place and remove the lenses should not be provided with them. Failure to follow handling and cleaning instructions could lead to serious eye infections which might result in corneal ulcers. Malfunction and rusting of the metal interior of the BAUSCH & LOMB™ Disinfecting Unit I (ASEPTOR®) as well as discoloration and cracking of the lens case has been reported after varying periods of use. If such occurs, appropriate replacement is indicated to avoid interference with the disinfection procedure. Keep the BAUSCH & LOMB™ Disinfecting Unit I (ASEPTOR®) and II (ASEPTRON™) clean and dry at all times. **Medicants and Eye Drops:** When the lenses are used by persons requiring only vision correction no ophthalmic solutions or medicants should be used. No conventional (hard) contact lens solutions and eye drops, except BAUSCH & LOMB™ Lens Lubricant should be used by SOFLENS Contact Lens wearers prior to placement, or while the lens is in place, on the eye. Also, no solutions, including conventional (hard) contact lens solutions should be used. Only daily cleaner, lens lubricant, saline and the solution made from the SOFLENS™ Enzymatic Contact Lens Cleaning Tablets are to be used on a SOFLENS Contact Lens when the lens is off the eye. **Wearing Restrictions:** SOFLENS Contact Lenses when used only for vision correction should be removed before sleeping or swimming and in the presence of noxious and irritating vapors. Insufficient lacrimal secretions or hypoesthesia may be restricting factors to the wearing of soft contact lenses. **PRECAUTIONS: Storage**—SOFLENS Contact Lenses must be stored ONLY in saline solution or BAUSCH & LOMB™ Sterile Disinfecting Solution. If left exposed to air, the lenses will dehydrate, become brittle, and break readily. If a lens dehydrates, it should be resoaked in saline solution or BAUSCH & LOMB™ Sterile Disinfecting Solution until it returns to its soft, pliable state which may take as long as forty minutes. Saline solution must be used DAILY for cleaning, storing, and disinfection of lenses. The carrying case must be emptied and refilled with fresh saline solution just before disinfecting the lenses. **Lens Handling:** Hands must be washed, rinsed thoroughly, and dried with a lint-free towel before handling the lenses. Cosmetics, lotions, soaps, and creams must not come in contact with the lenses since eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until the hair spray has settled. A SOFLENS Contact Lens may be damaged by nicking or tearing if care is not exercised during placement on or removal from the eye, replacing or removing it from the carrying case or in the cleaning process. **Disinfecting:** Saline solution must be used DAILY for cleaning, storing, and disinfection of lenses. After removal from the eye, the SOFLENS Contact Lens must be rubbed gently with Daily Cleaner then rinsed with saline solution to remove mucus and film from the lens surface. The BAUSCH & LOMB™ Lens Carrying Case (LENSGARD™) must be emptied and refilled ⅔ full with fresh saline solution just before disinfecting the lenses and must always be tightly closed to prevent leakage of saline from the case and subsequent dehydration of the lenses. In order to remove protein deposits which may form on the lenses, wearers should use the SOFLENS™ Enzymatic Contact Lens Cleaning Tablets according to the directions for use which accompany the tablets. To help remove tear residues and mucus as well as other deposits that tend to accumulate on the lens surface, patients should use Daily Cleaner according to the directions for use which accompany the product. Disinfecting with a BAUSCH & LOMB™ Disinfecting Unit I (ASEPTOR®) or II (ASEPTRON™) is necessary to kill microorganisms. If a BAUSCH & LOMB™ Disinfecting Unit I (ASEPTOR®) or II (ASEPTRON™) is not available, lenses may be disinfected by immersing your lenses in BAUSCH & LOMB™ Saline Solution in the carrying case and dropping the sealed case into a pan of already boiling water. Remove the pan from heat after ten minutes and allow the water to cool before removing your lenses. When this boiling method of disinfection is used, the lenses can be damaged if the boiling water is allowed to completely evaporate. Chemical disinfection with BAUSCH & LOMB™ Sterile Disinfecting Solution has been tested microbiologically and shown to be an effective disinfection procedure for the SOFLENS hydrophilic contact lens. If chemical disinfection is used, the SOFLENS hydrophilic contact lens must be cleaned and rinsed daily with BAUSCH & LOMB™ Sterile Daily Cleaner and BAUSCH & LOMB™ Sterile Disinfecting Solution. The lens carrying case must be emptied and refilled with fresh BAUSCH & LOMB™ Sterile Disinfecting Solution prior to disinfecting the lens. Fresh BAUSCH & LOMB™ Sterile Disinfecting Solution must be used daily for storing and disinfecting the lens. **WARNING:** BAUSCH & LOMB™ STERILE DISINFECTING SOLUTION SHOULD NOT BE USED WITH HEAT. **Fluorescein:** Never use fluorescein while the patient is wearing the lenses because the lenses will become discolored. Whenever fluorescein is used, flush the eyes with sterile normal saline solution and wait at least one hour before replacing the lenses. Earlier replacement may cause the lenses to absorb residual fluorescein. **ADVERSE REACTIONS:** Serious corneal damage may result from wearing a SOFLENS Contact Lens which has been soaked in a conventional contact lens solution. Eye irritation may occur within a short time after putting on a hypertonic lens. Removal of the lens will relieve the irritation. A lens adheres very rarely to an eye as a result of sleeping with the lens on, or wearing a hypotonic lens. If a lens adheres for any reason, apply fresh saline and wait until the lens moves freely before removing it. Clinical studies indicate that corneal edema, as manifested by symptoms such as rainbows, halos around lights, or foggy vision, may occur in less than 5% of SOFLENS Contact Lens wearers. If these symptoms occur, the lenses should be removed and professional consultation obtained. Excessive tearing, unusual eye secretions, and photophobia are not normal. If these symptoms occur, the patient should be examined to determine their cause. Neovascularization of the cornea has been observed in some aphakic patients fitted with the SOFLENS Contact Lens, which may require discontinuation of the lens. Medical consultation should be obtained in such an instance.

4/80

BAUSCH & LOMB
SOFLENS DIVISION
Rochester, New York 14602

THE LACRIMAL SYSTEM DIAGNOSIS AND THERAPY

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OCTOBER 3-4, 1980

EMBRYOLOGY ANATOMY
SECRETORY/EXCRETORY THERAPEUTICS
MEDICAL AND SURGICAL DIAGNOSIS
RADIOLOGICAL TECHNIQUES

Faculty: Hampson A. Sisler, Course Coordinator; Drs. Byron C. Smith; William Cooper; Ira Jones; Harold Kirshner; Alvin Brackup; Gerhart Schwarz, R. Linsy Farris and John T. Simonton.

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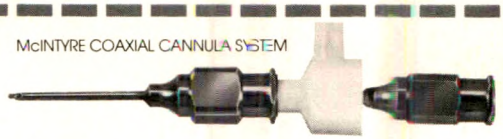
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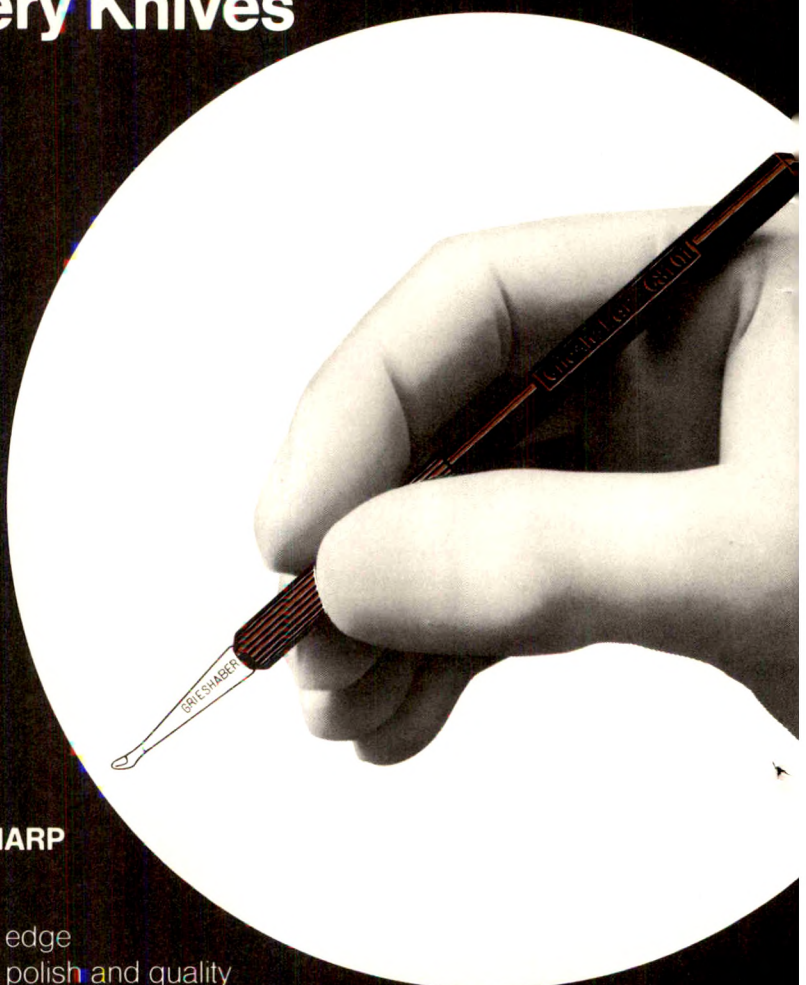
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A concise and detailed survey of the field of Intraocular Lens Implantation as compiled by the world's foremost and most experienced implant surgeons. The indications are described, both for the United States and overseas, from several different points of view. Complications are described and therapies are suggested. The results are tabulated including visual results, fusion and strabismus associations and accessory optical corrections. A comprehensive medical-legal section is included which alerts the implant surgeon to his responsibilities to himself and his patients.

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1980, 300 pp., $23.50
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Edited by JOEL POKORNY, PH.D., VIVIANNE C. SMITH, PH.D., GUY VERRIEST, M.D., and A. J. L. G. PINCKERS, M.D.

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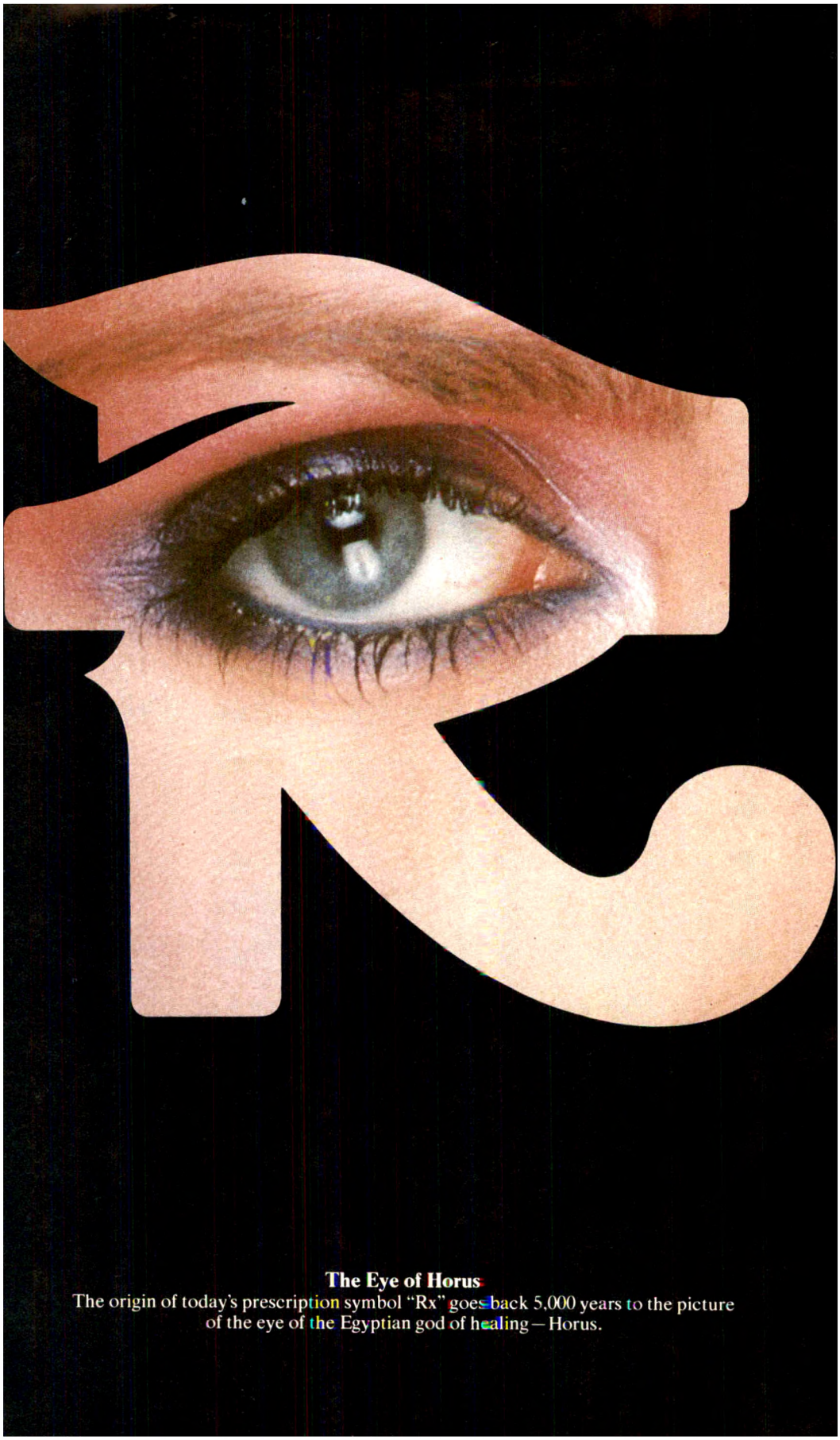
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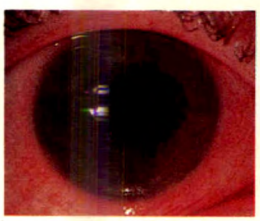
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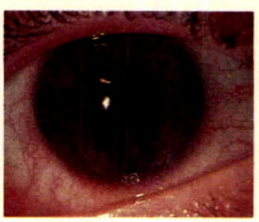
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In studies of patients unresponsive or intolerant to idoxuridine or vidarabine, VIROPTIC demonstrated 92% effectiveness. Dendritic and geographic ulcers were re-epithelialized in a mean time of 6 and 12 days, respectively.

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DESCRIPTION: VIROPTIC® is the brand name for trifluridine (also known as trifluorothymidine, F_3TdR,F_3T), an antiviral drug for topical treatment of epithelial keratitis caused by Herpes simplex virus. The chemical name of trifluridine is 5-trifluoromethyl-2′-deoxyuridine.

VIROPTIC sterile ophthalmic solution contains 1% trifluridine in an aqueous solution with acetic acid and sodium acetate (buffers), sodium chloride, and thimerosal 0.001% (added as a preservative).

CLINICAL PHARMACOLOGY: Trifluridine is a fluorinated pyrimidine nucleoside with *in vitro* and *in vivo* activity against Herpes simplex virus, types 1 and 2 and vacciniavirus. Some strains of Adenovirus are also inhibited *in vitro.*

Intraocular penetration of trifluridine occurs after topical instillation of VIROPTIC into human eyes. Decreased corneal integrity or stromal or uveal inflammation may enhance the penetration of trifluridine into the aqueous humor. Unlike the results of ocular penetration of trifluridine *in vitro,* 5-carboxy-2′-deoxyuridine was not found in detectable concentrations within the aqueous humor of the human eye.

INDICATIONS AND USAGE: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% is indicated for the treatment of primary keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus, types 1 and 2. VIROPTIC is also effective in the treatment of epithelial keratitis that has not responded clinically to the topical administration of idoxuridine or when ocular toxicity or hypersensitivity to idoxuridine has occurred. In a smaller number of patients found to be resistant to topical vidarabine, VIROPTIC was also effective.

The clinical efficacy of VIROPTIC in the treatment of stromal keratitis and uveitis due to Herpes simplex or ophthalmic infections caused by vacciniavirus and Adenovirus has not been established by well-controlled clinical trials. VIROPTIC has not been shown to be effective in the prophylaxis of Herpes simplex virus keratoconjunctivitis and epithelial keratitis by well-controlled clinical trials. VIROPTIC is not effective against bacterial, fungal or chlamydial infections of the cornea or nonviral trophic lesions.

During controlled multicenter clinical trials, 92 of 97 (95%) patients (78 of 81 with dendritic and 14 of 16 with geographic ulcers) responded to VIROPTIC therapy as evidenced by complete corneal re-epithelialization within the 14 day therapy period. In these controlled studies, 56 of 75 (75%) patients (49 of 58 with dendritic and 7 of 17 with geographic ulcers) responded to idoxuridine therapy. The mean time to corneal re-epithelialization for dendritic ulcers (6 days) and geographic ulcers (7 days) was similar for both therapies. In other clinical studies, VIROPTIC was evaluated in the treatment of Herpes simplex virus keratitis in patients who were unresponsive or intolerant to the topical administration of idoxuridine or vidarabine. VIROPTIC was effective in 138 of 150 (92%) patients (109 of 114 with dendritic and 29 of 36 with geographic ulcers) as evidenced by corneal re-epithelialization. The mean time to corneal re-epithelialization was 6 days for patients with dendritic ulcers and 12 days for patients with geographic ulcers.

CONTRAINDICATIONS: VIROPTIC brand Trifluridine Ophthalmic Solution, 1%, is contraindicated for patients who develop hypersensitivity reactions or chemical intolerance to trifluridine.

WARNINGS: The recommended dosage and frequency of administration should not be exceeded (see Dosage and Administration).

PRECAUTIONS:

General: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% should be prescribed only for patients who have a clinical diagnosis of herpetic keratitis.

VIROPTIC may cause mild local irritation of the conjunctiva and cornea when instilled but these effects are usually transient.

Although documented *in vitro* viral resistance to trifluridine has not been reported following multiple exposure to VIROPTIC, the possibility exists of viral resistance development.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Mutagenic Potential. Trifluridine has been shown to exert mutagenic, DNA damaging and cell transforming activities in various standard *in vitro* test systems, and clastogenic activity in *Vicia faba* cells.

Although the significance of these test results is not clear or fully understood, there exists the possibility that mutagenic agents may cause genetic damage in humans.

Oncogenic Potential. The oncogenic potential of trifluridine is unknown at this time. The oncogenic potential of trifluridine in rodents is being evaluated.

Pregnancy: The drug should not be prescribed for pregnant women unless the potential benefits outweigh the potential risks.

Nursing Mothers: It is unlikely that trifluridine is excreted in human milk after ophthalmic instillation of VIROPTIC because of the relatively small dosage (≤5.0 mg/day), its dilution in body fluids and its extremely short half-life (approximately 12 minutes). The drug should not be prescribed for nursing mothers unless the potential benefits outweigh the potential risks.

ADVERSE REACTIONS: The most frequent adverse reactions reported during controlled clinical trials were mild, transient burning or stinging upon instillation (4.6%) and palpebral edema (2.8%). Other adverse reactions in decreasing order of reported frequency were superficial punctate keratopathy, epithelial keratopathy, hypersensitivity reaction, stromal edema, irritation, keratitis sicca, hyperemia, and increased intraocular pressure.

OVERDOSAGE: Overdosage by ocular instillation is unlikely because any excess solution should be quickly expelled from the conjunctival sac.

Acute overdosage by accidental oral ingestion of VIROPTIC has not occurred. However, should such ingestion occur, the 75 mg dosage of trifluridine in a 7.5 ml bottle of VIROPTIC is not likely to produce adverse effects. Single intravenous doses of 15-30 mg/kg/day in children and adults with neoplastic disease produce reversible bone marrow depression as the only potentially serious toxic effect and only after 3-5 courses of therapy. The acute oral LD_{50} in the mouse and rat was 4379 mg/kg or higher.

DOSAGE AND ADMINISTRATION: Instill one drop of VIROPTIC Ophthalmic Solution, 1% onto the cornea of the affected eye every two hours while awake for a maximum daily dosage of nine drops until the corneal ulcer has completely re-epithelialized. Following re-epithelialization, treatment for an additional seven days of one drop every four hours while awake for a minimum daily dosage of five drops is recommended.

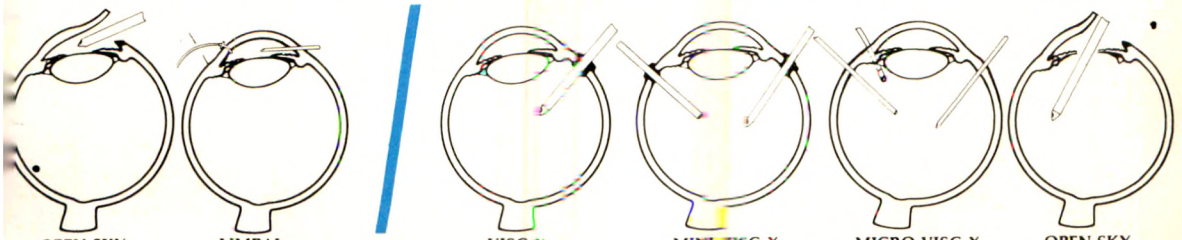
If there are no signs of improvement after seven days of therapy or complete re-epithelialization has not occurred after 14 days of therapy, other forms of therapy should be considered. Continuous adminsitration of VIROPTIC for periods exceeding 21 days should be avoided because of potential ocular toxicity.

HOW SUPPLIED: VIROPTIC Ophthalmic Solution, 1% is supplied as a sterile ophthalmic solution in a plastic Drop-Dose® dispenser bottle of 7.5 ml.

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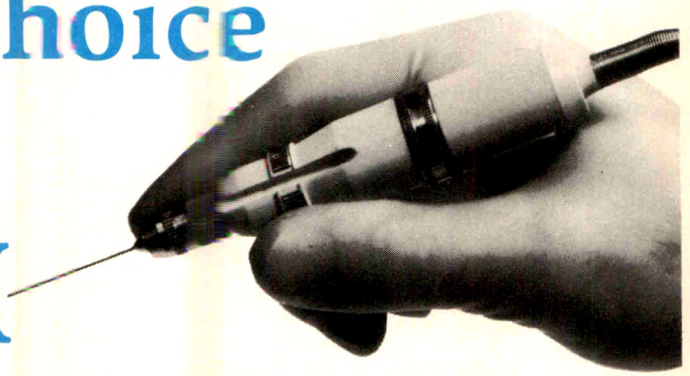
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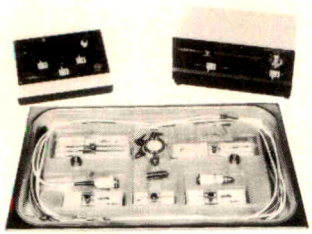
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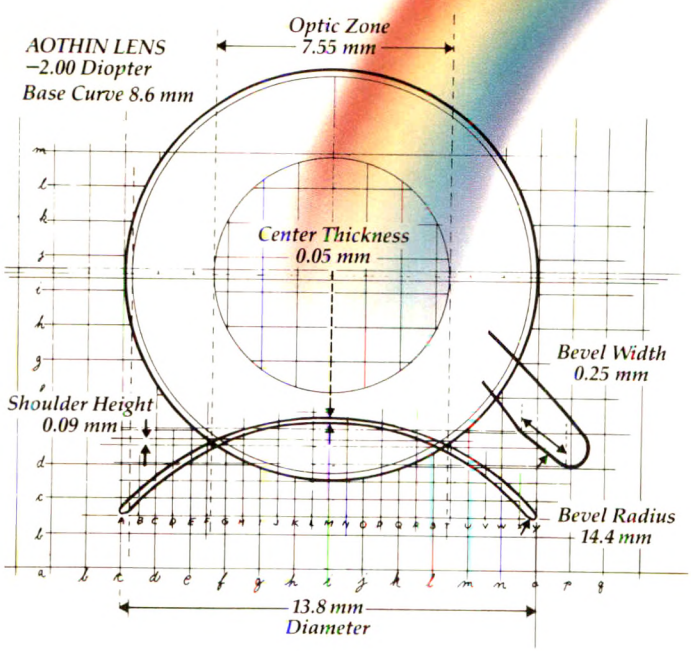
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Reference:
[1]Mandell RB: The "tight" soft contact lens. *Contact Lens Forum*, December, 1979, pp 21-32.

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TO BRING MORE OXYGEN TO THE CORNEA

NEW AOTHIN™

(TETRAFILCON A)

SOFT CONTACT LENS

AOTHIN™ (tetrafilcon A) Soft Contact Lens

DESCRIPTION: AOTHIN™ (tetrafilcon A) Soft Contact Lens is a hemispherical shell which covers the cornea and may cover a portion of the adjacent sclera. The lens material, tetrafilcon A, is a hydrophilic random terpolymer of 2-hydroxyethyl methacrylate, N-vinyl-2-pyrrolidone and methylmethacrylate. The polymer is a three-dimensional network of terpolymer chains joined by divinylbenzene cross-links. It consists of 57.5% tetrafilcon A and 42.5% water by weight when fully hydrated in 0.9% sodium chloride solution buffered with monobasic and dibasic sodium phosphates.

ACTIONS: In its hydrated state, the AOTHIN™ Soft Contact Lens is soft and pliable. When dry, the lens becomes hard and brittle. These states are completely reversible and a lens which has been permitted to dry out will recover all of its hydrated properties when placed in normal saline for a period of two hours. When placed on the human cornea, the hydrated lens acts as a refracting medium to compensate spherical ametropias. The material has a refractive index of 1.43 and the lens has a visible light transmittance greater than 97%.

INDICATIONS: AOTHIN™ Soft Contact Lenses are indicated for the correction of vision in persons with non-diseased eyes who have spherical ametropias, corneal astigmatism of 2.50 diopters or less and/or refractive astigmatism of 2.00 diopters or less.

CONTRAINDICATIONS: AOTHIN™ Soft Contact Lenses are contraindicated in the presence of any of the following conditions: (1) Acute and subacute inflammation of the anterior segment of the eyes. (2) Any eye disease which affects the cornea and conjunctiva. (3) Insufficiency of lacrimal secretion. (4) Corneal hypoesthesia. (5) Any systemic disease which may affect the eye or be exaggerated by wearing contact lenses.

WARNINGS: Medications and Eye Drops: AOTHIN™ Lenses must be stored in an appropriate solution when off the eyes, the type of solution being dependent on the system used for disinfection. When the lenses are disinfected with the thermal disinfection system, they may be stored *only* in LENSRINS® or BOIL n SOAK® Solution (sterile, buffered preserved saline solution). When lenses are disinfected with the chemical disinfection system, they may be stored *only* in FLEXSOL® Solution (sterile, isotonic buffered solution containing Adsorbobase — a water soluble polymeric vehicle — and preserved with thimerosal 0.001%, chlorhexidine 0.005% and edetate disodium 0.1%).

No ophthalmic solutions or medications, including hard contact lens solution, can be used by AOTHIN™ Lens wearers prior to or while the lenses are in place on the eyes. Also, no solutions, including hard contact lens solutions, other than LENSRINS® or BOIL n SOAK® Solution or FLEXSOL® Solution may be used on AOTHIN™ Lenses when they are off the eyes.

Since liquids and vapors may be absorbed by AOTHIN™ Lenses, they should not be placed in the mouth for wetting, nor wiped with a cloth or tissue.

Abrasions and Infections: If a lens becomes less comfortable than it was when first placed on the wearer's cornea, the lens should be removed immediately and the wearer's eye and the lens examined for the possible presence of a foreign body. If any eye abrasion, ulceration, irritation or infection is present, or any abnormal eye condition is observed concurrently with lens wear, the lens should be removed immediately and a physician consulted.

Wearing Restrictions: AOTHIN™ Soft Contact Lenses should not be worn while swimming, sleeping, or in the presence of irritating fumes or vapors.

Visual Blurring: If visual blurring occurs, the lens must be removed until the condition subsides.

Lens Sanitation: Patients must adhere to the recommended daily sanitary care procedures for AOTHIN™ Soft Contact Lenses. Failure to follow this procedure may result in the development of serious ocular infections.

PRECAUTIONS: Storage: AOTHIN™ Lenses may be stored *only* in the appropriate storage solution: LENSRINS® or BOIL n SOAK® Solution or FLEXSOL® Solution depending on disinfecting methods used. If left exposed to air, the lenses will dehydrate, become brittle and break readily. If a lens dehydrates, it should be soaked in either LENSRINS® BOIL n SOAK® or FLEXSOL® Solution until it returns to a soft, supple state.

Cleaning and Disinfecting: AOTHIN™ Lenses must be both cleaned and disinfected daily. Separate procedures and products are needed to clean and to disinfect. Two methods of disinfection, thermal or chemical, have been shown to be equally effective. The choice of disinfection system should be made in consultation with your eye care practitioner.

Cleaning: Daily cleaning is necessary to remove mucus and other deposits which may have accumulated on the lens surface. Each time the lenses are removed from the wearer's eyes, both surfaces of the lenses must be cleaned using several drops of PREFLEX® Cleaning Solution (sterile solution of a nonionic cleaner preserved with thimerosal 0.004% and edetate disodium 0.2%). Lenses must be cleaned before they are disinfected, as deposits on the lenses tend to harden and become more difficult to remove after the lenses are disinfected.

Disinfecting: AOTHIN™ Soft Contact Lenses may be disinfected with either a thermal or chemical regimen. One method or the other must be selected, but not both. THE USER MUST NOT ALTERNATE BETWEEN METHODS.

Thermal Disinfection Method: AOTHIN™ Lenses may be effectively disinfected using a disinfecting unit approved for use with tetrafilcon A hydrophilic contact lenses. After cleaning with PREFLEX® Cleaning Solution, rinse lenses with LENSRINS® or BOIL n SOAK® solution and disinfect in an approved thermal disinfection unit. The lens storage containers must be emptied and filled with fresh LENSRINS® or BOIL n SOAK® solution just prior to disinfecting the lenses.

Alternate Thermal Disinfection Method: If a thermal unit is not available, the following disinfection system can be used temporarily. After cleaning the lenses according to instructions, fill the lens storage container with fresh LENSRINS® or BOIL n SOAK® solution. Bring a pan of water to a rolling boil. Remove the pan from heat. Place the tightly sealed lens storage container into the hot water and allow to cool. When the water is cool, lenses have been disinfected.

NOTE: Use of an approved thermal disinfection unit should be resumed as soon as possible.

Chemical Disinfection: Disinfection with PREFLEX® Cleaning Solution, FLEXSOL® Solution and NORMOL® Rinsing Solution (sterile isotonic saline solution preserved with thimerosal 0.001% and chlorhexidine 0.005%) has also been shown to be an effective disinfection system for daily care of AOTHIN™ (tetrafilcon A) Soft Contact Lenses.

AOTHIN™ Lenses must be cleaned and rinsed daily (or after wearing) with PREFLEX® Cleaning Solution and NORMOL® Rinsing Solution. The FLEXKIT Lens Case must be emptied and refilled with fresh FLEXSOL® Solution each time the lens is stored. Fresh FLEXSOL® Solution must be used daily for storage and disinfection.

WARNING: DO NOT MIX OR ALTERNATE THE DISINFECTION AND STORAGE SYSTEMS. FLEXSOL® SOLUTION SHOULD NOT BE USED WITH HEAT.

Hygiene: Before handling the lenses, hands must be washed, rinsed thoroughly and dried with a lint-free towel. Cosmetics, lotions, soaps, oil and hand creams must not come in contact with the lenses since eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until the spray has settled.

Fluorescein: Never use fluorescein while the patient is wearing the lenses because the lenses will become discolored. Whenever fluorescein is used, flush the eyes with LENSRINS® or BOIL n SOAK® and wait at least one hour before replacing the lenses. Too early replacement may allow the lenses to absorb residual fluorescein.

ADVERSE REACTIONS: Serious corneal damage may result from wearing lenses which may have soaked in hard contact lens solutions. Eye irritation may occur within a short time after putting on a hypotonic lens. Removal of the lens will relieve the irritation.

Very rarely a lens may adhere to an eye as a result of a patient sleeping with the lens on, or as a result of wearing a hypotonic lens. If a lens adheres for any reason, the patient may be instructed to apply a few drops of LENSRINS® or BOIL n SOAK® Solution (if using a thermal disinfection regimen) or ADAPETTES® Cleaning and Rewetting Solution (sterile, isotonic, buffered solution containing Adsorbobase and preserved with thimerosal 0.004% and edetate disodium 0.1%) (if using a chemical disinfection regimen), and wait until the lens moves freely before removing it.

Clinical studies indicate that corneal edema as manifested by symptoms such as rainbows or halos around light or visual blurring may occur if lenses are worn continuously for too long a time. Removal of the lenses and a rest period of at least one hour generally relieve these symptoms. If symptoms do not subside promptly, professional consultation should be obtained.

Excessive tearing, unusual eye secretions and photophobia are not normal. If these symptoms occur, the patient should be examined to determine their cause.

DOSAGE AND ADMINISTRATION: Fitting: Conventional methods of fitting contact lenses do not apply to AOTHIN™ Soft Contact Lenses. For a detailed description of the fitting technique, refer to the Fitting Guide for AOTHIN™ (tetrafilcon A) Soft Contact Lenses, copies of which are available from: American Optical Corp., Vision Care Products, Southbridge, MA 01550.

Wearing Schedule: There may be a tendency for the patient to overwear the lenses initially. Therefore, the importance of adhering to the following initial daily wearing schedule should be stressed to the patient.

Day	Wear Time (Hours)	Rest Period (Hours)	Wear Time (Hours)
1	4	2	4
2	4	2	4
3	5	2	5
4	6	2	5
5	7	2	5
6	7	1	6
7	8	1	7
8	8	1	8
9	9	1	8
10–14	10	1	balance of
Thereafter	all waking hours		waking hours*

*lenses should never be worn 24 hours a day.

LENS CARE AND HANDLING: Care must be taken on the initial visit to assure that the patient is supplied with an approved disinfection system and fully understands all care and handling instructions for the lenses. As with any contact lens, regular recall visits are necessary to assure patient health and compliance with instructions.

HOW SUPPLIED: Each lens is supplied sterile in a glass vial containing 0.9% sodium chloride solution buffered with monobasic and dibasic sodium phosphates. The glass vial is marked with the Base Curve, Diameter, Dioptric Power, and Lot Number.

Approved thermal and chemical disinfection systems and care kits are available from a variety of manufacturers. Instructions on the use of these systems are contained in the AOTHIN™ Patient Instruction Booklet.

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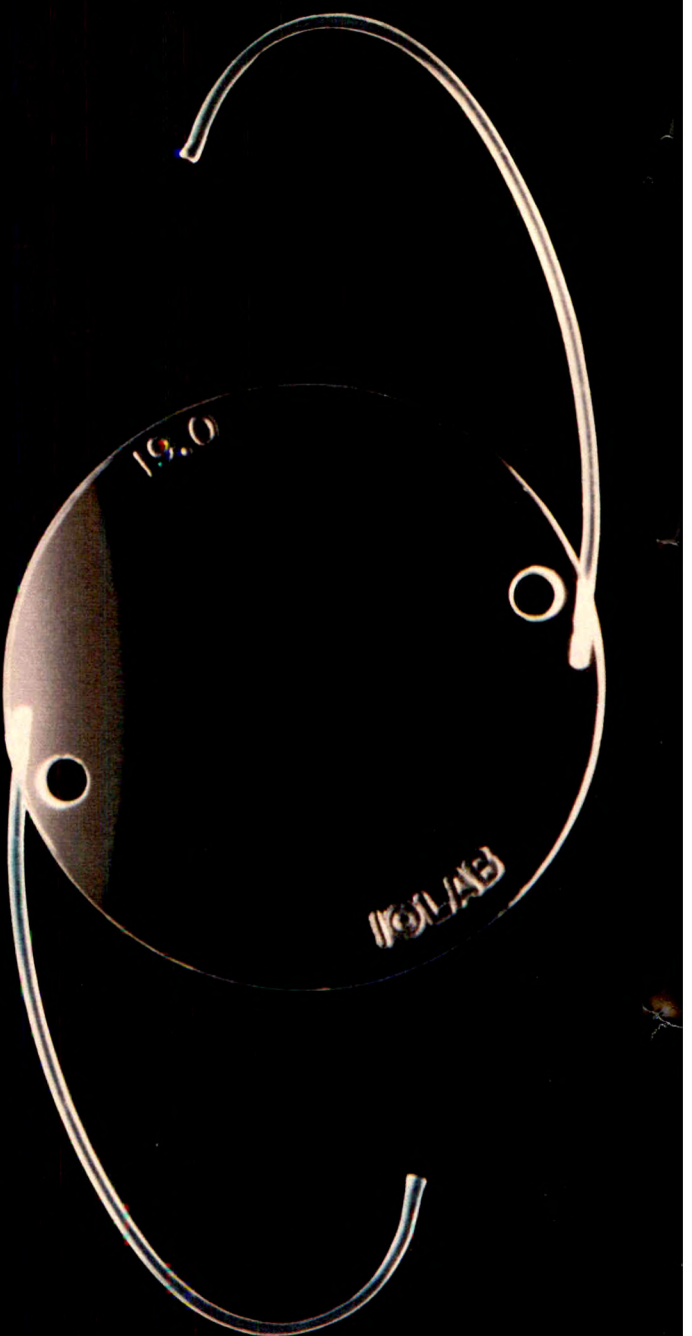
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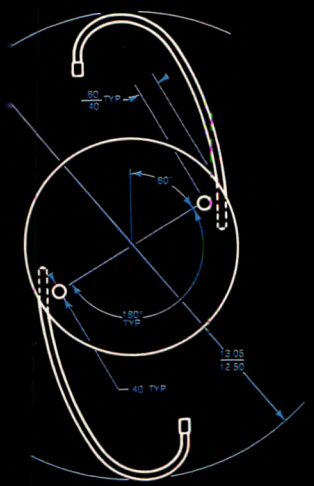
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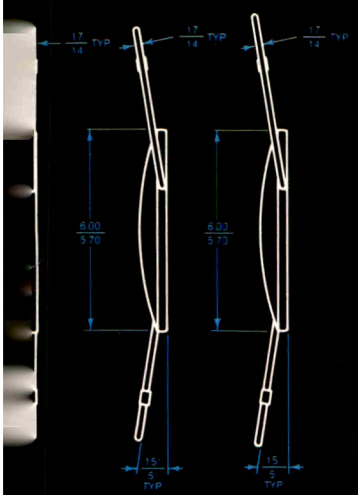
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ACTIONS The gram-positive bacteria against which gentamicin sulfate is active include coagulase-positive and coagulase-negative staphylococci, including certain strains that are resistant to penicillin; Group A beta-hemolytic and nonhemolytic streptococci; and *Diplococcus pneumoniae*. The gram-negative bacteria against which gentamicin sulfate is active include certain strains of *Pseudomonas aeruginosa*, indole-positive and indole-negative *Proteus* species, *Escherichia coli*, *Klebsiella pneumoniae* (Friedlander's bacillus), *Haemophilus influenzae* and *Haemophilus aegyptius* (Koch-Weeks bacillus), *Aerobacter aerogenes*, *Moraxella lacunata* (diplobacillus of Morax-Axenfeld), and *Neisseria* species, including *Neisseria gonorrhoeae*. Although significant resistant organisms have not been isolated from patients treated with gentamicin at the present time, this may occur in the future as resistance has been produced with difficulty *in vitro* by repeated exposures.

INDICATIONS GARAMYCIN **Ophthalmic Solution** and **Ointment** are indicated in the topical treatment of infections of the external eye and its adnexa caused by susceptible bacteria. Such infections embrace conjunctivitis, keratitis and keratoconjunctivitis, corneal ulcers, blepharitis and blepharoconjunctivitis, acute meibomianitis, and dacryocystitis.

CONTRAINDICATIONS GARAMYCIN **Ophthalmic Solution** and **Ointment** are contraindicated in patients with known hypersensitivity to any of the components.

WARNINGS GARAMYCIN **Ophthalmic Solution** is not for injection. It should never be injected subconjunctivally, nor should it be directly introduced into the anterior chamber of the eye.

PRECAUTIONS Prolonged use of topical antibiotics may give rise to overgrowth of nonsusceptible organisms, such as fungi. Should this occur, or if irritation or hypersensitivity to any component of the drug develops, discontinue use of the preparation and institute appropriate therapy.

Ophthalmic ointments may retard corneal healing.

ADVERSE REACTIONS Transient irritation has been reported with the use of GARAMYCIN **Ophthalmic Solution.**

Occasional burning or stinging may occur with the use of GARAMYCIN **Ophthalmic Ointment.**

DOSAGE AND ADMINISTRATION GARAMYCIN **Ophthalmic Solution:** instill one or two drops into the affected eye every four hours. In severe infections, dosage may be increased to as much as two drops once every hour.

GARAMYCIN **Ophthalmic Ointment:** apply a small amount to the affected eye two to three times a day.

HOW SUPPLIED GARAMYCIN **Ophthalmic Solution—Sterile,** 5-ml. plastic dropper bottle, sterile, boxes of one and six. Store away from heat.

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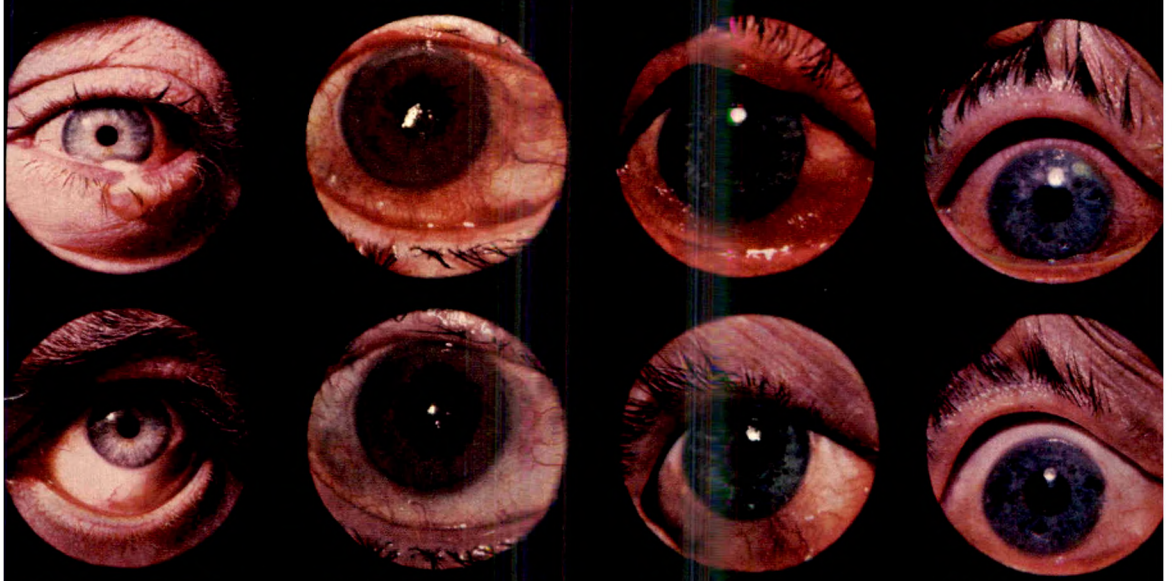
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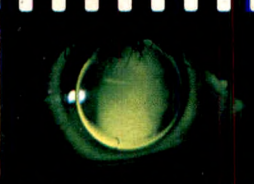
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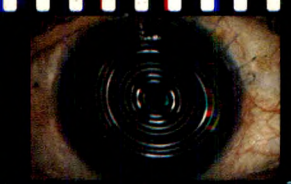
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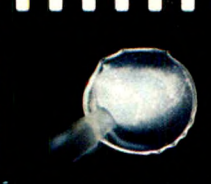
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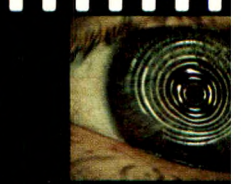
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CONTRAINDICATIONS
Acute and subacute inflammations of the anterior segment of the eye; any eye disease which affects the cornea or conjunctiva; insufficiency of lacrimal secretion; corneal hypoesthesia; or any systemic disease which may affect the eye or be exacerbated by wearing contact lenses.

WARNINGS
(1) If the lens becomes less comfortable to the wearer than when first placed on the wearer's cornea, a foreign body may be present. The patient should remove the lens immediately. If discomfort persists, an eye care practitioner should examine the patient as soon as possible to determine if any corneal abrasion, ulceration, irritation or infection is present.

(2) Aphakic patients should not be fitted during the postoperative period until the surgeon determines that the eye has healed completely.

(3) Only use Preflex® Sterile Cleaning Solution, Flex-Care® Sterile Solution, and Adapettes® Sterile Lubricating Solution with the POLYCON lens. Store ONLY in Flex-Care® Sterile Solution. Patients must adhere to recommended daily lens care regimen. FAILURE TO DO SO MAY RESULT IN DEVELOPMENT OF SERIOUS OCULAR INFECTIONS.

(4) Wearers must not use any medicaments or ophthalmic solutions other than Adapettes® solution prior to inserting the lens or while the lens is on the eye.

(5) Remove before sleeping or in the presence of noxious and irritating vapors.

PRECAUTIONS
THE POLYCON CONTACT LENS MUST BE BOTH CLEANED AND DISINFECTED DAILY. ONE PROCEDURE DOES NOT REPLACE THE OTHER. To disinfect the lens, it is stored in fresh Flex-Care® solution for a minimum of four hours daily in the patient lens storage case. After removal of the lens from the storage case, rinse the lens thoroughly with Flex-Care® solution and apply Adapettes® lubricating solution to the lens surfaces as a wetting agent prior to inserting the lens in the eye.

ADVERSE REACTIONS
Rainbows or halos around objects or blurring of the vision. Excessive tearing, unusual eye secretions, and photophobia.

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References

1 Multi-clinic study of 418 patients, data on file, Syntex Ophthalmics, Inc.

2 Korb JE, Finnemore VM: Corneal edema with polymethylmethacrylate vs. gas-permeable rigid polymer contact lenses of identical design. To be published.

3 Williams CE: New design concepts for permeable rigid contact lenses. *JAOA* 50:331-336, 1979.

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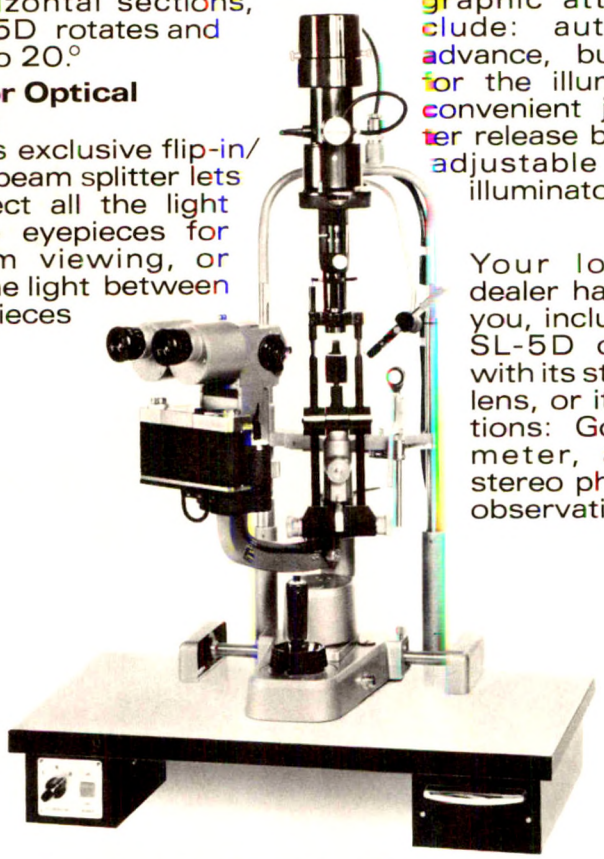
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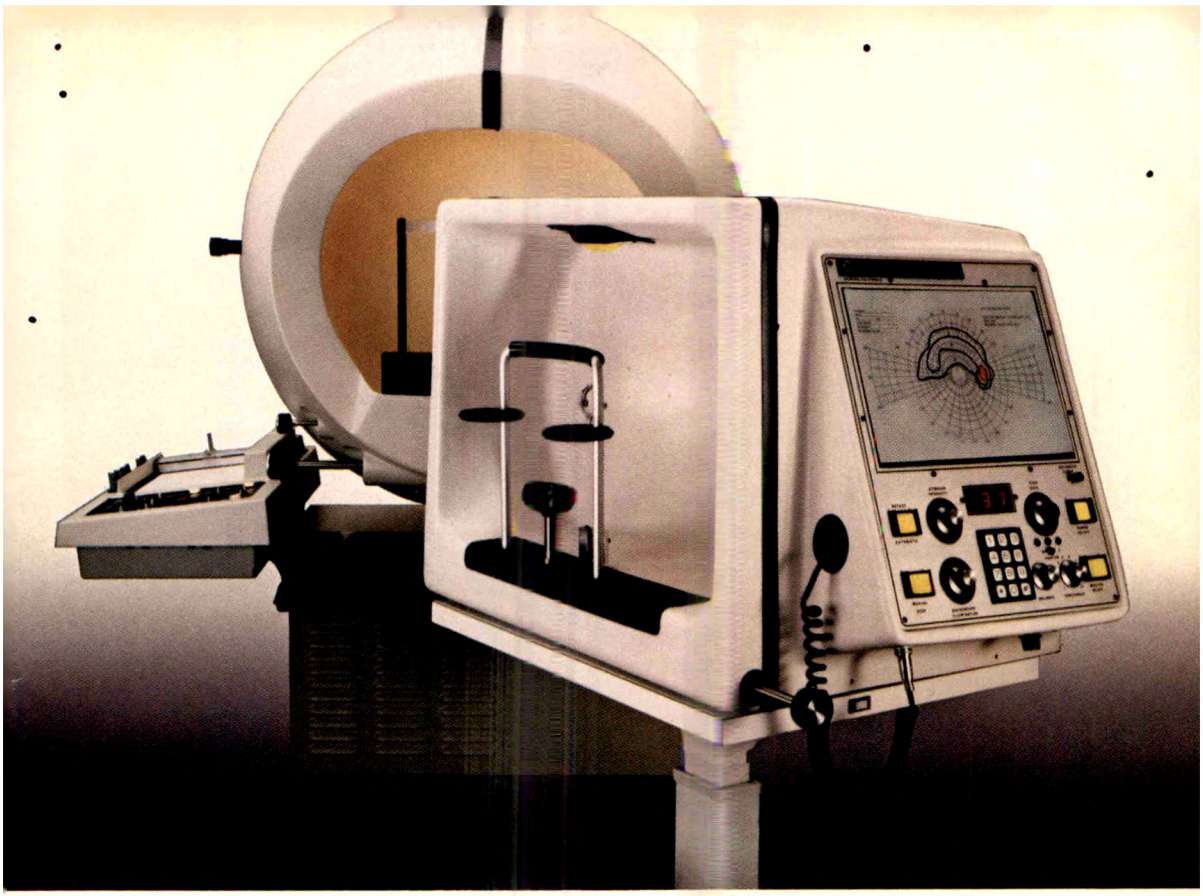


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NASAL VISUAL FIELD LOSS WITH INTRACRANIAL LESIONS OF THE OPTIC NERVE PATHWAYS

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Petah Tiqva, Israel

Nasal field abnormalities are most frequently encountered in retinal and anterior optic nerve pathology. Nasal field loss caused by intracranial lesions of the optic nerve pathways is rare. We report an analysis of the results obtained in five such cases.

CASE REPORTS

Case 1—A 61-year-old man was referred to our neurosurgical department because of severe headaches, obtundation, and psychomotor agitation. His medical history revealed that he had been under the care of the endocrinology department for ten years because of acromegaly and had been treated for hypertensive cardiovascular disease for three years. The patient had high blood pressure of 220/120 mm Hg. Neurologic examination revealed the patient to be obtunded, with nuchal rigidity, positive Kernig and Brudzinski signs, and a positive plantar reflex on the right. A complete ophthalmologic examination could not be performed at this time because of obtundation. Ocular pressure was normal. The pupils were of equal size and reacted normally to light. Examination of the ocular fundi showed a slight pallor without pathologic excavation of the optic disks. Lumbal puncture revealed the presence of a bloody liquid with a high protein level (170 mg/100 ml). Films of the skull and tomogram showed an enlargement of the sella turcica and a straight dorsum (Fig. 1). Bilateral carotid and vertebral angiography revealed large bilateral dolichoectatic carotid (Fig. 2) and vertebral arteries. Pneumoencephalogram showed that there was no suprasellar extension of the tumor (Fig. 3). Radioactive iodine serum albumin cisternography showed normal circulation of intracranial cerebrospinal fluid. Electroencephalography revealed a slight bilateral disturbance.

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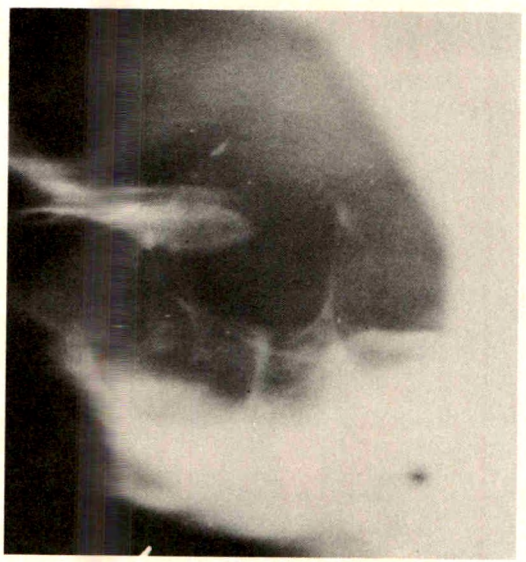


Fig. 1 (Manor and associates). Case 1. Skull films showing a markedly enlarged sella turcica, with stretching of dorsum and slight dorsal reclination.

The patient was given only symptomatic treatment. Subsequently there was a gradual improvement in his condition and his blood pressure decreased to 190/90 mm Hg. Because there was no suprasellar extension of the hypophyseal tumor, surgery was not performed. At ophthalmologic examination visual acuity was R. E.: 6/6 (20/20) and L. E.: 6/7.5 (20/25). Visual field examination revealed a bilateral loss of the inferior nasal quadrant (Fig. 4), with sloping margin. Using the I_3 isopter there was also an evident inferior temporal defect in the left eye. When the patient was again hospitalized two years later for resection of a sigmoid carcinoma, there were no evident changes in his visual fields but there were signs of Group I hypertensive retinopathy.

Case 2—An 18-year-old man had an episode of severe headache and fever associated with leukocytosis and an accelerated erythrocyte sedimentation rate (47 mm in the first hour), after which he complained of a dramatically diminished visual acuity in both eyes. Visual acuity was R. E.: counting fingers eccentrically from a distance of .5 meter; L. E.: 6/7.5

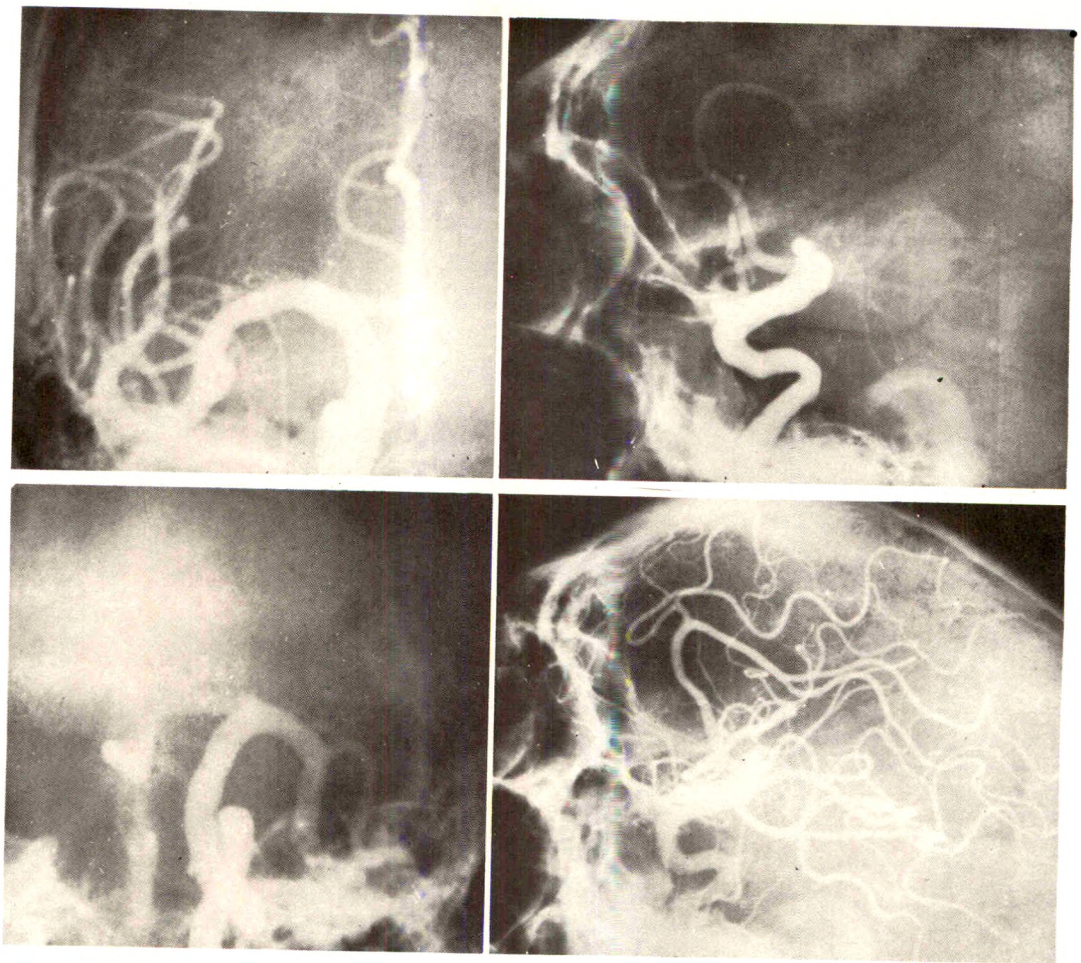


Fig. 2 (Manor and associates). Case 1. Top, Right carotid angiography. Bottom, Left carotid angiography. Both internal carotid arteries were markedly dilated and tortuous with small atheromatous plaques on their walls.



Fig. 3 (Manor and associates). Case 1. Pneumoencephalography showing normal shadows of the infundibulum and of the carotid arteries and no evidence of suprasellar space-occupying lesion.

(20/25) and one week later 6/12 (20/40). The right visual field (Fig. 5, right) showed a loss of the inferior field with a central scotoma and a concentric contraction of the upper field with an arcuate scotoma breaking out. The left visual field (Fig. 5, left) had an arcuate scotoma breaking down in a nasal visual field loss. Examination of the ocular fundi showed edema of both disks with an apparent halo at the border of the edematous area and a star figure in the macula. Several weeks later the temporal part of the optic disks was pallid (Fig. 6).

The results of the general and neurologic examinations as well as those of carotid angiography and pneumoencephalography were all within normal limits. Electroencephalography showed a slight diffuse irregularity, which was more pronounced on the right side. The results of blood tests, including erythrocyte sedimentation rate, complete blood cell count, blood urea, nitrogen, glucose, electrolytes, protein phoresis, and the Kahn test, as well as

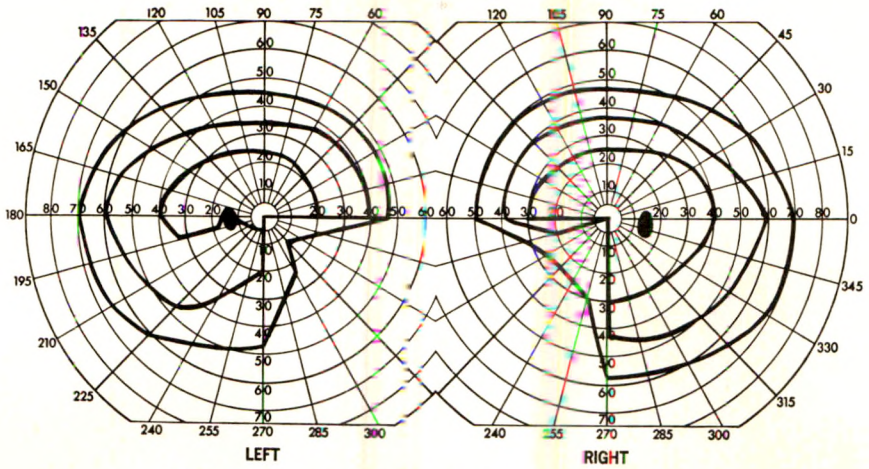


Fig. 4 (Manor and associates). Case 1. Visual fields.

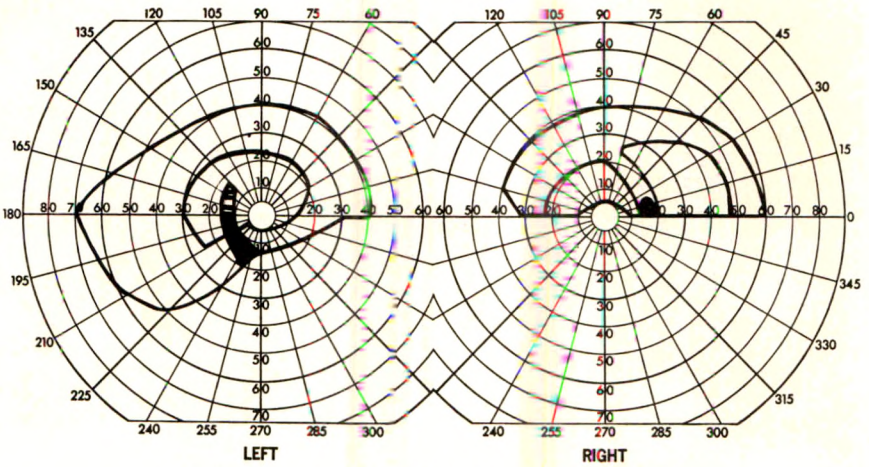


Fig. 5 (Manor and associates). Case 2. Visual fields.

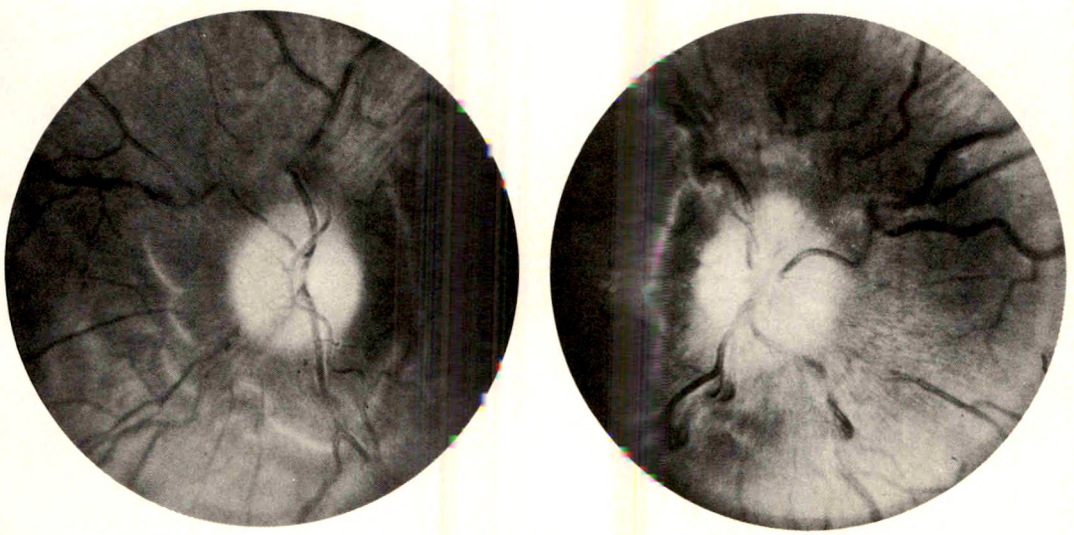


Fig. 6 (Manor and associates). Case 2. Optic disks showing a combination of edema and atrophy.

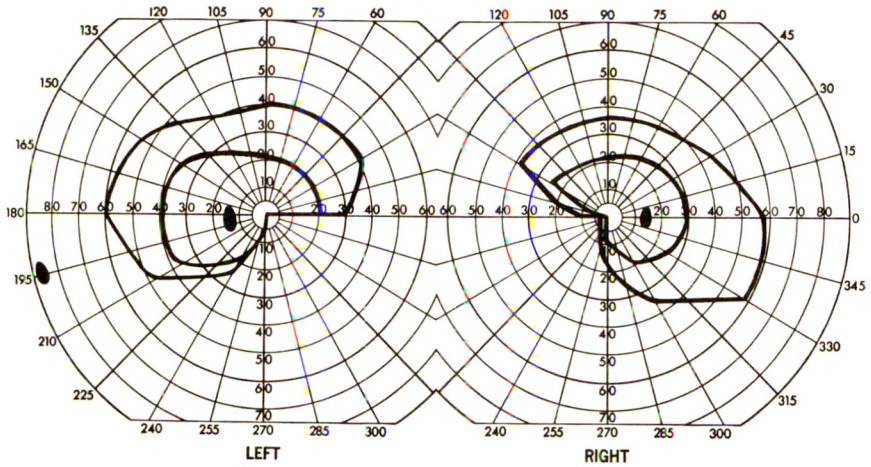


Fig. 7 (Manor and associates). Case 3. Visual fields.

cerebrospinal fluid analysis were all within normal limits.

A continuing deterioration in the visual acuity and fields was noted, with no improvement in response to corticosteroid treatment. Because of this, and despite the negative findings in the laboratory and contrast studies, exploration of the chiasmal area was undertaken one month after his initial examination. This revealed a thick membrane enveloping the superior part of the optic nerves and chiasm, suggesting optochiasmatic arachnoiditis. The adhesions connecting the membrane to the optic structures were severed and the membrane dissected as much as possible. After the operation, the patient's visual acuity stabilized at R. E.: 6/90 (20/300)and L. E.: 6/6 (20/20) and there was an improvement in visual fields. The optic disk edema disappeared with a partial residual optic atrophy.

Case 3—A 43-year-old woman complained of headaches, dizziness, a weakness of the left side of the body, and a bilateral decrease in visual acuity. On admission results of the general physical examination were unremarkable and the neurologic examination confirmed the presence of a motor weakness in the left leg. There was right anosmia. Visual acuity was 6/30 (20/100) in each eye and there was a bilateral loss of the inferior nasal quadrants as well as depression of the lower part of the inferior temporal quadrants (Fig. 7). Examination of the optic disks revealed papilledema in the right eye and blurring of disk margins in the left eye without any pallor. Ocular pressure was normal.

Electroencephalography showed a severe disturbance in the anterior portion of the right hemisphere. Right carotid angiography revealed a space-occupying lesion in the right frontobasal area.

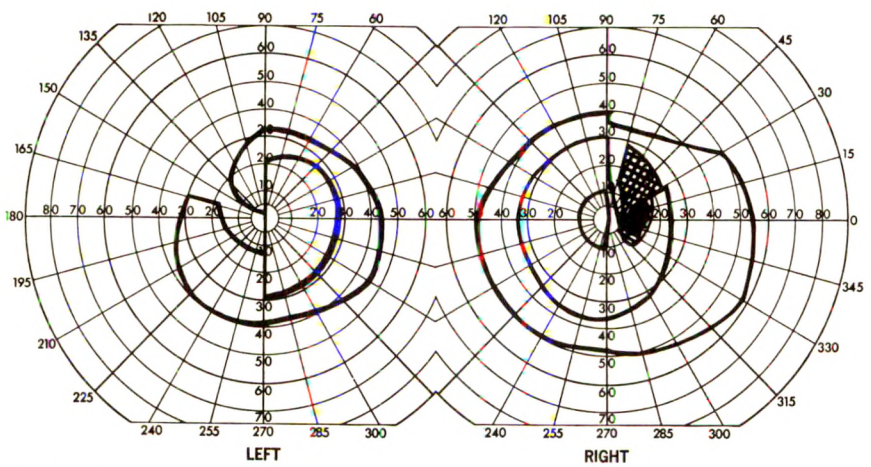


Fig. 8 (Manor and associates). Case 4. Visual fields before the pituitary apoplexy appearance.

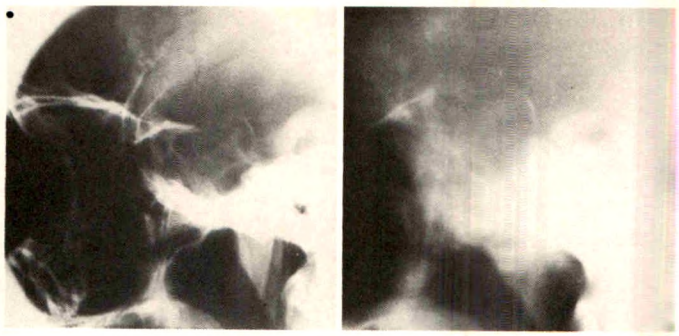


Fig. 9 (Manor and associates). Case 4. The skull X-ray and lateral tomographic cuts showing marked enlargement of the sella turcica, ballooning of the sella (left), and severe porosis of the anterior wall of the floor and dorsum (right).

During surgery a meningioma located in the right olfactory groove was found pushing the optic nerve downward and laterally against the carotid artery. On the other side the carotid artery was pushed toward the optic nerve by the anterior cerebral artery, which was displaced by the mass. The postoperative course was uneventful. The right papilledema subsided. The disks were sharp with slight pallor. Although the visual acuity improved to 6/7.5 (20/25) in each eye, the nasal field defects were unchanged.

The patient was lost to follow-up but we received information from another hospital that there had been a recurrence of the tumor with involvement of both sides. The patient refused a second operation and died after four years of total blindness.

Case 4—A 40-year-old man was hospitalized because of a pituitary tumor. The visual fields showed bilateral upper temporal defects (Fig. 8). There was slight pallor of the disks on the temporal side with the cup:disk ratio less than 0.3. Tomographic examination of the sella turcica showed severe porosis of the anterior wall, floor, and dorsum and ballooning of the sella (Fig. 9). Right carotid angiography revealed slight elevation and stretching of the proximal segment of the anterior cerebral artery (Fig. 10). An intrasellar adenoma with suprasellar extension was presumed. During the hospitalization pituitary apoplexy occurred with bilateral blindness, mydriasis, and complete ophthalmoplegia. In an urgent operation a necrotic tumor mass with intratumoral hematoma was removed through a transsphenoidal approach. Immediately after the operation the patient began to see, but only with the right eye.

One week postoperatively visual acuity of the right eye was 6/10 (20/32) and the visual field had changed; the upper temporal defect had disappeared and there was an inferior arcuate scotoma breaking down in a quadrantic nasal field loss (Fig. 11). This defect persisted. The optic disks showed pallor, more on the left than the right, and the left eye has remained amaurotic.

Case 5—Two months before hospitalization a 37-year-old man noticed decreased visual acuity, particularly in the right eye, instability in walking, fatigue, weight loss, and diminished libido. He was suffering from hypertension and coronary insufficiency and twice in the past had had myocardial infarction. On general examination he had a pale dry skin. His blood pressure was 200/120 mg Hg and his pulse was 88 beats per minute and regular. Results of a neurologic examination were normal. Ophthalmologic examination revealed visual acuity to be R. E.: 6/15 (20/50) and L. E.: 6/7.5 (20/25). Intraocular pressure was 16 mm Hg in each eye. The visual field of the right eye had an absolute arcuate scotoma that began in the upper temporal visual field and broke down in a complete quadrantic lower nasal visual field loss. The visual field of the left eye had a lower arcuate scotoma showed an enlargement of the sella turcica with double contours and porosis of the dorsum sella. The left side of the sellar floor was much deeper than the right (Fig. 13). On brain scan with technetium-99 breaking down in a relatively lower nasal visual field loss (Fig. 12). Pupillary reflexes were normal.

Examination of the ocular fundi revealed a normal cup:disk ratio, temporal pallor of the right optic disk, and a normal-appearing left disk.

X-rays of the skull and tomograms of the sellar area



Fig. 10 (Manor and associates). Case 4. Right carotid angiography showing slight elevation and stretching of the proximal segment of the anterior cerebral artery.

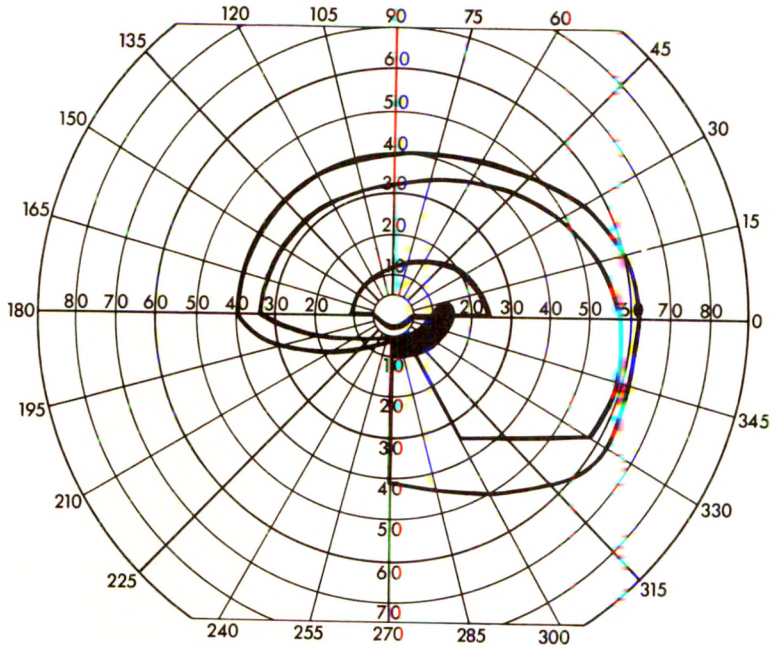


Fig. 11 (Manor and associates). Case 4. Right visual field after pituitary apoplexy, reversible blindness, and surgical intervention.

there was a pathologic concentration in the right suprasellar area. Computed tomography (Fig. 14) showed a suprasellar mass lesion of high density that was somewhat irregular in shape with homogeneous enhancement. There was considerable ventricular dilatation and moderate cortical atrophy. Endocrinologic tests indicated adrenal and thyroid insufficiency. The results of general laboratory tests were within normal limits.

A frontal craniotomy was performed and a partially cystic pituitary tumor was removed through the subfrontal approach. The tumor was pushing both optic nerves upward and laterally. The optic nerves appeared to be stretched and under tension; the optic chiasm was postfixed. After incision of the sellar diaphragm the cystic content of the tumor burst with pressure. Histologic examination confirmed the presence of a chromophobe adenoma of the hypophysis.

The postoperative course was uneventful. Ophthalmologic examination done three weeks after the operation showed normalization of visual acuity to 6/6 (20/20) in each eye and the disappearance of the

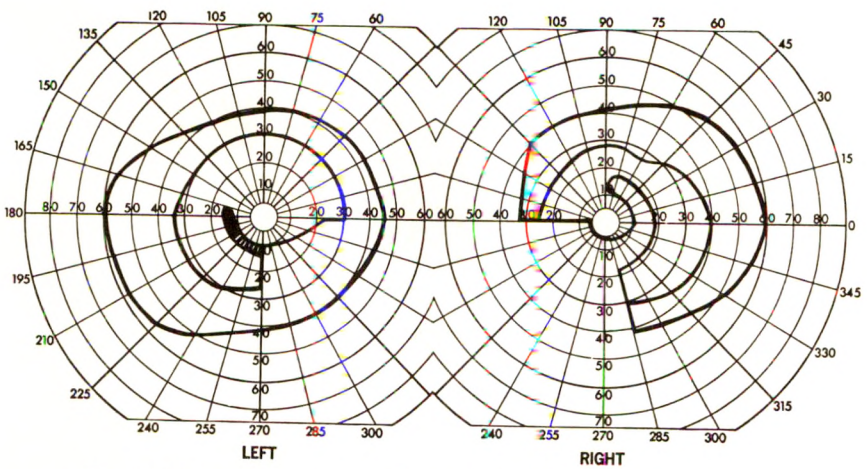


Fig. 12 (Manor and associates). Case 5. Visual fields before operation.

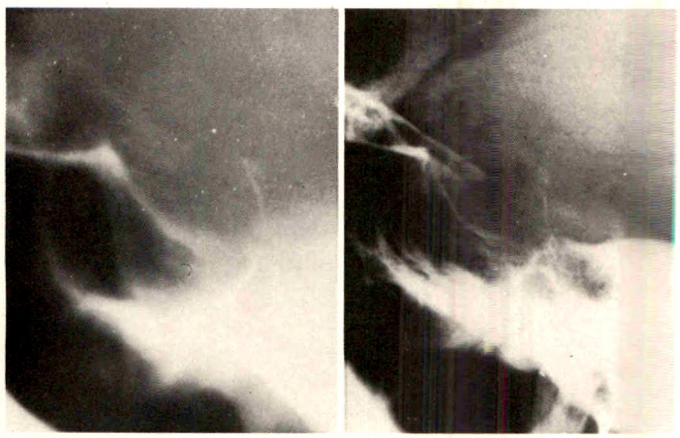


Fig. 13 (Manor and associates). Case 5. X-ray of skull showing enlargement of the sella turcica, reclination of the dorsum (left), and porosis of the dorsum and the posterior floor (right) sellar. The contour of the floor is double with marked, unilateral, deep protrusion into the sphenoid sinus.

abnormality in the upper temporal visual field. However, there was still a bilateral lower quadrant visual field loss with arcuate scotoma breaking into the defect (Fig. 15) and temporal pallor of both optic nerve heads was evident.

Discussion

In neurosurgical patients the nasal visual field loss is usually caused by



Fig. 14 (Manor and associates) Case 5. Computed tomography scan showing a well-defined suprasellar mass that obliterated the suprasellar cisterns. There was also considerable ventricular dilatation.

chronic atrophic papilledema.[1-5] Salinas-Garcia and Smith,[6] in their report of eight cases of nasal visual field loss referred for neuro-ophthalmologic examination, found only two cases with intracranial involvement of the optic nerve, both of which had hydrocephalus.

Sclerotic, ectatic internal carotid arteries have long been recognized as a cause of optic atrophy and nasal visual field defects.[7-26] but the frequency of occurrence and pathophysiology of optic nerve damage are still undetermined. Deep grooving of the optic nerve by an adjacent artery does not necessarily cause atrophy of nerve fibers.[27] Conversely, dolichoectasia of arteries at the base of the brain can sometimes cause atrophy of adjacent nerves, including the optic nerves and chiasm, as shown in the neuropathologic studies of Sacks and Linderberg.[28] Hilton and Hoyt[29] suggested that tortuous sclerotic carotid arteries cause optic atrophy by traction and stretching of nutrient vessels and by obliteration of their ostia.

The pathogenesis of nasal visual field defects resulting from intracranial compression of the optic nerves by a tumor or dilated arteries, or both, remains unclear.[3-34] When an optic nerve is elevated and deflected laterally by a slowly devel-

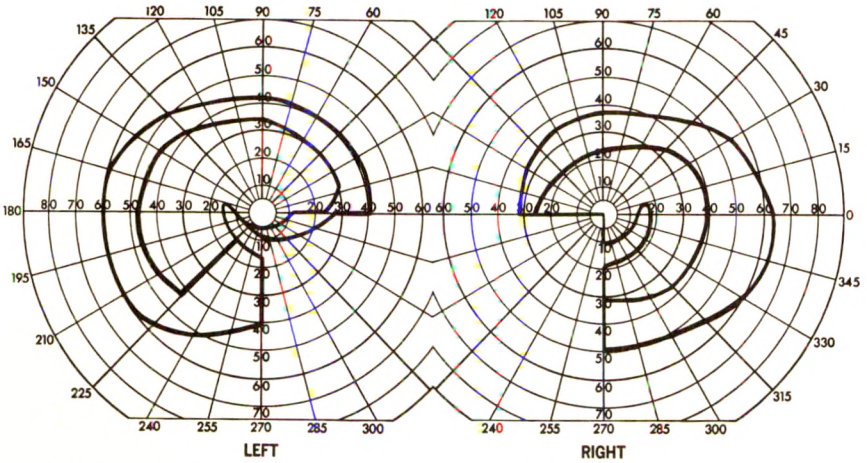


Fig. 15 (Manor and associates). Case 5. Visual fields after the surgical intervention.

oping compressive force, it may be injured on its lateral side by the dural fold at the entrance to the optic canal. Hoyt[35] stressed that optic atrophy caused by tumor pressure begins in those areas of the anterior visual pathways in which the intrinsic capillary network first collapses.

A summary of the intracranial causes of nasal visual field defects based on a review of previous reports[1-34] and our cases is presented in Figure 16. In our patients the causes included dolichoectatic carotid arteries, optochiasmatic arachnoiditis, olfactory-groove meningioma, pituitary apoplexy, and chromophobe adenoma. The common pathogenesis of these visual field defects was probably an impairment of circulation in the anastomotic prechiasmal arterial plexus. There must also have been other individual factors that determined the development of a nasal or altitudinal defect rather than the more common temporal visual field defect. For example, the decussated fibers of a postfixed chiasm may not be subject to direct pressure by a tumor. The presence of diseased arterial walls, (atherosclerosis, hypertension) in association with a space-occupying lesion may lead to more rapid vascular decompensation. When there is a congenital dolichoectatic artery, atherosclerosis and aging may induce a further development of this anomaly.

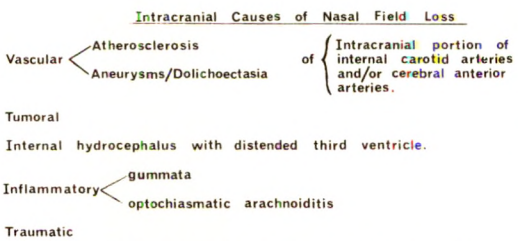


Fig. 16 (Manor and associates). Intracranial causes of nasal field loss.

The acromegalic, hypertensive patient who developed a subarachnoid hemorrhage (Case 1), deserves special attention because of the unexpected results: the bilateral lower nasal visual field loss instead of temporal visual field defects; the lack of correlation between the visual field defects and the pituitary tumor, which was only intrasellar; and the dolichoectatic carotid and vertebral arteries.

In Case 2, the low degree of edema of the disks was accompanied from the beginning by a decrease in visual acuity and a progressive worsening of the visual

field defects. These findings and the disk changes might have suggested a form of anterior ischemic optic neuropathy. However, considering the young age of the patient, the normal laboratory results, and the relentless progression of the visual field defects, exploratory surgery was done; the operation finding suggested optochiasmatic arachnoiditis.

The characteristics of Case 3 were almost identical to those of a case described by O'Connell and du Boulay.[33] In our patient the lower nasal visual field defects also appeared to be the consequence of a forward displacement of the vessels of the circle of Willis by an olfactory-groove meningioma. Papilledema was excluded as a possible cause for the visual field defects in our Case 3 because it was only unilateral and the visual field defects were bilateral.

The case of pituitary apoplexy (Case 4) is particularly interesting because the usual pattern of visual fields in hypophyseal tumors, temporal defects, changed after the pituitary apoplexy. After apoplexy and the operation, the visual field of the one eye that regained vision presented no further upper temporal visual field defect, presumably because the inferior crossed fibers were no longer compressed by the tumor and the damage was apparently reversible. However, after recovering from blindness, the visual field was similar to that seen in glaucoma, with an arcuate scotoma breaking down in inferior nasal quadrant loss. The subsequent persistence of this visual field defect can be explained by irreversible ischemic changes produced in the optic nerve during the time of pituitary apoplexy; that is, a form of posterior ischemic optic neuropathy.

Our fifth patient (Case 5), had two myocardial infarctions and a history of hypertension, a bilateral asymmetric lowering of visual acuity, visual field defects manifested mainly in arcuate scotomas breaking down in quadrantic nasal visual field loss, and a chromophobe adenoma of the hypophysis, a finding unusual for such a clinical appearance. The explanation for the lesion to the upper temporal nerve fibers in this case may lie in the surgical finding of a postfixed chiasm with a tumor process that impinged upon both optic nerves laterally. Possibly, in this particular situation a patient with diseased arteries is more likely to develop local ischemic changes.

In none of these five cases did the nasal visual field defect disappear, a fact that may suggest irreversible ischemia. Only in the case of optochiasmatic arachnoiditis was there any improvement after the operation. From our own experience and other published reports, we find that other characteristics of these visual field defects are as follows: a tendency to be asymmetrical, to involve the lower visual field, and to resemble the defects seen in glaucoma, although, unlike glaucoma, the central vision is also affected. Another characteristic is the tendency of the defect, in contrast to temporal hemianopia, ". . . to pay little, if any, respect to the vertical meridian . . . ,"[32] and thus to produce altitudinal defects. It appears that the term nasal hemianopia is not suitable in most cases because the visual field defects actually constitute a lower nasal quadrantanopia and usually involve the lower temporal quadrants as well. In such cases, therefore, the term, nasal visual field loss from intracranial causes would seem more appropriate.

Summary

Five patients developed nasal visual field defects as a result of involvement of the intracranial portion of the optic nerves. The cause in each patient, respectively, was as follows: (1) dolichoectatic carotid arteries, (2) optochiasmatic arachnoiditis, (3) meningioma of the olfactory groove, (4) pituitary apoplexy, and (5) pituitary chromophobe adenoma. The common factor in these cases was probably impaired circu-

lation in the prechiasmal arterial anastomotic network. The nasal visual field loss present in these cases was characterized by a pattern similar to that seen in glaucoma but with impairment of visual acuity. The superior nasal visual field was usually normal and the lower temporal visual field often defective.

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EARLY-ONSET ACCOMMODATIVE ESOTROPIA

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The onset of accommodative esotropia has previously been described as occurring between 6 months and 7 years of age[1]; the average age is 2½ years. At first the deviation of the eyes is intermittent, varying in frequency, duration, and angle size and it can be eliminated by antiaccommodative therapy. In contrast, congenital esotropia typically occurs as a constant large-angle deviation of the eyes observed at birth or during the first weeks of life; this disorder does not respond to antiaccommodative therapy. Because the cause, clinical course, therapy, and prognosis for cure of these two major components of esotropia are so different, it is convenient to categorize patients as having one or the other. Occasionally categorizing an infant patient is difficult because the clinical characteristics are typical for neither accommodative esotropia nor congenital esotropia. Particularly difficult is the diagnosis in infants whose esotropia was present at birth while the deviation is intermittent, whose esotropia began later during the first year of life while the deviation is constant, or who have a large hypermetropic refraction and a constant deviation regardless of the age at which the deviation began.

Accommodative esodeviations may be the result of either the need to clear the blurred vision caused by hypermetropia, or the exaggerated accommodative convergence associated with normal accommodation in a patient who has a high accommodative convergence ratio. Although accommodation previously was considered not to begin functioning until 1 year of age, it probably is present after the first few months of life.[2] Evidence of good vision within the first few months of life and clinical documentation of accommodative esotropia in patients as early as 3 months of age[3] supports the newer concept regarding early development of accommodation. Esophoria and intermittent esotropia with onset before 1 year of age previously were reported by Costenbader[4] who unfortunately did not provide details about antiaccommodative therapy in these patients; thus the presence of an accommodative factor is unknown. Pollard[5] and Burian and von Noorden[5] have reported cases of esotropia with hyperopia greater than 3 diopters occurring before age 12 months and aligned with spectacle correction.

We describe a group of infants with esotropia who were examined by 12 months of age and whose eyes were straightened at least initially with glasses or miotics, indicating that accommoda-

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tive convergence was the probable cause.

Subjects and methods

We examined our records for patients with esotropia who, during their first year of life, were examined and were prescribed glasses or a miotic, or both. This therapy straightened the eyes at least initially while fixating at 6 meters or one-third meter, or both. We followed up all patients for two or more years.

The age of onset of esotropia was established according to the history supplied by the parents. The ophthalmologic findings and clinical course are recorded in Table 1. A cycloplegic refraction was performed at the initial examination 40 minutes after instillation of one drop of 2% cyclopentolate HCl or 30 minutes after instillation of the combination of one drop of 1% cyclopentolate HCl and one drop of 1% tropicamide.

Treatment was invariably instituted at the first examination. The cycloplegic refraction finding was prescribed as the initial spectacles. The miotic prescribed was either one drop of 0.125% echothiophate or instillation of 0.025% isoflurophate ointment once daily in each eye.

A high accommodative convergence ratio was identified by the near esotropia exceeding the distance esotropia by 10 prism diopters. The nature of the accommodative convergence ratio was declared either to be normal or high, while 20 of the 21 patients wore their full hypermetropic spectacle correction. One patient, with a refraction of +0.50 diopter, had a high accommodative convergence ratio although glasses were not prescribed. Classification was made at an age when reliable near and distance measurements were obtainable, before any surgery was performed, and before the age when the accommodative convergence ratio would tend to become normal.

If the esotropia that was initially eliminated by antiaccommodative treatment returned, another cycloplegic refraction was done. New lenses were prescribed if increased hypermetropia was detected. This procedure was repeated at any time a deterioration in control of the esodeviation was seen.

Where deterioration to a constant esotropia occurred while fixing at 6 meters and no increased hypermetropia was detected, then recession of the medial rectus muscles was performed. Patients with nystagmus, motility restriction, unilateral visual anomaly, evidence of a palsied muscle, and esotropia associated with brain damage or dysfunction were excluded.

Results

The age of onset of esotropia varied from birth to 11 months with an average age of 4.6 months. The age of initial examination varied from 3 to 12 months with an average of 8.1 months, indicating an average delay of 3.5 months between age of onset and initial treatment.

The esotropia was intermittent on initial examination in 15 of the patients and constant in six. Only one patient initially had a constant distant esotropia greater than 40 prism diopters. This patient had a +7.00 diopter refraction.

Table 1 separates the 11 patients with a normal accommodative convergence ratio from the ten who had a high accommodative convergence ratio. Using the spherical equivalent of the least hyperopic eye, the hypermetropia in the normal accommodative convergence ratio group ranged from 2 to 7 diopters with an average of 4.5 diopters. The hypermetropia in the high accommoda-

tive convergence ratio group ranged from 0.5 to 4.5 diopters with an average of 2.3 diopters.

Thirteen patients received hyperopic spectacles at the initial examination and eight received a miotic. Glasses replaced miotics as treatment in seven of these patients. Six patients eventually wore bifocals, although none wore them initially.

Eleven patients' eyes remained aligned with antiaccommodative therapy alone. Some patients who initially received glasses had a miotic added to the spectacle therapy in addition to re-refraction; we exerted every measure to control accommodation in an effort to stop the development of an increasing nonaccommodative esodeviation component. Despite this effort, ten patients' accommodative esodeviation control deteriorated, and they eventually required surgery. The accommodative convergence ratio was normal in five of these patients with deteriorated accommodative esotropia and high in the others. Two patients, one each with a normal and a high accommodative convergence ratio, required a second operation for consecutive exotropia. The average age of deterioration in the five patients with normal accommodative convergence ratios was 22.4 months, with the average interval between onset of esotropia and deterioration being 17.4 months. Surgery was performed on these five patients at an average age of 26 months, indicating a lag of 3.6 months between deterioration and surgical correction. Deterioration occurred at a later age in the five patients with high accommodative convergence ratios. The average age at deterioration was 40.4 months with the average interval between onset of esotropia and deterioration being 34.4 months. Surgery in this group was performed at an average age of 46.6 months, indicating a lag time of six months between deterioration and surgical correction.

The horizontal alignment of all the patients' eyes at their last examination was within 10 prism diopters of orthophoria as determined by simultaneous prism and cover test for distance fixation. Twenty patients still required glasses to control their accommodative esotropia; of these, six required bifocals. Patients 11 and 14 manifested dissociated vertical deviation during the cover test. All patients in this study maintained good vision in each eye, but 13 required occlusion therapy to combat amblyopia.

Sensory testing was attempted in all patients, but failed in 13 because they were too young to give consistently adequate subjective responses. Of the eight patients successfully tested, seven fused the near Worth 4 dots at one-third meter, indicating peripheral fusion. In six of these seven patients with peripheral fusion, stereoacuity ranging from 67 to 400 seconds of arc was shown. A Polaroid vectographic test was used in testing stereopsis. One patient of the eight who were testable (Patient 14) showed no binocularity.

Discussion

Differentiation of congenital and acquired esotropia after age 1 year is often impossible because of the parents inability to remember, which results in inaccurate recall of the age of onset, characteristics of the initial deviation, and details about its clinical course. A parental history that clearly describes a large constant angle of esotropia unchanged since first noted a few days or weeks after birth and confirming ophthalmologic examination no later than 6 months of age, although not absolute, are the ideal stipulations for accurately diagnosing congenital esotropia. However, a later

TABLE 1

SUMMARY OF EARLY ONSET ACCOMMODATIVE ESOTROPIA PATIENTS*

Patient No.	Age at Onset By History (mos)	Age at First Examination (mos)	Initial Alignment	Initial Refraction	Treatment	Interval Between Onset and Deterioration to Constant ET (mos)	Horizontal Surgery†	Age at Surgery (mos)	Follow-Up (yrs)	Final Alignment
				NORMAL ACCOMMODATIVE CONVERGENCE						
1	11	12	ET = 30	+3.75	Gls	7	Rec. MR	22	18	Ortho c
			ET′ = 40	+3.75	Surg., Occl.	—	Res. LR	38		Ortho′c
2	6	9	ET = 60	+7.50	Gls	—	—	—	3	Ortho c
			ET′ = 60	+7.00	—	—	—	—		Ortho′c
3	8	11	ET = small	+6.00	Gls	—	—	—	3	Ortho c
			ET′ = small	+5.00	—	—	—	—		Ortho′c
4	8	12	ET = 30	+6.00	Gls	10	Rec. MR	25	7	Ortho s
			ET′ = 30	+5.00	Surg., Occl.	—	—	—		XT′s = 8
5	3	4	E(T)′ =	+2.50	Gls, PI	9	Rec. MR	15	2.5	ETc = 4
			Variable	+2.50	Surg., Occl.	—	—	—		ET′c = 4
6	2	6	E(T)′ = 60	+5.50	Gls	45	Rec. MR	50	5.5	Ortho c
				+5.50	Surg.	—	—	—		ET′c = 10
7	9	12	ET = 10	+4.75	Gls, Occl.	—	—	—	3.5	Ec = 3
			E(T)′ = 30	+6.75	—	—	—	—		E′c = 4
8	5	11	E(T)′ = 45	+6.00	Gls, Occl.	—	—	—	3	Ortho c
				+5.25		—	—	—		E(T)′c = 8
9	1	3	E(T)′ = 40	+2.00	Gls, PI	—	—	—	3	ETc = 2-8
				+2.00	Surg., Occl.	—	—	—		ET′c = 2-12
10	0	4	E(T)′ = 40	+4.75	Gls, Occl.	—	—	—	2	Ec = 5
				+5.00	—	—	—	—		E(T)′c = 3-8
11	0	5	ET = 30	+4.25	Gls, PI	16	—	—	2	ETc = 5-10
			ET′ = 35	+4.25	Surg., Occl.	—	Rec. MR	18		ET′c = 6-10

HIGH ACCOMMODATIVE CONVERGENCE

12	5	9	E(T) = 30 E(T)′ = 30	+2.25 +2.25	Gls, PI Surg., Occl.	17 —	Rec. MR —	25 —	3.5	ETc = 8-12 ET′c = 10-25 ET′BF = 8-12
13	3	8	E(T)′ = 60	+1.00 +1.00	Gls, PI Surg., Occl.	61 —	Rec. MR —	72 —	13	ETc = 4 ET′c = 4
14	7	9	Ortho E(T)′ = 25	+2.00 +1.75	Gls Surg.	63 —	Rec. MR Rec. LR	79 88	14	ETc = 10 Ortho c
15	3	10	ET′ = 25	+3.50 +4.00	Gls	— —	— —	— —	2.5	ETc = 8 E(T)′c = 25 E′BF = 8
16	6	12	Ortho ET′ = 40	+1.25 +1.25	DFP, Gls Surg.	26 —	Rec. MR —	41 —	15	Ortho c ET′c = 35 ET′BF = 12
17	1	6	E(T)′ = 30	+3.00 +3.00	DFP, Gls. —	— —	—	— —	15	ETc = 6 ET′c = 10
18	9	10	E(T)′ = small	+0.50 +0.50	DFP, Surg. —	5 —	Rec. MR Rec. MR	16 114	15	Ortho c Ortho′c
19	0	4	E(T)′ =35	+3.25 +3.00	Gls, Occl —	— —	— —	—	2.5	Ec = 6 E(T)′c = 25 E(T)′BF = 6
20	9	10	E = 10 E(T)′ = 40	+4.50 +5.00	DFP, Gls Occl.	— —	—	—	17	Ec = 6 ET′c = 16 E BF = 6
21	0	3	ET′ = 70	+2.50 +2.25	Gls, PI Occl.	— —	— —	— —	3.5 —	E(T)c = 4-25 E(T)′c = 4-35 E(T)′BF = 4-25

*E designates esophoria; E′, esophoria at ⅓ meter; ET, esotropia at 6 meters; ET′, esotropia at ⅓ meters; E(T), intermittent esotropia; E(T)′, intermittent esotropia at ⅓ meter; BF, bifocal segment; XT′, exotropia at ⅓ meter; c, with spectacle correction; s, without spectacle correction; Ortho, straight eyes on alternate cover test at 6 meters; Ortho′, straight eyes on alternate cover test at ⅓ meter; Res, resection of extraocular muscles; Rec, recession of extraocular muscles; MR, medial rectus muscle; LR, lateral rectus muscle; Gls, glasses; Surg., surgery; Occl., occlusion; PI, phospholine iodide (echothiophate); and DFP, diisopropyl flurophosphate (isoflurophate).

† Both eyes.

age at initial examination, or any question in the parents' minds concerning date of onset or variability of the deviation, makes the diagnosis of congenital esotropia suspect.

Congenital esotropia is often confused with infantile facial features that cause pseude-esotropia. The prominent epicanthal folds, wide nasal bridge, and reduced amount of nasally visible sclera often make it difficult for the untrained observer to be sure of the child's true ocular alignment. Therefore, when their nonstrabismic child begins gradually to manifest an acquired esotropia, the parents may give a history that suggests congenital esotropia. The inaccurate history supplied by the parents is possible regardless of the patient's age when first examined, but the recognition rate of this error by an adequately trained person is inversely related to the patient's age at the initial examination.

Acquired esotropia secondary to a unilateral visual anomaly usually begins at approximately 6 months of age. Significant anisometropia, optic nerve anomaly, retinal disease, or ocular media opacities are all possible causes. These patients with esotropia usually have a variable angle of deviation diagnosed by complete ophthalmologic examination, and as a group tend not to be confused by the ophthalmologist with those who have congenital esotropia.

The 21 patients in this study closely resemble the 667 patients with accommodative esotropia in an earlier study[1] with respect to similar distribution of normal and high accommodative convergence ratios and the average hypermetropia refraction associated with each ratio (Table 2). In contrast, a previous report[4] showed that 85% of congenital esotropia patients had a normal accommodative convergence ratio and 15% had a high accommodative convergence ratio. An additional report shows that 88% of patients with congenital esotropia followed up through 4 years of age had a normal accommodative convergence ratio and 12% had a high accommodative convergence ratio (unpublished data). These data support the finding that most of our patients had early-onset accommodative esotropia and not congenital esotropia. Further, the esotropia in all patients initially was controlled at 6 meters and one-third meter with antiaccommodative therapy, and 95% still required spectacles to keep their eyes aligned when last examined.

Only patients 11 and 21 had the combination of a congenital onset of the esotropia by history and a constant angle of esotropia at the initial examination. The other two patients who had congenital deviations had variable deviation angles, according to their histories. All but one of the remaining six patients who had

TABLE 2

COMPARISON OF THE RELATIONSHIP BETWEEN ACCOMMODATIVE CONVERGENCE RATIO AND REFRACTION (IN TWO STUDIES OF PATIENTS WITH ACCOMMODATIVE ESOTROPIA)

Studies	No. of Patients	Accommodative Convergence	Refraction
Previous study by Parks[1]	289	Normal	+4.75
	378	High	+2.25
Present study	11	Normal	+4.50
	10	High	+2.25

a constant deviation angle at initial examination had hypermetropia ranging from 3.50 to 7 diopters (average, +5.00). The range of hypermetropia among the 13 patients with variable angles was 0.5 to 5.5 diopters (average, +3.00). This suggests that most patients with larger degrees of hypermetropia are prone to adopt a pattern of constantly accommodating to maintain good vision, and consequently have fixed deviation angles.

Patient 21 is an exception to this general rule. On initial examination this patient showed a large constant angle of esotropia and a small to moderate amount of hyperopia (+2.25). Initial miotic therapy caused a reduction and a markedly variable angle of esotropia, but not elimination of the deviation. Because of this change in the deviation with miotic therapy, spectacles were prescribed. Spectacle therapy resulted in a well-controlled esodeviation as measured by simultaneous prism and cover test. At age 14 months, the true nature of this patient's high accommodative convergence ratio surfaced and he subsequently has worn bifocals with no surgery ever being performed. Failure to investigate the possible cause of accommodative convergence of this patient's deviation would have resulted in an incorrect diagnosis.

Some of the patients included in this study may have had small-angle congenital esotropia that deteriorated into a larger deviation during the first year of life. Patients 11 and 14 are thought to have essentially congenital esotropia, because of their dissociative vertical deviation and also because Patient 14 had an absence of binocularity. These findings are more indicative of a diagnosis of congenital esotropia than accommodative esotropia, although an accommodative element may have accounted for the increase in their deviation angle. The difficulty associated with accurately diagnosing the cause in all infants with esotropia is shown in these two patients.

Another frustration associated with the management of the patient with early-onset accommodative esotropia is the approximately 50% prognosis for gradual development of a superimposed nonaccommodative esotropia component. The accommodation control that initially contained the esodeviation decompensates, the esophoria deteriorates into esotropia, and the case becomes a complex mixture of accommodative and nonaccommodative esotropia. Surgery then becomes a necessity to restore the alignment of the eyes to the status that prevailed before the deterioration. The breakdown in the ability of the antiaccommodative therapy to control the esodeviation occurs on the average from 1½ to three years after treatment has begun. The threat of deterioration requires careful observation for many years after an initial success in reversing the esotropia with glasses or miotics.

The risk of amblyopia occurring in the patient with early-onset accommodative esotropia is greater than 50%. Because these children do not bifixate, despite their eyes being straight after beginning antiaccommodation therapy, only the chance that both eyes will alternately monofixate rather than exclusively fixate with the same eye determines whether amblyopia will occur. The absence of bifixation among patients with early-onset accommodative esotropia is a characteristic shared by patients with congenital esotropia. In this regard the groups are similar.

In addition to the clinical characteristics of intermittent and varied angle size deviation in an esotropic patient less than 1 year of age, if the refraction is as low as +2.25, despite the deviation angle being constant, accommodative esotropia should be suspected and antiaccommodation therapy should be instituted.

Summary

Twenty-one patients had esotropia diagnosed before age 12 months. In all of these patients esotropia was eliminated, at least initially, by antiaccommodative therapy consisting of either full hypermetropic spectacles or miotics, or both.

Clinical findings evaluated in these patients were comparable to similar clinical factors studied in a larger series of patients with accommodative esotropia, most with onset after 12 months of age.

In approximately 50% of these patients early-onset accommodative esotropia deteriorated to a nonaccommodative esotropia that required surgery despite aggressive antiaccommodative therapy. All but one of the surgically treated patients continued to need hypermetropia spectacles postoperatively to maintain alignment.

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MEASUREMENT OF THE RELATIVE AFFERENT PUPILLARY DEFECT IN RETINAL DETACHMENT

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A relative afferent pupillary defect usually indicates disease in the prechiasmal visual pathway.[1-7] An afferent pupillary response is characterized by the following: (1) constriction of both pupils when a light stimulus is applied to the normal eye; and (2) dilatation of both pupils when the light stimulus is transferred to the affected eye. With a mild afferent pupillary defect there is a small initial constriction of both pupils, followed by pupillary dilatation (pupillary escape) when the light stimulus is applied to the affected eye. Usually there is no anisocoria.

Widespread retinal disease, including retinal detachment, can produce an afferent pupillary defect.[4,8,9] Our study was designed to quantitate the pupillary responses in patients with rhegmatogenous retinal detachment and to assess the changes after scleral buckling surgery.

SUBJECTS AND METHODS

Patients with unilateral retinal detachment involving the macula were selected consecutively based on the following criteria: (1) no previous ocular surgery in either eye; (2) no dilating drops applied to either eye for 24 hours before examination; and (3) no coexisting ocular disease in either eye that might influence visual function.

Ages ranged from 11 to 83 years with a median age of 66 years. Duration of macular detachment could not be determined in three patients, but was five days or less in five patients.

Afferent pupillary responses were quantitated with neutral density filters, which were calibrated in log units of light attenuation and percentage of light transmission.[10,11] The swinging flashlight test was performed, while interposing filters between the light stimulus and the normal eye. The depth of the defect was recorded from the filter, which eliminated the pupillary escape.

Three months after surgery, the pupillary responses were remeasured and compared to the preoperative values (Figure).

RESULTS

Ten patients with retinal detachment and an associated relative afferent pupillary defect were examined by the swinging flashlight test and neutral density filters. Preoperatively, the depth of the defect ranged from 0.3 to 1.4 log units. The retina was reattached in nine patients; seven recovered normal pupillary reactions. Two patients with postoperative macular disease had persistent afferent defects, despite anatomically successful surgery. Two other patients with macular changes recovered normal pupillary responses. One patient with a persistent retinal detachment after sur-

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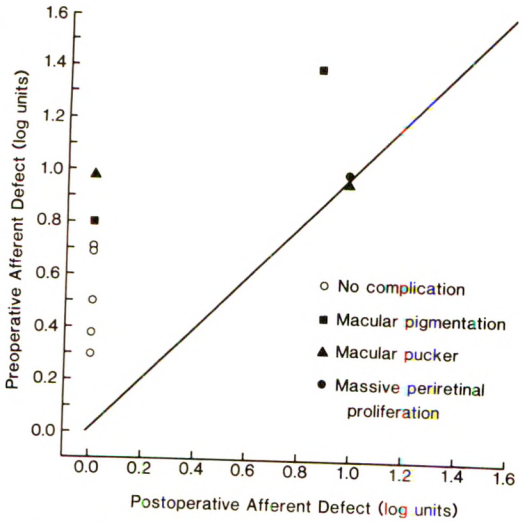


Figure (Bovino and Burton). Comparison of preoperative and postoperative relative afferent pupillary defects in retinal detachment patients. The swinging flashlight test was used with neutral density filters calibrated in log units of light attenuation. Data points above the diagonal line indicate improvement in pupillary responses.

gery showed no change in pupillary response.

Discussion

Pupillary defects secondary to retinal disease are similar to those produced by optic nerve lesions.[12] Apparently ophthalmoscopic examination has diminished the significance of abnormal pupillary reactions associated with retinal disease.[13] Frequently pupils are dilated pharmacologically to expedite diagnosis of retinal disease, and subtle pupillary signs go unobserved. A high proportion of aphakic patients with retinal detachments have surgically induced pupillary alterations, which impair accurate assessment of pupillary function.

In a study of anterior segment changes induced by scleral buckling surgery, some eyes developed sluggish pupillary reactions, attributed to poor postoperative retinal function.[14] The preoperative pupillary status had not been studied. Another study showed parallel improvement in thresholds for light and pupillary response after retinal reattachment, although there was no discussion of relative afferent defects.[15]

The frequency of afferent pupillary defects in a large retinal detachment population is unknown. Our clinical experience indicates that abnormal pupillary responses are uncommon in peripheral detachments, but occur in approximately one half of the detachments involving the macula. Postoperative visual acuity is predictably lower in patients with detachments which include the macula.[16] Further study is necessary to determine whether an afferent pupillary defect provides additional predictive information on recovery of visual acuity after retinal detachment surgery.

Summary

A swinging flashlight test and calibrated neutral density filters were used to quantitate the depth of relative afferent pupillary defects in ten patients with retinal detachment. Postoperatively, the pupillary responses returned to normal in seven of nine patients with anatomically successful surgery.

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ACCOMMODATION DEFECT IN WILSON'S DISEASE

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Wilson's disease is a hereditary disorder of copper metabolism, characterized by a progressive degeneration of the central nervous system, and associated with hepatic cirrhosis. The neurologic syndrome frequently includes difficulty speaking, swallowing, and chewing. A characteristic tremor of the wrists and shoulders is increased on voluntary action. Rigidity is common. Inappropriate laughing or crying is seen.

Ocular findings include a pigmented corneal ring, which is almost always present in cases with neurologic involvement (97%), and pigment under the lens capsule (17%).[1] Wilson,[2] Goldberg, and von Noorden,[3] and Walsh and Hoyt[4] all commented on the absence of involvement of ocular muscles. Walsh and Hoyt[4] stated that this was "most remarkable in view of the nature of the pathologic changes." Occasional references have been made to jerky oscillations of the eyes, involuntary upgaze, paresis of upgaze, and slowed saccadic movements.[5-7]

Wiebers, Hollenhorst, and Goldstein[1] recently reported ophthalmologic abnormalities in 53 patients with Wilson's disease and emphasized the presence of corneal rings and the absence of other ophthalmologic signs (such as nystagmus, cranial nerve palsies, and other movement disorders). One of their patients had "paralysis of accommodation and ataxic movements of the eyes."

We describe a patient with Wilson's disease who complained of difficulty reading and who had markedly reduced amplitude of accommodation.

CASE REPORT

A 22-year-old man was referred here for evaluation of suspected Wilson's disease. He had noted the onset of upper extremity tremor in October 1977. He had a one-year history of mild dysarthria, and personality and behavioral changes. There was a family history of psychiatric disorders. The patient was receiving no medications at the time of examination and had received no tranquilizers, sedatives, antidepressants, or any other medications during the preceding several weeks. Physical examination in April 1978 showed slurred, ataxic speech, mild "wing-beating" tremor, some coarsening in the finger-to-nose testing and difficulty with tandem gait. Liver scan showed an enlarged liver, the serum ceruloplasmin was less than 5 mg/dl (normal, 21 to 43 mg/dl); serum copper was 40 μg/dl (normal, 80 to 100 μg/dl); and 24-hour urine copper was 476 μg (normal < 30). At the time of his first ophthalmic examination in April 1978, he described a two-year history of blurred near vision. Uncorrected visual acuity was 6/6 (20/20) in both eyes, but the patient could only read 6/30 (20/100) near equivalent (J 10) at 32 to 44 cm. Manifest refraction showed the patient to be slightly hyperopic in both eyes: R.E.: $+1.00 + 0.25 \times 90$ degrees; and L.E.: $+1.00 + 0.25 \times 90$ degrees. This was later confirmed with cycloplegic examination using 1% tropicamide.) Accommodative range measured by Prince rule was 2.5 diopters in the right eye and 3.0 diopters in the left eye with correction. Pupillary reactions to light and near stimulus were normal. The patient was orthophoric at distance, but there was a five-prism diopter exophoria at near with a near point of convergence measured at 25 cm. Saccadic movements were mildly hypometric, and there was microsaccadic pursuit and upbeating nystagmus on upgaze. Slit-lamp examination confirmed the presence of a Kayser-Fleischer ring. There was no cataract. Results of examination of the retina were normal. Therapy was initiated with penicillamine, a low copper diet, potassium sulfide, and pyridoxine HCl.

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Discussion

Goldberg and von Noorden[3] emphasized the "remarkable normality of complex oculomotor functions in patients with Wilson's disease." However, they did not report any attempts to measure accommodative amplitude in their patients. Wiebers, Hollenhorst, and Goldstein[1] found one of their patients, who complained of inability to read, to have "paralysis of accommodation," but apparently did not test accommodation in any of the other patients. Heine[8] reported a case of internal ophthalmoplegia but did not measure accommodation.

The neurologic pathways subserving accommodation begin in the retina and afferent fibers and travel up the optic nerve, chiasm, and tracts. They are relayed in the lateral geniculate body where a further neuron travels to the calcarine cortex; there, a relay is made to the prestriate area. From this region, the reflex pathways travel down the internal corticotectal tract to the diencephalic region, where a connection is made with the caudal portion of the parasympathetic component of the oculomotor nuclear complex, the Edinger-Westphal nucleus. From the Edinger-Westphal nucleus the afferent path travels down the oculomotor nerve, and relays in the ciliary ganglion from which fibers reach the pupillary sphincter and ciliary muscles.[9]

Many diseases diminish accommodation.[9,10] Ocular problems such as iridocyclitis, congenital glaucoma, and trauma may do so by injuring the ciliary body. A variety of drugs and toxic chemicals cause cycloplegia. Myotonic dystrophy and myasthenia gravis may have a minimal effect on accommodation. Botulism, syphilis, tuberculosis, and leprosy can cause paralysis of accommodation by injury to the oculomotor nerve or nucleus. Viral disease such as mumps, hepatitis, herpes zoster, poliomyelitis, and encephalitis occasionally disturb accommodation, as do diabetic and alcoholic neuropathy. Pupillary and ciliary musculature may be paretic in the ophthalmoplegic form of acute idiopathic polyneuritis. Lesions of the tectal region, such as pinealomas and astrocytomas, commonly cause supranuclear vertical eye movement problems and may interfere with accommodation. Acute hemorrhagic anterior polio encephalitis of Wernicke is sometimes associated with accommodative paralysis. Finally, cerebral concussion and craniocervical extension injuries may be followed by symptoms of accommodative disturbance.

Pathologic changes in Wilson's disease are most prominent in the corpus striatum. However, degeneration may be present in the diencephalon and upper brainstem. The thalamus, red nucleus, and dentate nucleus are occasionally involved.[11] Greenfield[12] described shrinkage of the corpus striatum involving the putamen and caudate nucleus bilaterally. Microscopic examination also showed degeneration in the tracts leading from the putamen to the red nucleus and the corpus luysii, and to the posterior longitudinal bundles and superior cerebellar peduncles.

In a normal individual, accommodative amplitude decreases with age from about 12 diopters at age 8 years to 2 diopters at age 50 years.[13] Emmetropic individuals usually develop symptoms of accommodative weakness between the ages of 40 and 45 years. Our patient, at age 22 years, would be expected to have an amplitude of accommodation between 8 and 12 diopters,[13] but he had only 2.5 to 3 diopters. He had had symptoms of deficient accommodation lasting several months. There was no evidence of any other disorder that might explain his accommodative weakness, and he had not received any known cycloplegic agents before our examination.

However, primary insufficiency of accommodation is occasionally seen in young adults and cannot be absolutely excluded here, although we consider it quite unlikely.

Our patient had signs of extensive disease in the mesencephalon and brainstem tegmentum. These included the characteristic tremor, dysarthria, gait disturbance, hypometric saccades, microsaccadic pursuit movements, and diminished convergence in addition to a reduced amplitude of accommodation. However, normal ductions and normal pupillary reactions suggest that the oculomotor nuclear complex was spared. Other authors[3] have suggested that disturbance of ocular motility and the near reflex might be found in Wilson's disease where descending pathways from the occipital cortex could be interrupted in the diencephalon or mesencephalon. We agree, and we believe that the clinical findings in our patient point to a supranuclear defect in accommodation. To the best of our knowledge there are no previous published reports of measurements of accommodation in Wilson's disease.

We believe a significant percentage of patients with Wilson's disease may have a disturbance of accommodation, which is overlooked because the patient is young enough to have a large accommodative reserve or is so neurologically disabled that a careful visual system examination is not possible. We suggest that measurements of accommodation should be part of the evaluation of suspected Wilson's disease whenever clinically possible.

Summary

A 22-year-old man with Wilson's disease had blurred vision caused by a defect of accommodation that we believed to be supranuclear in origin.

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OCULAR TOXOPLASMOSIS AND VISUAL FIELD DEFECTS

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Toxoplasma gondii is the most common cause of primary retinochoroiditis, and has been estimated to account for as much as 50 to 75% of cases in some series.[1,2] The drugs currently available for its treatment are highly toxic or have a number of serious side effects.[1-5] Because of the complications of these drugs, it has been necessary to consider carefully which lesions require treatment and which do not. At the present time there are four generally accepted indications for treatment. These include active lesions of the macula or papillomacular bundle, those threatening or involving the optic nerve, and those severe enough to cause massive vitreous reaction, with subsequent contracture and possible retinal detachment.[4]

We noticed, however, that several patients with peripapillary lesions not affecting the disk or papillomacular bundle had substantial visual field loss. The presence of a sector scotoma resulting from toxoplasmosis has been recognized,[6] but the extent of this problem has not been examined. Therefore, we undertook a study to determine the clinical significance of these visual field changes and whether this constitutes an indication for treatment.

SUBJECTS AND METHODS

The records of the Retina Service here were reviewed and those patients who had lesions characteristic of either acute or inactive peripapillary ocular toxoplasmosis were recalled and had Goldmann perimetry performed in each eye. Criteria for inclusion consisted of the presence of fluffy yellow-white lesions of the retina, with overlying vitreous cells in the acute phase or the classic appearance of a yellow retinochoroidal scar, or both, with surrounding pigment proliferation, in the inactive stage. All lesions were located within two disk diameters of the optic nerve head. The fluorescent antibody test to toxoplasmosis was performed in most cases and those cases with negative results or questionable diagnosis on the basis of clinical examination were excluded. All patients were screened for increased intraocular pressure, glaucomatous optic disk changes, or evidence of other lesions that might produce visual field defects.

A total of 34 patients were examined, 30 with inactive peripapillary lesions, and four with active peripapillary lesions of toxoplasmosis. Although several patients had evidence of disease in both eyes, no bilateral peripapillary lesions were seen.

RESULTS

All patients with peripapillary lesions showed scotomas with sloping borders that resulted from disruption of the nerve fiber layer and interruption of peripheral nerve fibers (Fig. 1). The nearer lesions of similar size were located

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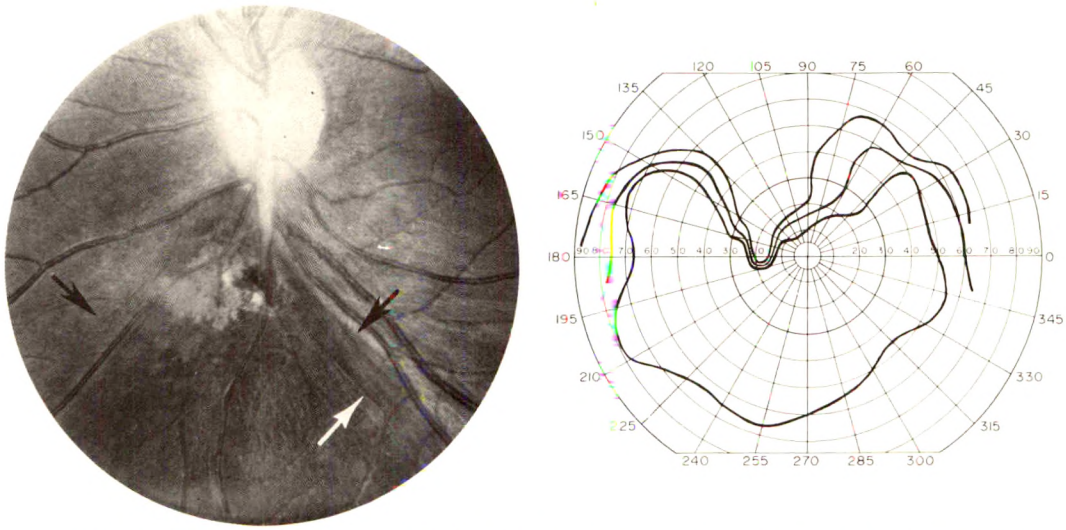


Fig. 1 (Martin and associates). Left, Left fundus of a patient with an inactive peripapillary toxoplasmosis scar located inferior to the optic disk. Prominent absence of the nerve fiber secondary to disruption from the lesion can be seen and the boundaries of the defect are noted (black arrows). Along the temporal border of the defect is a possible area of relative, rather than absolute, loss of the nerve fiber layer (white arrow). Right, Goldmann visual field of the fundus shown at left. A superior nerve fiber layer defect with sloping borders is present. These sloping borders may correspond to areas of relative absence of the nerve fiber layer, such as seen at left.

to the disk, the greater the area of visual field loss. Small isolated lesions further from the disk produced arcuate defects, and larger lesions or small lesions close to the disk were noted to cause sector defects (Fig. 2). In two cases one half of the visual field was destroyed (Fig. 3).

Sector optic atrophy was seen in five cases and corresponded to the areas of nerve fiber layer loss. Although usually mild, in one case the pallor was sufficiently prominent to produce a pseudo-

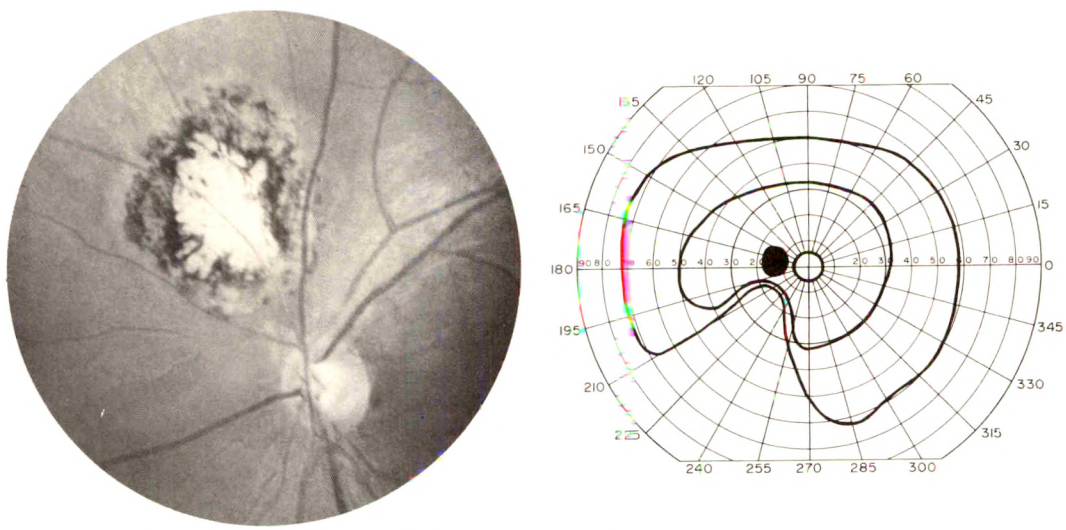


Fig. 2 (Martin and associates). Left, Left fundus with a chorioretinal lesion secondary to toxoplasmosis. Right, Goldmann visual field of left fundus shows a sector defect, almost quadrantic in configuration, caused by the lesion. Visual acuity was 6/6 (20/20) in the eye. Note that while the field defect is similiar in size to that shown in Figure 1, the lesion at left is larger. The closer in proximity a lesion is located to the disk the more corresponding nerve fiber layer is lost.

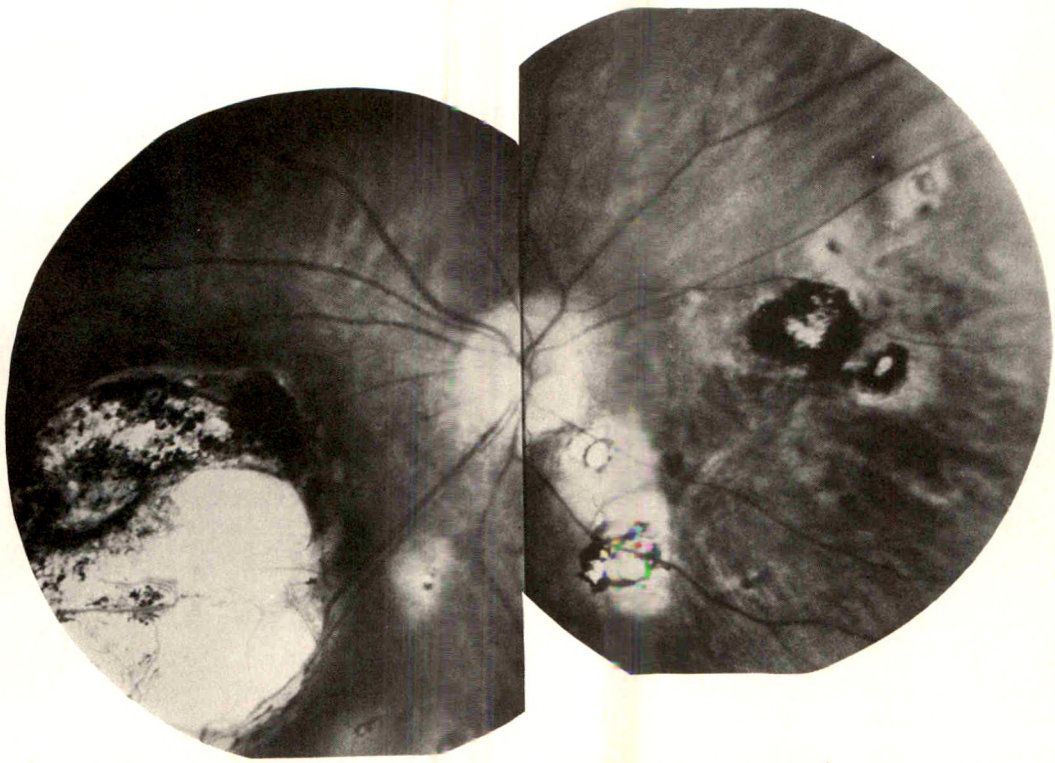
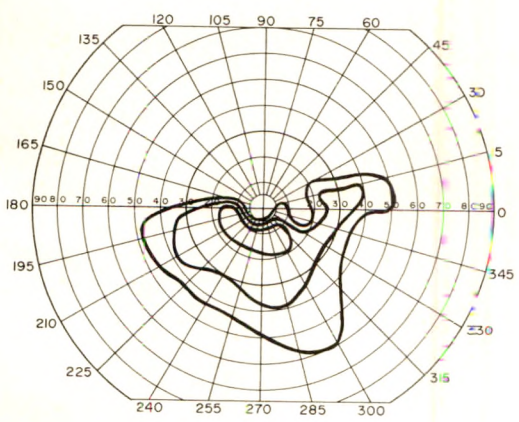


Fig. 3 (Martin and associates). Right fundus of a 37-year-old patient with bilateral congenital ocular toxoplasmosis and congenital nystagmus. A large macular chorioretinal scar is present, as well as multiple smaller scars inferior and nasal to the disk. The scars have interrupted most of the nerve fiber layer to the inferior retina and produced the field defect seen in Figure 4.

ischemic optic neuropathy appearance (Fig. 4).

One case was observed in the acute phase of the disease, and then again 2½ months later. The field defect was noted to enlarge slightly over this period of



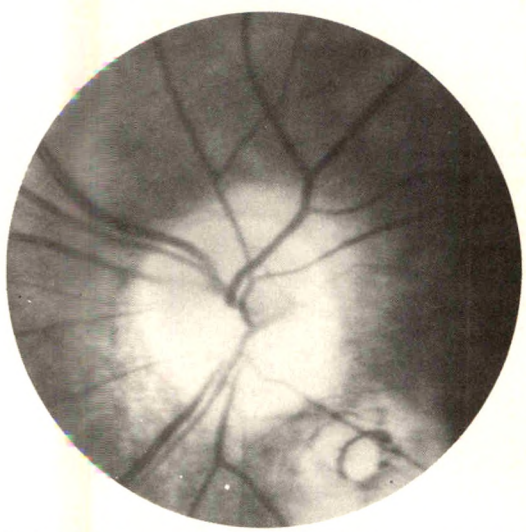


Fig. 4 (Martin and associates). Left, Goldmann visual field of a 37-year-old patient (Fig. 3) shows a superior altitudinal defect, such as can be seen with ischemic optic neuropathy. Right, Pallor of the inferior one half of the nerve head of the same patient is visible and is probably related to interruption of nerve fibers extending from the retinal ganglion cells to the lateral geniculate body.

time as more of the nerve fiber layer was interrupted.

Discussion

The results of this study indicate that even small peripapillary toxoplasmosis lesions can produce extensive visual field defects. As similar-sized lesions more closely approach the disk, the field defects become correspondingly larger.

The explanation for these findings is in the histopathologic examination of eyes with toxoplasmosis. Characteristically, *T. gondii* in the posterior segment produces a segmental area of panophthalmitis, with destruction of the nerve fiber layer.[7] This is not the case in choroiditis, such as the presumed ocular histoplasmosis syndrome, in which the inner retinal layers may be left relatively intact.[8] As expected from histologic findings, toxoplasmosis scars produced not only focal scotomas, but also nerve fiber layer defects. Although the chorioretinal lesions usually have well-delineated margins, the borders may contain chronically inflamed, but non-necrotic, retina.[7] Possibly the nerve fiber layer within this border tissue may function only partially in the acute and quiescent stages and account for the sloping borders of the scotomas. An abrupt demarcation between a viable and a nonviable retina would be expected to produce absolute defects, without relative defects along the borders. Such defects were not seen in this study.

The presence of disk pallor corresponding to the area of the nerve head that normally received the interrupted nerve fibers from the retina varied from mild to marked. Possibly, interruption and subsequent degeneration of nerve fibers extending from the retinal ganglion cells to the lateral geniculate body accounts for this phenomenon. Although primary involvement of the disk may occur in cases with juxtapapillary chorioretinitis, this is not as likely to be the situation with peripapillary lesions, as in our patients, not located directly adjacent to the nerve.

Hogan and associates[9] noted that eyes with peripapillary toxoplasmosis lesions alone appear to comprise about 8% of cases of posterior segment involvement seen with the disease. Among all toxoplasmosis cases with retinal involvement they found about 20% to have peripapillary lesions, either alone or in conjunction with a lesion elsewhere.

Treatment has been advocated in acute ocular toxoplasmosis for those foci involving the macular region, particularly the papillomacular bundle, or threatening the optic nerve. In those cases with sufficient vitreous reaction to lead to contracture and resultant retinal detachment, therapy also has been recommended.[4] Varied therapeutic treatment regimens have been suggested,[1-5,10] and the best will probably be determined by a well-controlled clinical study. It has been stated that peripapillary lesions of toxoplasmosis should receive treatment,[2] and we emphasize this point to show the severe irreversible damage to the visual field that these lesions may produce. Early aggressive treatment may prevent an increase in size of the lesion, an increase which even if small, may produce a substantially larger visual field defect.

Summary

We studied 34 patients with peripapillary lesions secondary to toxoplasmosis. All patients had visual field defects corresponding to interruption of the nerve fiber layer of the retina. The extent of visual field loss increased with proximity to the disk.

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Fifty years ago this month in THE JOURNAL

Not very long since, Gonin, after a relatively silent interval of several years (see Gradle's paper in THE AMERICAN JOURNAL OF OPHTHALMOLOGY, April, 1930), again claimed an important percentage of successes with his method of ignipuncture. Other workers have been less satisfied as to the value of this form of treatment. But it must be admitted that the technique of the procedure is still young and is perhaps rather vaguely understood by those who have had only limited experience in its application.

W. H. Crisp, "Retinal detachment"

Am. J. Ophthalmol. 13:628, 1930

LACTOFERRIN IN HUMAN OCULAR TISSUES

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The natural protective factors identified in normal human tears may be categorized as specific (antibody,[1,2] lymphocytes[3,4]) or nonspecific (phagocytic cells,[4,5] lactoferrin,[6] lysozyme,[7] nonlysozyme antibacterial factor,[8] interferon[9]). Nonantibiotic approaches to the treatment of human external ocular infections could result from augmentation or manipulation of these factors.

One of the natural protective factors present in tears, lactoferrin, which is an iron complexing protein, was identified in bovine milk in 1939,[10] in human milk in 1951,[11] and in human tears in 1966.[6] Lactoferrin has both bacteriostatic[12] and bactericidal[13] properties, and may interact with specific antibody to produce an antimicrobial system more powerful than either lactoferrin or specific antibody alone.[14] Additionally, lactoferrin has been shown to affect complement activity in vitro.[15] Thus, it may be important in the natural defense of the ocular surface.

Broekuyse[16] confirmed the presence of lactoferrin in human tears in 1974. No postulates as to its origin were offered, but Franklin, Kenyon, and Tomasi[17] had noted immunofluorescent staining for lactoferrin along the apical border of lacrimal acinar epithelial cells. Because the normal plasma lactoferrin level is 0.0015 mg/ml,[18] and the normal tear lactoferrin level is 1.45 mg/ml,[16] local lactoferrin production at or near the ocular surface is likely. Evidence from immunohistologic studies of other secretory tissues (the bronchial submucosal glands,[12] the submaxillary gland,[19] and the pancreas[20]) suggests that the acinar epithelium of the lacrimal gland may be a local source of tear lactoferrin. Accessory lacrimal tissue, which is histologically similar to the main lacrimal gland, is another likely source of tear lactoferrin, and the finding of secretory granules in normal conjunctival epithelium[21] makes the conjunctiva another possibility.

The study reported herein was undertaken to identify, by an immunofluorescence technique, the sources of human tear lactoferrin.

MATERIAL AND METHODS

Autopsy specimens—Lacrimal tissues were obtained at autopsy from seven cadavers, age 54 to 73 years, with no known ocular disease (Table). The postmortem interval before enucleation varied between nine hours and five days. The globes were enucleated by peritomy close to the corneoscleral limbus. The bulbar and palpebral conjunctiva and the main and accessory lacrimal tissues were then excised whole from the orbit (Fig. 1). Conjunctivas from three of these subjects were examined for lactoferrin.

Biopsy specimens—Specimens from normal-appearing lacrimal glands were obtained from two patients during orbital decompression for thyroid ophthalmopathy and from one patient during orbital exploration for venous aneurysm (Table). Conjunctival biopsy specimens were obtained from three patients free of ocular disease and ten patients with a variety of inflammatory ocular diseases,

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TABLE

DATA ON LACRIMAL TISSUE DONORS

Case No.	Age (yrs)	No. of Hours Postmortem	Diagnosis
Tissue from autopsy			
1	54	22	Alcoholic cirrhosis of liver with jaundice
2	67	23	Severe rheumatoid arthritis; pneumonia; subacute bacterial endocarditis
3	72	20	Carcinoma of pancreas with metastasis
4	73	22	Progressive supranuclear palsy
5	59	120	Cirrhosis; portal hypertension; no jaundice
6	65	9	Acute renal tubular necrosis with renal failure; *Staphylococcus* bacteremia
7	65	31	Heart failure
Tissue from surgery			
8	35	—	Thyroid exophthalmos
9	30	—	Epibulbar venous aneurysm
10	31	—	Thyroid exophthalmos

including episcleritis, marginal blepharitis, benign lymphoid hyperplasia, herpes simplex keratitis, atopic keratoconjunctivitis, and benign mucosal pemphigoic.

Preparation of tissues for immunofluorescence—The excised tissue was fixed in 19 parts absolute alcohol to one part glacial acetic acid at room temperature for 48 hours, in a modified version of a technique described by Wolman and Behar.[22] The tissue was dehydrated, embedded in paraffin, cut into 4-μ sections, and then deparaffinized as described by Sainte-Marie.[23]

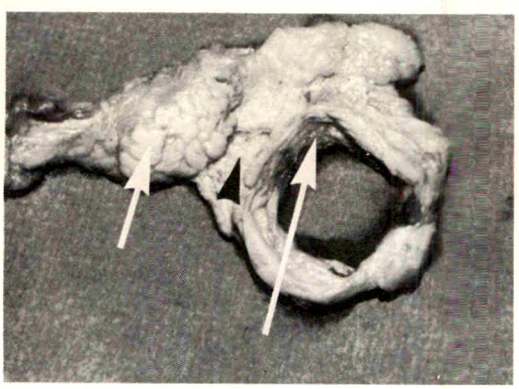


Fig. 1 (Gillette and Allansmith). Autopsy specimen. Orbital lobe of main lacrimal gland (short white arrow); accessory lacrimal gland (black arrowhead); upper forniceal conjunctiva (long white arrow).

Immunofluorescence—A two-layer (indirect) immunofluorescence technique was used. Deparaffinized tissue sections were incubated in a moist chamber with a commercially obtained rabbit antilactoferrin antiserum diluted 1:10 in 0.1M phosphate-buffered saline for 15 minutes, and then subjected to two five-minute washings in phosphate-buffered saline. The tissue sections were then incubated with a commercially obtained fluorescein isothiocyanate-conjugated goat antirabbit IgG diluted 1:40 in phosphate-buffered saline for 15 minutes, and given two more five-minute washings in phosphate-buffered saline.

Control sections were incubated with normal rabbit sera at a 1:10 dilution, washed, and then incubated with the fluorescein-conjugated goat antirabbit IgG diluted 1:40 in phosphate-buffered saline.

Characterization of antisera—Monospecific antiserum to human lactoferrin was obtained commercially from two sources. Both antisera were found to be one-line-specific by Ouchterlony analysis against both colostrum and tears. Both antisera showed no identity with antisera against human secretory component and human IgA (Fig. 2). Goat antirabbit IgG was one-line-specific against normal rabbit serum.

Observation and photography—Stained sections were examined with a Zeiss fluorescence microscope fitted with an HBO 200-W high-pressure mercury vapor lamp, BG 38 (suppression) and UG 5 (excitation) filters, and a 410-nm barrier filter. Photomicrographs were taken on Ektachrome ASA 200 or ASA 400 color film, through the same combination of filters used for observation.

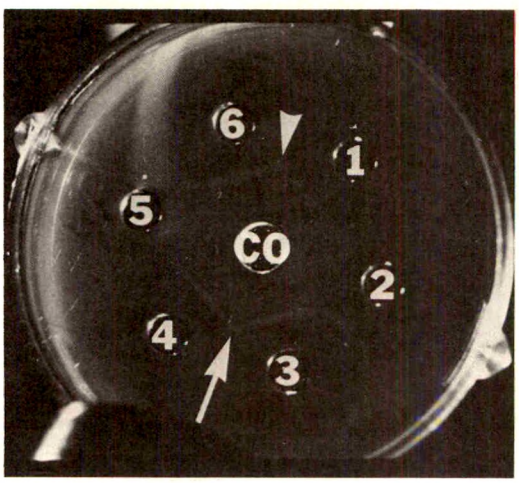


Fig. 2 (Gillette and Allansmith). Ouchterlony analysis of rabbit antihuman lactoferrin antiserum. Central well, colostrum (co). Well 1, rabbit antihuman IgA. Well 2, goat antihuman secretory component (absorbed with human lactoferrin). Well 3, rabbit antihuman lactoferrin. Well 4, rabbit antihuman secretory component (unabsorbed). Well 5, empty. Well 6, rabbit antihuman lactoferrin. Rabbit antihuman lactoferrin shows intersections of nonidentity with rabbit antihuman secretory component (arrow) and with rabbit antihuman IgA (arrowhead).

Results

Brilliant, apple-green fluorescence, indicating the presence of lactoferrin, was seen in approximately 90% of acinar epithelial cells. Often, all cells of a single acinus were stained, but in some acini one or several unstained cells contrasted strikingly with the brightly stained cells (Fig. 3a). The unstained cells were distributed randomly, and rarely accounted for most cells in a single acinus (Fig. 3a). The distribution of stained and unstained cells was uniform throughout the lacrimal gland. The distribution of specific fluorescence within the acinar cells was uniform, with strongly fluorescent material homogeneously filling stained cells (Fig. 3b).

Lacrimal tubules (identified by large luminal diameter relative to epithelial circumference) were mostly unstained (Fig. 3c), but an occasional tubular epithelial cell was stained. We did not see any tubule with most of its epithelial cells stained. Overall, fewer than 1% of tubular cells were stained.

Most acinar and tubular lumina were unstained, but stained material was seen adjacent to the apical surfaces of some epithelial cells. No staining of interstitial tissues, vascular components, or lymphoid elements was seen (Fig. 3). Lacrimal-gland biopsy specimens showed a staining pattern identical to that of the autopsy specimens.

Accessory lacrimal tissue showed a staining pattern identical to that of tissue from the main lacrimal gland, with approximately 90% of acinar epithelial cells and fewer than 1% of the tubular epithelial cells staining for lactoferrin (Fig. 3d).

All sections of conjunctiva examined,

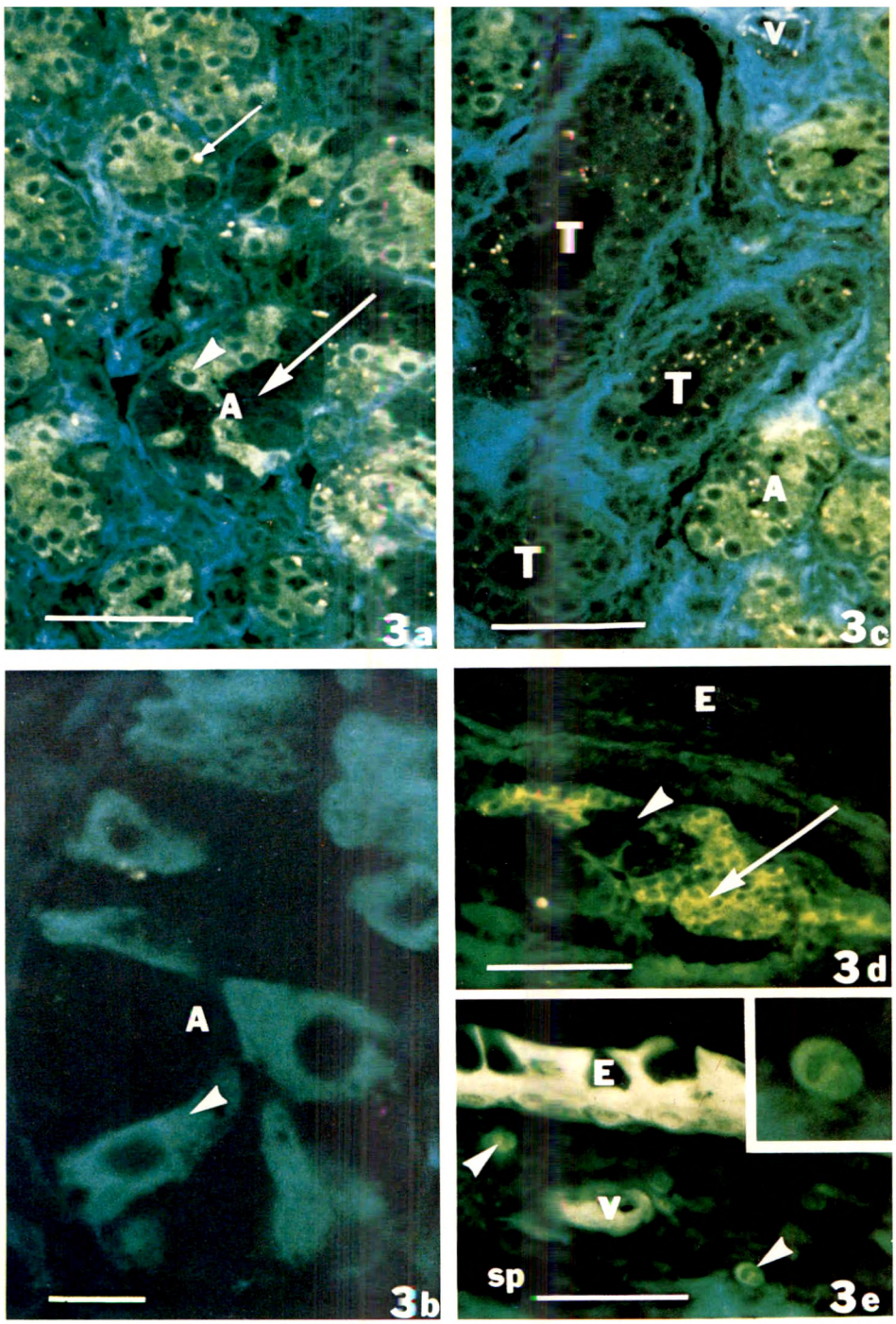


Fig. 3 (Gillette and Allansmith). Fluorescent staining for lactoferrin in human lacrimal gland, accessory lacrimal gland, and conjunctiva. (a) Main lacrimal gland. Majority of acinar cells (A) stain for lactoferrin (arrowhead), but some are completely unstained (large arrow). Small arrow indicates autofluorescent lipofuschin granule. Bar gauge = 50 μm (x200). (b) Acinus (A) of main lacrimal gland. Staining for lactoferrin within acinar cell (arrowhead) is diffuse throughout the cell, with the exception of the nucleus. Bar gauge = 10 μm (x800). (c) Main lacrimal gland. Acinus (A) is stained; tubules (T) and blood vessels (V) are not. Bar gauge = 50 μm (x200). (d) Conjunctiva and accessory lacrimal gland. Arrow indicates staining of acinar cells of accessory lacrimal gland. Arrowhead indicates unstained tubular cells. Epithelium (E) of conjunctiva is unstained. Bar gauge = 50 μm (x200). (e) Conjunctiva. Epithelium (E) and substantia propria (sp) are unstained. Neutrophils in substantia propria are stained for lactoferrin (arrowheads). Bar gauge = 50 μm (x200). Inset (x800) shows staining for lactoferrin in a neutrophil of the substantia propria. Epithelium (E) and blood vessel (V) are autofluorescent.

including biopsy and autopsy specimens, revealed staining of occasional neutrophils (Fig. 3e). These neutrophils were usually located in the substantia propria. No staining above background of the epithelium or the substantia propria was seen (Fig. 3d).

Other ocular tissues were examined for the presence of lactoferrin to ensure a complete study of the eye and its appendages. All tissues were fixed and processed in a fashion identical to that done for lacrimal tissues and conjunctiva. No staining for lactoferrin was observed in the cornea, the sclera, the choroid, the iris, the retina, or the extraocular muscles.

Discussion

We have confirmed the presence of lactoferrin in the main lacrimal gland, which was reported by Franklin, Kenyon, and Tomasi[17] in 1973. Our data extend their findings by showing that lactoferrin is present in approximately 90% of lacrimal acinar epithelial cells. Fewer than 1% of tubular epithelial cells stained for lactoferrin.

Fluorescence was distributed within the acinar cells as a dense homogeneous filling like that seen in serous acinar cells of human pulmonary tissue by Masson and associates.[12] However, serous acinar cells (as defined by the criteria of Allen, Wright, and Reid[24]) constitute less than half of the lacrimal gland's volume, so lactoferrin synthesis in the lacrimal gland cannot be attributed to only serous cells. No group of cells exhibited a fluorescent peripheral rim like that seen by Masson and associates[12] in groups of bronchial mucous cells.

The positive identification of lactoferrin in most lacrimal acinar epithelial cells is strong evidence for the synthesis of tear lactoferrin in the main lacrimal gland. The tubular epithelium of the lacrimal gland, however, does not appear to contribute significantly to tear lactoferrin synthesis. The identical fluorescent staining pattern of accessory lacrimal tissue implicates the accessory lacrimal glands as an additional source of tear lactoferrin, particularly if normal basal tear secretion is solely from accessory lacrimal tissue as suggested by Jones.[25]

The absence of fluorescent staining for lactoferrin in the human conjunctiva eliminates this tissue as a source of tear lactoferrin and leaves open for investigation the identity of the conjunctival secretory granules noted by Takakusaki.[21]

Neutrophilic granulocytes have been shown to synthesize and secrete lactoferrin in vitro.[26] Therefore, the neutrophilic granulocytes present in the normal human tear film may be an additional source of tear lactoferrin. Because the average number of neutrophils in the normal tear film is less than 250,[4] total release of their contained lactoferrin could produce an expected maximum of only 1.1×10^{-4} mg/ml lactoferrin, according to the following calculation (the lactoferrin/neutrophil value is taken from Masson, Heremans, and Schonne[26]):

$$\frac{(3 \times 10^{-6}\ \mu g \text{ lactoferrin/neutrophil}) \times (250 \text{ neutrophils})}{7\ \mu l \text{ preocular tear film}} =$$

$$1.1 \times 10^{-4}\ \mu g/\mu l = 1.1 \times 10^{-4} \text{ mg/ml}$$

The normal concentration of lactoferrin in tear film is 1.45 mg/ml.[16] Tear-film neutrophils could therefore not be expected to contribute significantly to normal tear-film lactoferrin content.

Another possible source of tear lactoferrin is the mantle of neutrophils known to be present in the normal human conjunctiva.[27] Adding the average numbers of neutrophils present in epithelium and substantia propria of tarsal and forniceal conjunctiva gives 8,000 cells/mm^3 for tarsal conjunctiva and 6,000 cells/mm^3 for forniceal conjunctiva. Multiplying the average of 7,000 cells/mm^3 by the conjuncti-

val surface area, which is 350 mm^2 (forniceal) + 350 mm^2 (tarsal), gives 49.0×10^5 cells/mm. Multiplying this by the average conjunctival depth of 0.1 mm noted in routine histologic examinations yields a total ocular neutrophil count of 4.9×10^5 cells. This gives a total maximum lactoferrin production by periocular neutrophils (achieved by total intracellular-content release) of 1.47 μg (obtained by the calculation [4.9×10^5 cells] × [3×10^{-6} μg lactoferrin/cell]). Dividing by the preocular tear-film volume of 7 μl[28] gives 0.21 mg/ml of lactoferrin. This value clearly indicates that the periocular mantle of neutrophilic leukocytes could only contribute significantly to normal tear-film lactoferrin levels if they were to release their total intracellular content.

The staining of conjunctival granulocytes for lactoferrin shows the sensitivity of our immunofluorescence technique (average neutrophil lactoferrin content = 3×10^{-6} μg). The absence of conjunctival staining in the presence of positively staining neutrophils is evidence against the possibility that the human conjunctiva or its contained neutrophils is a source of lactoferrin in human tears.

The lack of staining for lactoferrin in the lacrimal interstitium and the substantia propria of the conjunctiva seems to preclude a vascular source of lactoferrin in human tears. This conclusion is further supported by the much greater concentration of lactoferrin in tears than in serum. However, selective permeability of the acinar basement membrane or the conjunctival basement membrane to small amounts of lactoferrin beyond the reach of our immunohistochemical technique cannot be eliminated.

Summary

The main lacrimal gland and accessory lacrimal tissue from seven autopsy cases, lacrimal biopsy specimens from three patients, and conjunctival biopsy specimens from ten patients were examined for lactoferrin by an immunohistologic technique. Lactoferrin was identified and localized to acinar epithelial cells of both main and accessory lacrimal tissue. Lactoferrin was not found in conjunctival tissue except within conjunctival neutrophils.

Other possible sources of human tear lactoferrin were considered, but we concluded from our data that the primary source of lactoferrin in normal human tears is the acinar epithelium of the main and accessory lacrimal glands.

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SPECTRUM OF MICROBIAL KERATITIS IN SOUTH FLORIDA

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Just as different regions of the world are characterized by specific endemic infections, the predominating organisms in ocular infections also vary throughout the world. This is dramatically demonstrated by the frequency of fungal keratitis in the southern United States. Even the type of fungi seen in the South contrasts with that seen in northern climates.[1-3] These regional differences in causative organisms in microbial keratitis are clinically important because they influence the stains and media used for isolation, as well as the initial and more definitive therapy.

Recently, ocular microbiology laboratories have evolved systems capable of recovering almost all possible organisms that produce microbial keratitis.[4-7] We studied the causes of microbial keratitis in south Florida and compared this profile with other geographic reported series. The specificity and value of the different laboratory tests, especially new diagnostic aids performed in these cases, were evaluated. Because of the frequency of fungal keratitis and *Pseudomonas* keratitis in south Florida, their clinical profiles were studied.

SUBJECTS AND METHODS

We scraped and evaluated 663 corneal ulcers in the microbiology laboratory here during the nine years between Jan. 1, 1969, and Dec. 31, 1977. Only patients seen on a private referral basis or from the resident teaching service were included. Rescraped and recultured ulcers were excluded unless they were positive for a different organism.

All patients suspected of having microbial keratitis underwent corneal scraping with a platinum spatula under biomicroscopy. Scraped material was routinely stained with Gram and Giemsa preparations. A 20% potassium hydroxide preparation routinely used initially was later replaced by the Grocott methenamine silver stain for suspected fungal ulcers.[8] The limulus lysate assay for detection of gram-negative endotoxin was also recently incorporated.[9]

Culture material from either corneal scrapings or keratoplasty specimens was routinely plated on the following media: rabbit blood agar at 37°C; sheep blood agar at 25°C; chocolate blood agar at 37°C under increased CO_2 tension; Sabouraud's medium of dextrose-peptone agar with yeast extract and 50 μg/ml of gentamicin sulfate, without cycloheximide, at 25°C; brain-heart infusion liquid medium with 50 μg/ml of gentamicin, maintained on a rotary shaker at 25°C; and thioglycollate broth at 37°C. The solid medium was inoculated with multiple scrapings, and each scraping was made into a row of "C" streaks.[5] Liquid medium was inoculated directly from the spatula, or scraped material was transferred to a sterile cotton swab applicator moistened in Trypticase soy broth and placed in the liquid medium.

Bacteria were classified according to the usual laboratory guides; an attempt was made to determine the genus and

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species of all fungi with use of aids mentioned previously.[5,10]

The criteria for considering a corneal ulcer infected were the following: growth of the same organism on more than one culture medium; semiconfluent growth on several "C" streaks on one solid medium; or heavy growth within the liquid medium confirmed by a positive-stained smear. Several cultures yielded only a few organisms but did not meet the above criteria and were considered noninfected.

RESULTS

In the nine years of the study, 663 corneal ulcers were scraped; the frequency increased slightly in the last three years (Fig. 1). Fungal ulcers steadily averaged 15 per year, but bacterial ulcers increased in recent years. Of the cultures, 292 (44%) did not fulfill the criteria established for the presence of infection. In this culture-negative group, there were cases of inflammatory keratitis and perhaps some cases of infectious keratitis that were treated before culturing. In 16 negative cultures, definite organisms were seen with either the Gram stain (15 cases) or the Giemsa stain (one case).

All ulcers were unilateral except for one bilateral *Pseudomonas* ulcer and one bilateral *Serratia* ulcer; both occurred in hospitalized patients with exposure keratitis. Different organisms were identified in sequential ulcers on only two occasions; in both instances, beta-hemolytic streptococci were isolated from a previously confirmed fungal keratitis.

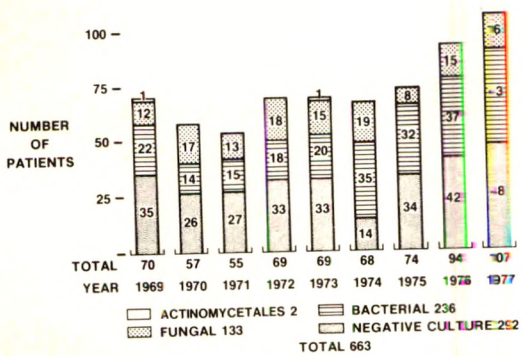


Fig. 1 (Liesegang and Forster). Frequency and causes of ulcerative keratitis in Miami, Florida, 1969 through 1977.

Bacteria—The isolates from the 238 bacterial ulcers are listed in Table 1. *Staphylococcus aureus* and *S. pneumoniae* represented 69 (29%) of the bacterial ulcers. *P. aeruginosa* was the most common organism (74, or 31%). A variety of gram-negative rods were seen but *Mora-*

TABLE 1

ORGANISMS IN BACTERIAL KERATITIS (MIAMI, 1969 THROUGH 1977)

Organism		No. of Patients
Gram-positive cocci		115
Staphylococcus aureus	51	
S. epidermidis	19	
Streptococcus pneumoniae	18	
Alpha-hemolytic streptococci	13	
Micrococcus	7	
Beta-hemolytic streptococci	4	
St. faecalis	2	
Sarcina	1	
Gram-positive rods		23
Corynebacterium	17	
Bacillus	4	
Propionibacterium	2	
Gram-negative cocci		5
Moraxella	4	
Neisseria	1	
Gram-negative rods		108
Pseudomonas	74	
Proteus	18	
Serratia	6	
Klebsiella	4	
Enterobacter	2	
Haemophilus	2	
Acinetobacter	1	
Citrobacter	1	
Actinomycetales		2
Mycobacterium	1	
Streptomyces	1	
Total		253*

*In 238 patients (>one organism in 15 patients).

xella species were infrequent (four cases). *Staphylococcus epidermidis* and *Corynebacterium* species, considered nonpathogens in the past, were found with increasing frequency. Anaerobes were identified only twice (both *Propionibacterium* species), and *Mycobacterium* and *Streptomyces* were isolated in one case each. Two organisms were isolated from 15 eyes; usually, *S. aureus* predominated, but other combinations were encountered. No notable shifts in the causative bacterial organisms in south Florida occurred during this nine-year period.

The present series of 236 bacterial ulcers (eliminating the two Actinomycetales) was compared with previously published data from other areas[11-13] and a previous series from Miami for the period 1963 through 1969,[4] in order to construct a temporal and geographic profile of bacterial corneal ulcers in the United States (Fig. 2). In these reported series, gram-positive cocci were the predominant bacterial organisms seen in all regions of the United States except in south Florida. *Pseudomonas* keratitis comprised 49% (33 of 67) of the original south Florida series and 31% (74 of 238) of the present series. *Pseudomonas* was seen considerably less often in other series, although other gram-negative organisms were not uncommon in San Francisco[12] and Wisconsin.[13]

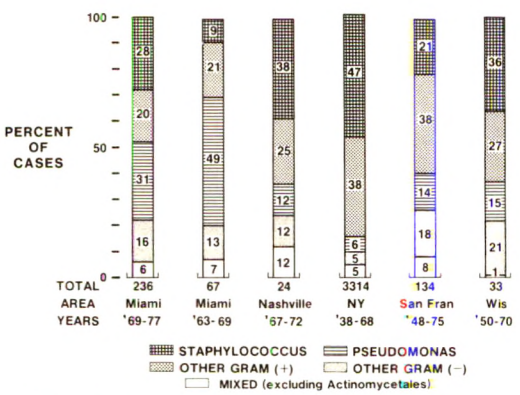


Fig. 2 (Liesegang and Forster). Variation in bacterial causes of corneal ulcers in various geographic areas of the United States.

Fungi—Fungal organisms were isolated from 133 patients; this is the largest reported series of fungal ulcers and emphasizes the unusual geographic and perhaps agricultural conditions in south Florida. The fungal isolates were arbitrarily classified into four groups: (1) *Fusarium solani;* (2) other Moniliaceae (nonpigmented filamentous fungi); (3) Dematiaceae (pigmented filamentous fungi; and (4) yeasts (Table 2).

As in previous series from Florida,[14,15] *F. solani* was the predominant filamentous fungus—61% of the 124 fungal isolates in this series. Other nonpigmented fungi included *Aspergillus* and *Acremonium.* The frequency of *Aspergillus* in this series was distinctly lower than that reported in other areas of the world,[16] but comparable to that reported in other series from the southern United States.[15,17]

Curvularia was the most common pigmented fungus isolated (eight cases), and *Lasiodiplodia,* a unique tropical isolate, was seen in five cases.[18] A large number of species were occasionally isolated in both the pigmented and the nonpigmented classes, but certain genera were more prevalent as repeating isolates. Although ten yeasts (all *C. albicans)* were isolated, only one was isolated after 1974.

Seasonal variation—A previous series of fungal ulcers from south Florida showed an absence of fungal keratitis during the hot, humid, rainy months from June to September.[19] In the present series *Fusarium* species were not cultured during these months until 1972, and only one other filamentous fungus was found during these months until 1971. In more recent years, fungal keratitis was seen during these months but

TABLE 2

ORGANISMS IN FUNGAL KERATITIS (MIAMI, 1969 THROUGH 1977)

Organism	No. of Patients
Moniliaceae (Nonpigmented Filamentous Fungi)	
Fusarium solani	76
Other *Fusarium* species	6
F. episphaeria (dimerum)	3
F. moniliforme	1
F. nivale	1
F. oxysporum	1
Aspergillus	6
A. fumigatus	3
A. flavus	3
Acremonium (Cephalosporium)	4
Penicillium	4
Paecilomyces	2
Petriellidium boydii (Allescheria boydii)	2
Geotrichum candidum	1
Myrathecum	1
Volutella	1
Cylindrocarpon	1
Dematiaceae (Pigmented Filamentous Fungi)	
Curvularia	8
C. senegalensis	3
C. verruculosa	1
C. pallescens	1
Unidentified species	3
Sphaeropsidales (Lasiodiplodia theobromae)	5
Melanconiales (Colletotrichum atramentum)	3
Alternaria	2
Drechslera (Helminthosporium)	1
Cladosporium	1
Yeasts	
Candida albicans	10
Total	134*

*In 133 patients (>one organism in one patient).

was more frequently caused by the non-*Fusarium* filamentous fungi (Fig. 3). November and March, usually dry and windy months in South Florida, were the peak months for occurrence of fungal keratitis.

The seasonal variation of gram-positive and gram-negative bacterial infections was not striking, but November was the peak month for occurrence of both bacterial and fungal ulcers (Fig. 4).

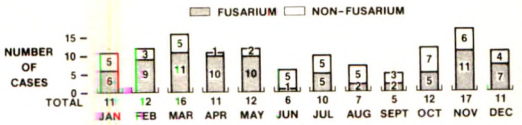


Fig. 3 (Liesegang and Forster). Seasonal variation in corneal fungal ulcers in Miami, Florida.

Laboratory stains—Bacterial organisms seen with the Gram stain were consistent with the cultured bacterial organisms 66% of the time (in 103 cases) (Table 3). No organisms were seen in 32% (50 cases), and the interpretation was incorrect on three occasions. Gram-positive organisms were identified correctly by Gram stain more frequently than either *Pseudomonas* species or other gram-negative organisms. In four cases, the Gram stain was negative but the Giemsa stain showed distinct morphologic forms consistent with *Pseudomonas* cultured later. However, the Gram stain was more sensitive than the Giemsa stain in identification of bacteria.

Among the cases of fungal keratitis, the Giemsa stain showed fungal fragments 66% of the time (in 62 cultures), compared with 55% of the time (in 52 cultures) for the Gram stain (Table 4). With the Grocott methenamine silver stain, we were able to identify fungal fragments in 18 of the last 21 fungal ulcers confirmed by culture.

In cultured fungal keratitis, hyphae were seen on two or more stains 60% of the time (in 56 cases) and were absent

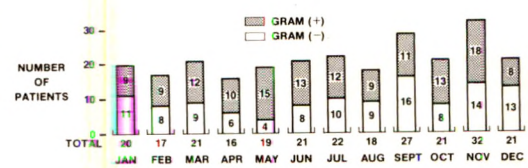


Fig. 4 (Liesegang and Forster). Seasonal variation in corneal bacterial ulcers in Miami, Florida.

TABLE 3

GRAM STAIN IN DIAGNOSIS OF BACTERIAL KERATITIS*

Result	Gram-Positive Organisms (N = 77)		Gram-Negative Organisms				Total (N = 156)	
			Pseudomonas (N = 63)		Other (N = 16)			
	No.	%	No.	%	No.	%	No.	%
Correct organisms seen	55	71	39	62	9	56	103	66
No organisms seen	22	29	21	33	7	44	50	32
Incorrect interpretation	0	—	3	5	0	—	3	2

*In eight cases, both gram-positive and gram-negative organisms were cultured; with the stain, gram-positive organisms had been seen in six of these cases and gram-negative organisms had been seen in two.

from all stains only 19% of the time (in 18 cases) (Table 5). Therefore, 81% (76 cases) of the fungal infections were diagnosed by using this battery of stains, whereas only 66% (103 cases) of the bacterial infections were identified by Gram stain alone. When only one stain was positive for fungus, most frequently it was the Giemsa stain, but some fungi were detected only with the Gram stain. The true value of the Grocott methenamine silver stain will become more evident with increased use.

Limulus lysate assay—We used the limulus lysate assay for the detection of gram-negative endotoxin in 30 clinical cases (Table 6). Of these, 12 were correctly identified as gram-negative; in the rest of the cases, the results have been negative, with no false-positive or false-negative results to date. In two cases of *Pseudomonas* keratitis, results of the limulus lysate test were positive, whereas the routine Gram stains were negative.

Isolation media—In the series of bacterial ulcers, organisms were almost routinely recovered from the blood agar plate at 37°C as well as from other culture media. In a few cases, the blood agar plate at 37°C was negative, whereas one or several of the other media were

TABLE 4

DIAGNOSTIC LABORATORY STAINS IN FUNGAL KERATITIS

Stain	Total	Positive	
	No.	No.	%
Gram	94	52	55
Giemsa	94	62	66
Potassium hydroxide*	30	10	33
GMS†	21	18	86

*Used initially but later replaced by the Grocott methenamine silver stain.
†GMS designates Grocott methenamine silver.

TABLE 5

PRESENCE OF HYPHAE IN 94 CASES OF FUNGAL KERATITIS*

Hyphae	Cases	
	No.	%
Present on two or more stains	56	60
Present on only one stain	20	21
Only Giemsa-positive	12	
Only gram-positive	5	
Only GMS-positive	2	
Only potassium hydroxide preparation positive	1	
Not seen	18	19

*GMS designates Grocott methenamine silver.

TABLE 6

LIMULUS LYSATE ASSAY IN ULCERATIVE KERATITIS

Result of Assay	No. of Cases
Positive with	
Pseudomonas species	9
P. mirabilis	1
Citrobacter diversus	1
Hemophilus influenzae	1
Negative with	
Gram-positive organisms	4
Fungal organisms	3
Negative cultures	11
Total	30

positive. In ten cases, the blood agar plate at 37°C was negative and only a single medium supported bacterial growth: thioglycollate in four cases, chocolate agar in three cases, blood agar at 25°C in two cases, and brain-heart infusion liquid in one case. All of these single cultures met the criteria of positive isolation listed previously and, additionally, were confirmed by the Gram stain.

Data on the isolation of fungi from the various culture media are presented in Table 7. Sabouraud's agar was positive 79% of the time (in 82 cultures), compared with 71% (74 cultures) with blood agar at room temperature. There was no difference between *Fusarium* species and other fungi. Blood agar at 37°C and other media were less supportive of growth. The fungi were isolated on multiple media 80% of the time (in 84 cases) (Table 8). When only one medium was positive, it was more likely to be Sabouraud's or blood agar at 25°C. All instances of these single medium growths were confirmed by detection of hyphal fragments in the original corneal scrapings. Growth characteristically took place at 36 to 72 hours; delayed recovery of organisms during the two-week holding time seldom occurred.

TABLE 7

CULTURE MEDIA FOR ISOLATION OF FUNGI

Medium	No. of Cultures	
	Total	Positive
Sabouraud's agar plate		
At 25°C	104	82
At 37°C	2	0
Blood agar plate		
At 25°C	104	74
At 37°C	104	38
Brain-heart infusion liquid at 25°C	104	55
Thioglycollate liquid at 37°C	53	9
Chocolate agar plate at 37°C	52	5

Sensitivity patterns—The Kirby-Bauer disk sensitivity patterns of both the gram-positive cocci and the *Pseudomonas* species during the past nine years were evaluated (Table 9). All gram-positive cocci were sensitive to a cephalosporin, and most that were tested were sensitive to bacitracin, gentamicin, and oxacillin, with no recent changes in this trend. *Pseudomonas* has remained sensitive to gentamicin except for one isolate in 1975. Carbenicillin and colistimethate

TABLE 8

ISOLATION OF FUNGI ON ONE OR MORE THAN ONE CULTURE MEDIUM

Isolation	No. of Cases	
On more than one medium		84
On only one medium		21
Sabouraud's agar plate at 25°C	11	
Blood agar plate:		
At 25°C	6	
At 37°C	3	
Brain-heart infusion liquid at 25°C	1	

TABLE 9

KERBY-BAUER DISK SENSITIVITY PATTERNS

Antibiotic	Total No.	Sensitive No.	Sensitive %
Sensitivity of Gram-Positive Cocci			
Cephaloridine	83	83	100
Bacitracin	58	56	97
Gentamicin	70	67	96
Oxacillin	77	73	95
Neomycin	57	43	75
Penicillin	82	50	61
Sensitivity of *Pseudomonas*			
Gentamicin	64	63	98
Carbenicillin	43	39	91
Colistimethate sodium	59	48	81
Neomycin	45	28	62
Chloramphenicol	63	21	33

sodium have recently shown increasing resistance in our laboratory. Because testing of antifungal sensitivities is still experimental in our laboratory, firm data are not available.

Clinical characteristics of patients with fungal and Pseudomonas *keraeratitis*—Inasmuch as fungal and *Pseudomonas* organisms are the most frequent causes of microbial keratitis in south Florida, we investigated the clinical profiles of these diseases by obtaining pertinent clinical information from the patients with these isolates (Table 10). Male patients predominated in both groups. A definite history of ocular trauma (usually with vegetable matter) was elicited in 60% (80) of the patients with fungal keratitis, compared with 22% (13) of those with *Pseudomonas* ulcers. Additionally, many of the patients with fungal keratitis gave a history of agricultural work without a definite history of trauma.

Three of the patients with fungal ulcers were wearing soft contact lenses at the onset of their disease; in two of these patients, the lenses had been prescribed for therapeutic purposes. The fungal organisms isolated in these patients were *C. albicans*, *A. flavus*, and *F. dimerum*, the least common organisms in this series. Of the patients with *Pseudomonas* keratitis, 20 were wearing contact lenses at the time of their infection, eight with hard lenses and 12 with soft lenses. Almost all of these patients wore contact lenses for cosmetic reasons or for apha-

TABLE 10

CLINICAL CHARACTERISTICS OF PATIENTS WITH FUNGAL AND *PSEUDOMONAS* KERATITIS

Characteristic	Fungal (N = 133) No.	Fungal (N = 133) %	*Pseudomonas* (N = 60) No.	*Pseudomonas* (N = 60) %
Males	109	82	39	65
History of trauma	80	60	13	22
Contact lens wear	3	2	20	33
Previous inflammatory eye disease	8	6	12	20
Use of corticosteroids before onset of keratitis	3	2	6	10
Corticosteroid therapy initiated before referral	33	25	4	7
Use of antiviral agents before referral	13	10	0	—
Significant general medical disease	24	18	22	37
Age (yrs): Average	44		54	
Range	2½-86		8 mos-85	

kia, and they had no history of trauma. The patients with *Pseudomonas* keratitis had more significant general medical disease and previous ocular inflammatory disease. This was especially evident with exclusion of those who wore contact lenses for cosmetic reasons. The ten patients with *Candida* keratitis as a group had more significant medical disease and previous inflammatory ocular disease than did the rest of the patients with fungal ulcer.

Only three of the patients with fungal ulcers were treated with corticosteroids before the onset of keratitis (topically administered corticosteroids were being given to one patient for uveitis and to another after penetrating keratoplasty, and systemic corticosteroids were being administered to one patient with lupus erythematosus). Six of the patients with *Pseudomonas* keratitis were being given topically administered corticosteroids for various inflammatory diseases of the anterior segment. Of the patients with fungal keratitis, 25% (33 patients) were treated with corticosteroids after development of symptoms, but before they were referred to us, and 10% (13 patients) were treated with antiviral agents. These findings are similar to those reported by Polack, Kaufman, and Newmark.[15] In all but 27% (36) of the patients with fungal keratitis, antibiotic, antiviral, or corticosteroid therapy was initiated after the onset of symptoms but before definitive diagnosis. Secondary fungal ulcers were documented in only two patients, in one after a bacterial keratitis (*S. pneumoniae*) and in one after herpes simplex epithelial keratitis. In two fungal ulcers, bacterial superinfection with beta-hemolytic streptococci developed subsequently.

Although fungal keratitis may be indolent, it usually was manifested initially as a rapidly developing process; the onset may resemble that of bacterial keratitis. Patients with *Pseudomonas* ulcers had a more fulminating course and with a much shorter delay until referral, although most were being treated with antibiotic therapy at the time of referral.

Discussion

Ulcerative keratitis is the primary medical cause for hospitalization here; 58 patients were admitted in 1977 and had an average length of stay of eight days. We prefer to hospitalize most of these patients for a short course of intensive treatment until the infection begins to respond. Treatment is predicated on the correct diagnosis, and this is supported by early laboratory tests and final culture results.

Each year, about 45% of cases of suspected infectious keratitis are culture-negative; some of these may represent partially treated bacterial or fungal infections whose growth in culture is inhibited because of use of topically administered antibiotic or antifungal agents. This does not include the herpes ulcers, which we rarely culture unless we suspect superinfection.

About 35% (133 of 371) of the isolated organisms were fungi, a much higher percentage than previously reported elsewhere in the world. *Fusarium solani* was the causative agent in 61% (82 of 134) of the fungal isolates. Numerous other species that were merely mentioned confirm that any fungus has potential for causing keratitis. The Giemsa stain and the more recent Grocott methenamine silver stain were the most reliable for rapid diagnosis; Sabouraud's agar and blood agar at 25°C were most dependable for culture in our study, although O'Day and associates[20] reported brain-heart infusion as their most dependable fungal medium. A significant number of fungi, however, were seen only on other stains or were cultured only on other media, which confirms the need for

multiple scrapings on different media. Although dermatophytes are frequently found in cutaneous disease in south Florida, they have not been seen in corneal disease.

Our bacterial spectrum also varied from that reported in most series in the United States, with a predominance of gram-negative rods. The classic "central hypopyon keratitis" of *S. pneumoniae*, *S. aureus*, and *Moraxella* is less frequent in south Florida. *Corynebacterium* was the distinct pathogen in a number of cases of suppurative keratitis seen both in normal hosts after trauma and in immunocompromised hosts. *Pseudomonas* keratitis continues to be the most frequent bacterial keratitis, with occasional startling progression. On the Gram stain, gram-negative organisms are more likely to be missed than gram-positive organisms, but the recently incorporated limulus lysate test has proved to be clinically useful and complements the Gram stain. Two thirds of our cases of bacterial and fungal keratitis could be suspected by the initial laboratory stains within one hour after scraping.

Many of the patients with *Pseudomonas* keratitis had significant medical disease or ocular inflammation. Preceding trauma was seen in only 13 of 60 patients (22%) with *Pseudomonas* keratitis, but 20 of these patients (33%) were wearing either hard or soft contact lenses at the onset of the keratitis.

Fungal keratitis occurred predominantly in young male patients in good health and without significant ocular disease but with a previous history of ocular trauma that had occurred outdoors or of agricultural occupations. *Candida* was seen in eyes with pre-existent disease and closely resembled the profile of disease seen in patients with *Pseudomonas* ulcers. Fungal keratitis continues to be confused with herpes and other types of inflammatory keratitis, but few patients examined by us had been given corticosteroids earlier. Only three patients were taking corticosteroids at the onset of the keratitis, but 25% (33 patients) had been given cortico-steroids therapeutically before referral. November, a dry windy month in south Florida, is the peak month for occurrence of both bacterial and fungal ulcers.

Although many clinical features could suggest a bacterial or fungal cause of keratitis before the scraping procedure, the variation in symptoms and in laboratory results confirms the need for the same thorough, systematic approach to each case of ulcerative keratitis. To ensure recovery of all possible organisms, we recommend use of the Gram, Giemsa, and Grocott methenamine silver stains and the limulus lysate assay. Multiple corneal scrapings from all areas of the infiltrate should be plated on culture media, including blood agar at 37°C and 25°C, chocolate agar at 37°C, Sabouraud's agar at 25°C, brain-heart infusion at 25°C, and thioglycollate at 37°C. Only through use of this combined battery of cultures and stains were some infections confirmed. Different laboratories may modify these culture media and stains depending on the potential organisms they might encounter in their region.

Antibiotic disk sensitivities have provided a guide to therapy, although they may be meaningless if a concentrated topically administered antibiotic regimen has been used. Rapid and reliable testing of antifungal sensitivities is difficult to perform, and development in this field, such as that by Polansky, Robinson, and Jones,[1] is encouraged.

Summary

During a nine-year period ending in 1977, we scraped and cultured 663 corneal ulcers. Of these cases of keratitis, 238 were bacterial infections, 133 were

fungal, and 292 were culture-negative. *Pseudomonas* was the predominant bacterial organism, and *Fusarium* was the most common fungus isolated. November was the peak month for both bacterial and fungal keratitis.

Direct inoculation of multiple media, including Sabouraud's agar, blood agar, chocolate agar, thioglycollate liquid, and brain-heart infusion liquid, enhanced the recovery rate; each medium provides special nutrients for different organisms. The Gram and Giemsa stains were satisfactory, but the newer Grocott methenamine silver stain for fungi and the limulus lysate test for gram-negative bacteria proved to be clinically useful.

The clinical profile of patients with fungal keratitis differed from that of patients with *Pseudomonas* keratitis. Thirty-three percent of the patients with *Pseudomonas* keratitis were wearing contact lenses at the time of their infection, and the remainder had a high incidence of predisposing ocular conditions. Fungal keratitis tended to occur in healthy male patients who had been subjected to outdoor trauma.

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AN ULTRASTRUCTURAL STUDY OF MELANOCYTOMAS (MAGNOCELLULAR NEVI) OF THE OPTIC DISK AND UVEA

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Melanocytoma, or magnocellular nevus, is a distinctive type of nevus that may originate in the optic nerve head, choroid, ciliary body, or iris.[1] Although these tumors may grow slowly and invade the surrounding ocular tissues, behaviorally they are benign.[2] In contrast with uveal malignant melanomas that show variable pigmentation and cell type, these tumors are consistently heavily pigmented. In bleached histologic preparations, they show remarkably uniform, plump, polyhedral nevus cells with relatively benign cytologic features.[3,4] Recently, these tumors have been shown histologically to have the potential to develop into malignant melanoma,[5,6] but no evidence of orbital recurrence or metastasis has been reported. Possibly because of the benign nature of the lesions, which seldom lead to enucleation, no previous ultrastructural studies of this tumor have been published. We have examined by light and electron microscopy five melanocytomas from four patients.

MATERIAL AND METHODS

Data on Cases 1 and 2 were obtained from our files and Cases 3 and 4 were obtained from the files at the Armed Forces Institute of Pathology (Table 1). In Case 1, two melanocytomas were found in the same eye; one was located in the ciliary body, and the other, in the equatorial choroid away from and not connected with the lesion in the ciliary body. On gross examination all tumors were black with dark brown areas. On sectioning, they were uniformly heavily pigmented.

The tumors from Case 1 and Case 3 were retrieved from paraffin blocks and processed for electron microscopy, by using methods previously described.[6] The other eyes were fixed immediately after enucleation in glutaraldehyde-paraformaldehyde fixative.[7] Selected tissues for electron microscopy were postfixed in Dalton's chrome osmium fixative, dehydrated in alcohol, and embedded in epoxy resin. Thick sections for light microscopy were cut at 1 to 2 μ and stained with Mallory's stain for light microscopy. Ultrathin sections for electron microscopy were stained with uranyl acetate and lead citrate and examined with an electron microscope.

RESULTS

In 8-μ thick paraffin sections, all tumors consisted of uniformly and densely pigmented polyhedral and spindle-shaped cells without recognizable cellular details (Fig. 1). Bleached paraffin sections showed that the tumor cells had abundant pale eosinophilic cytoplasm and relatively distinct plasmalemma membranes (Figs. 1 and 2). The nuclei were oval, densely stained, and centrally placed. Mitotic figures were not observed.

Epoxy sections of 1 μ thickness showed

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TABLE 1

CLINICAL DATA ON FOUR PATIENTS WITH MELANOCYTOMA

Case No., Age (yrs), Sex, Race	Duration of Clinical History	Initial Symptoms	Location	Clinical Diagnosis at Enucleation
1, 42, M, W	2 mos	Metamorphopsia	Ciliary body and choroid	Malignant melanoma of ciliary body
2, 58, F, W	2 yrs	Iris mass	Ciliary body	Malignant melanoma
3, 39, F, W	8 mos	"Spots" on temporal side of field	Choroid	Malignant melanoma
4, 55, M, W	Approximately 6 yrs	Initially, asymptomatic, found in routine eye examination; subsequent progressive visual loss	Optic nerve head and surrounding retina	Melanocytoma with possible malignant change

two types of melanocytoma cells in each of the tumors examined (Fig. 3). The Type 1 variants of the melanocytoma cells were the large polyhedral nevus cells, densely pigmented with large melanin granules. The nuclei were oval with occasional nucleoli. The Type 2 cells were smaller spindle-shaped cells. The cytoplasm of these cells was relatively scanty and only sparingly pigmented with smaller melanin granules. The nuclei were elongated and often contained a small but conspicuous nucleolus. These small cells were intermingled with the large nevus cells. No mitoses were observed in either type of tumor cell. Except in the melanocytoma of the optic nerve, the large polyhedral cell was the predominant cell type in all melanocytomas studied. In the tumor of the optic nerve, the small spindle cell was the predominant type, accounting for about 70 to 80% of the cells. In the melanocytomas of the ciliary body the small cells were observed mostly at the periphery of the tumors.

The morphologic differences between the two cell types (Table 2) were noticed readily ultrastructurally. The large cells were plump with eccentrically located nuclei (Fig. 4). Conspicuous nucleoli were only occasionally found. The heterochromatin was either evenly dispersed or slightly clumped along the nuclear membrane. The most characteristic cytoplasmic features of these tumor cells were the presence of giant melanosomes and relative sparsity of other cytoplasmic organelles. The giant pigment granules were densely osmophilic, round, and enveloped by the plasma membrane (Figs. 4 and 5). They ranged from 1 to 4 μ in diameter and were much larger than the granules present in Type 2 cells (Figs. 4, top left, and 6). At the periphery of these granules, a concentric array of electronlucent microvesicles 30 to 40 nm in diameter were observed. A few mitochondria, fine cytoplasmic filaments (Fig. 7), and occasional desmosomes, or macula adherens cell junctions, were also observed in these cells (Fig. 8).

Type 2 tumor cells (Fig. 9) were smaller. The nucleus was markedly indented with uniformly distributed hetero-

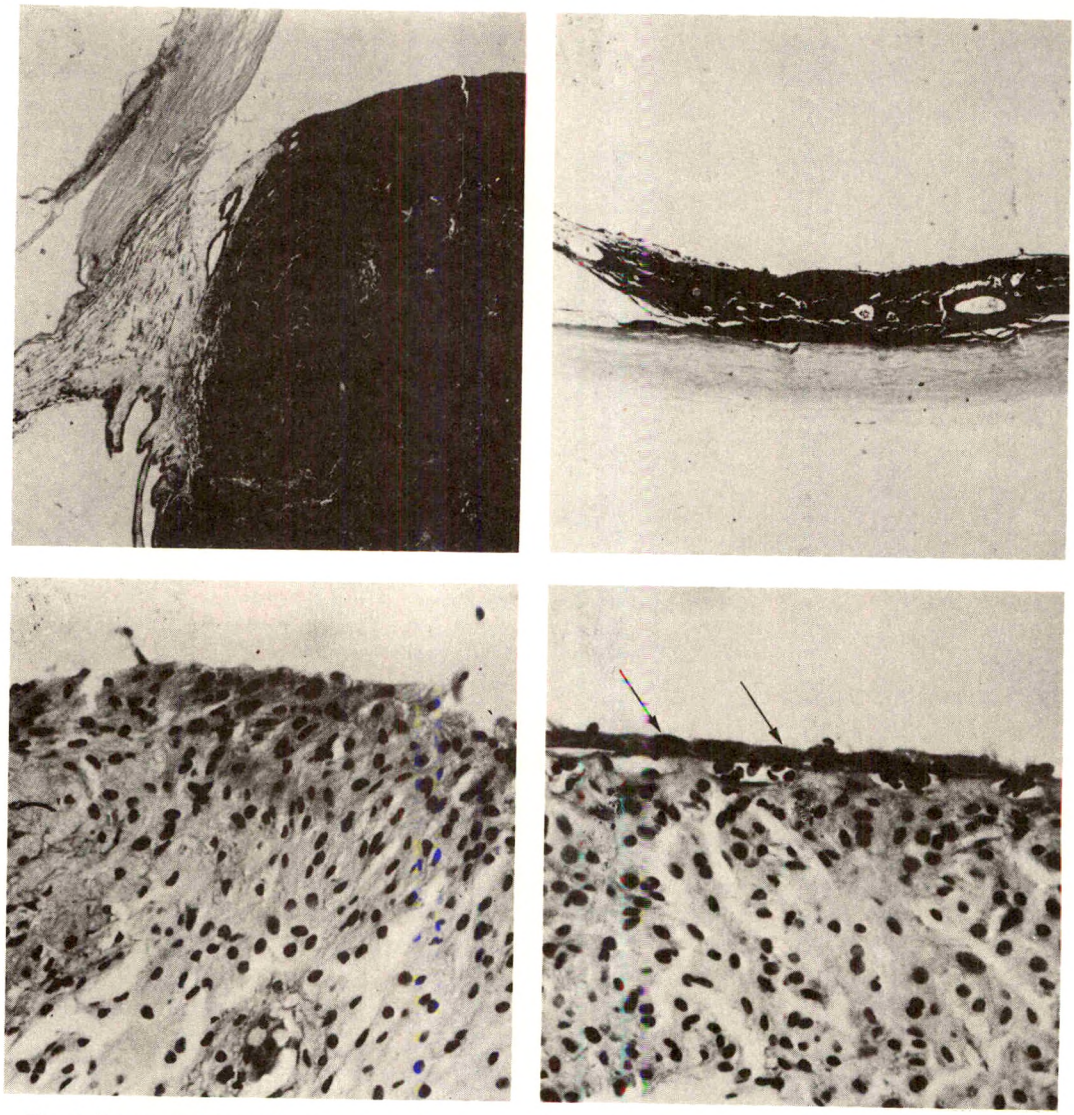


Fig. 1 (Juarez and Tso). Top left, A melanocytoma of the ciliary body. Note uniform dense pigmentation without recognizable cellular details (8 μ paraffin sections stained with hematoxylin and eosin, ×15). Top right, A densely pigmented choroidal melanocytoma (hematoxylin and eosin, ×37.5). Bottom left, Bleached preparation of the same tumor shown at top left. Plump cells with densely stained nuclei are present (8 μ paraffin section bleached with potassium permanganate and stained with hematoxylin eosin, ×210). Bottom right, A bleached paraffin preparation of tumor shown at top right shows plump tumor cells with densely stained, ovoid nuclei. The retinal pigment epithelium (arrows) appears undisturbed. The retina is artifactually detached and bleached with potassium permanganate (hematoxylin and eosin, ×210).

chromatin. The nucleoli were invariably present and showed a network pattern. The cytoplasm disclosed small melanin granules and numerous cytoplasmic organelles. The melanosomes were elongated and fusiform, ranging from 0.1 to 0.4 μ in length, and showed different stages of maturation. The immature granules showed a recognizable irregular filamentous substructural matrix. In the

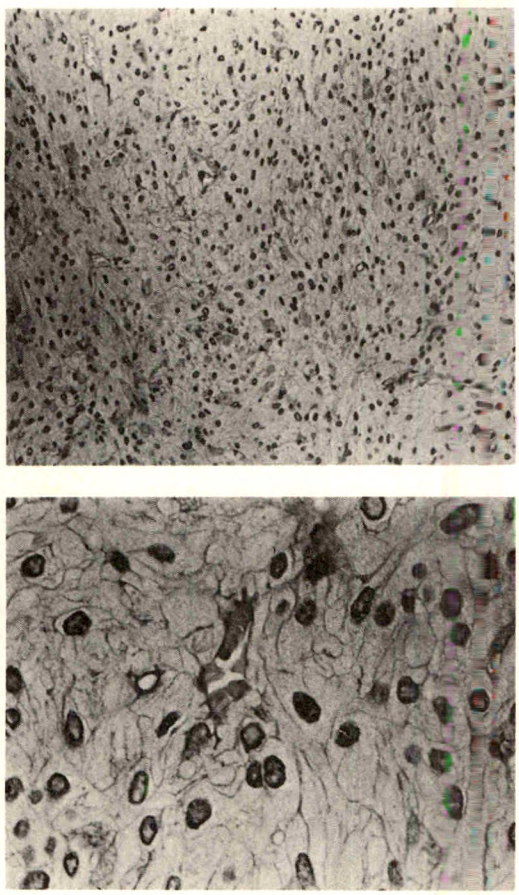


Fig. 2 (Juarez and Tso). A bleached paraffin preparation of a ciliary body melanocytoma. Top, Note uniform cellularity of the tumor cells (sections bleached with potassium permanganate and stained with hematoxylin and eosin, ×37.5). Bottom, Polyhedral nevus cells with central nuclei, abundant cytoplasm, and relatively distinct cytoplasmic membrane (hematoxylin and eosin, bleached with potassium permanganate, ×600).

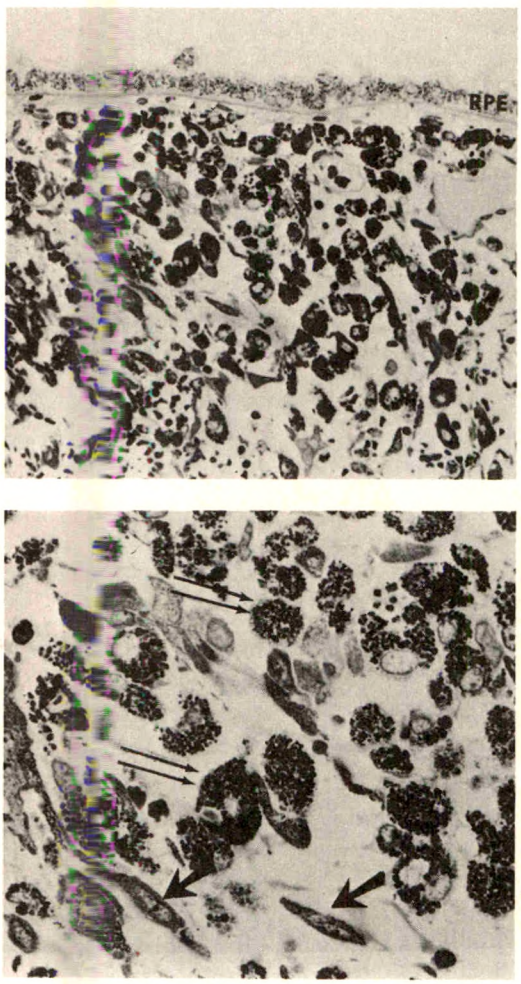


Fig. 3 (Juarez and Tso). Epoxyl sections of a choroidal melanocytoma. Top, Tumor cells appear to be heterogeneous with predominance of large, pigmented nevus cells (Mallory's stain, 1 μ Epon section, ×210). Bottom, Two types of tumor cells are evident: (1) Heavily pigmented, large, plump nevus cells (double small arrows). (2) Smaller spindle-shaped cells with fine pigment granules (single large arrows) Mallory's stain, 1 μ Epon section, ×600).

more mature granules, the filaments were thick and had a rodlike configuration. The cytoplasm of this variant of tumor cell contained numerous mitochondria, occasional Golgi apparatus, and prominent rough endoplasmic reticulum, which frequently appeared in whorls. Free polyribosomes were numerous. Abnormally arranged centrioles were occasionally observed in this type of cell, with their long axis placed parallel to each other. Occasional linear condensation of extracellular material was noted along the plasmalemma, indicating focal basement membrane production (Fig. 7). Fine cytoplasmic filaments and desmosomes, although seen, were infrequently found in this type of cell.

In one of the ciliary body tumors,

TABLE 2

CELL TYPES*

Type 1	Type 2
LIGHT MICROSCOPY	
Large polyhedral cells with round densely stained nuclei	Small spindle-shaped cells
Densely pigmented	Sparingly pigmented
ELECTION MICROSCOPY	
	Markedly infolded nuclei with conspicuous nucleoli
Giant melanosomes (1-4 μ)	Melanosomes (0.1-0.3 μ)
Few cytoplasmic organelles	Abundant cytoplasmic organelles

*Common features of both cell types included occasional desmosomelike attachment, fine cytoplasmic filaments (5-7.5 nm), and focal basement membrane production.

another type of pigmented cell was occasionally observed (Fig. 10). Around these cells, a well-formed basement membrane was noted. Their nuclei were centrally located and contained clumped, randomly dispersed, heterochromatin. Small, ovoid, mature melanin granules with a diameter of 1 to 2 μ were scattered in the cytoplasm. The cytoplasm of these cells showed few organelles with haphazardly arranged fine cytoplasmic filaments. The plasmalemma showed numerous small pinocytotic vesicles with focal cytoplasmic membrane densities.

Interspersed among tumor cells were occasional normal uveal melanocytes and macrophages. The dendritic uveal melanocytes (Fig. 11) appeared as spindle-shaped cells in sections with elongated ovoid nuclei. A layer of heterochromatin lined the nuclear membrane. Numerous oval melanosomes were present in their cytoplasm. Many of the melanosomes showed striated rods.

Macrophages (Fig. 12) were frequently observed in all cases. They were irregularly shaped, with usually eccentrically displaced, infolded nuclei. Their cytoplasms contained numerous phagosomes, which were bound by a single unit membrane and were filled with a fine, granular, medium-electron-dense material containing irregular melanin granules. Crystalloid structures were occasionally observed in a few phagosomes.

DISCUSSION

In our ultrastructural study of five melanocytomas, two types of tumor cells were found in each tumor. The morphologic features of these cells help to clarify some of the unusual behavioral characteristics of these tumors (Table 2).

The Type 1 melanocytoma cells consisted of the large ovoid polyhedral cells with centrally placed nuclei. Their abundant cytoplasm was filled with mature giant melanosomes. These cells, however, lacked prominent rough endoplastic reticulum, abundant free ribosomes, nuclear membrane infolding, and prominent nuclei. Cytologically, these heavily pigmented Type 1 cells appear to be benign and metabolically inactive, which may be correlated with the dense pigmentation and the lack of growth of most of these tumors.

Type 2 tumor cells were spindle-shaped cells and had a relatively high nucleus-cytoplasm ratio. They had a markedly infolded nuclear membrane and a more prominent nucleoli. In the cytoplasm of these cells were numerous mitochondria, prominent rough endoplasmic reticulum concentrically arranged in whorls, and an abundance of ribosomes. These features of Type 2 cells suggest an active metabolic rate.[8,9] We believe these cells account for the growth potential of some melanocytomas.

Fine cytoplasmic filaments, 5 to 7.5 nm, were only occasionally noted in the Type 2 cells but were prominent in the Type 1

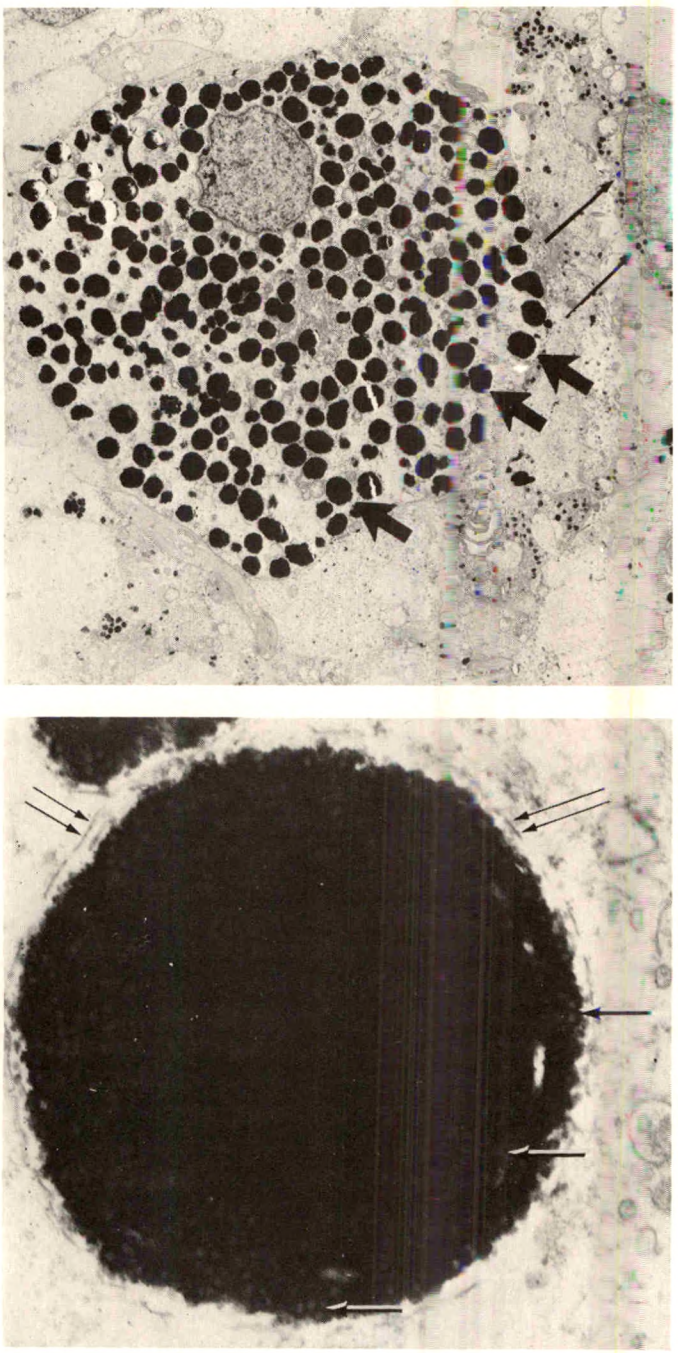


Fig. 4 (Juarez and Tso). Top, A large plump cell of melanocytoma (Type 1 variant) has an ovoid nucleus, numerous giant melanosomes (large arrows), and few cytoplasmic organelles. Compare the giant melanosomes (large arrows) with small melanosomes (small arrows) of an adjacent small spindle melanocytoma cell (Type 2) (×1,250). Bottom, A membrane-bound (double arrows) giant melanosome with a concentric array of 30 to 40 nm, electronlucent microvesicles, located at the periphery of the melanin granule (single arrows) (×22,720).

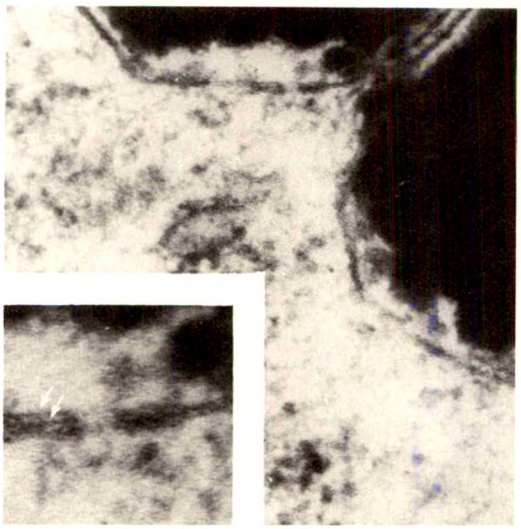


Fig. 5 (Juarex and Tso). A 7-nm thick plasma membrane surrounds each of the giant melanosomes (×40,000). Inset, Note trilaminar appearance at higher magnification (arrows), with inner and outer densely osmiophilic layers and an electronlucent layer in between (×86,000).

cells. Mishima[10] and Curran and McCann[11] observed that cytoplasmic filaments decrease reciprocally with the degree of cellular differentiation of melanoma. This observation indicates that the tumor cells of the Type 2 variant are less differentiated. Many morphologic features observed in these cells have also been noted in cells of uveal malignant melanomas,[12-14] such as the high nuclear cytoplasmic ratio, nuclei with marked infolding of the nuclear membrane, large conspicuous nucleoli, and prominent rough endoplasmic reticulum arranged in whorls. The melanocytoma of the optic disk had a disproportionately large number of Type 2 cells and by light microscopic examination, it was believed that malignant change to a spindle cell melanoma may have occurred. However, because the Type 2 cells were observed

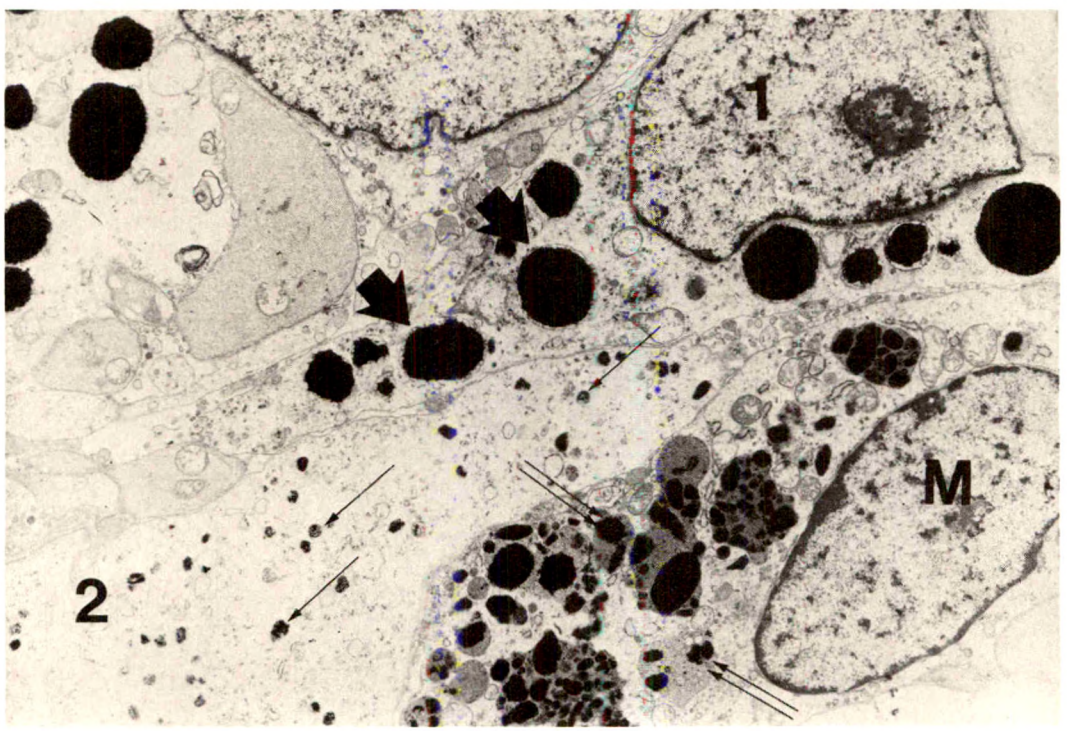


Fig. 6 (Juarez and Tso). Cells from a melanocytoma of the optic nerve head. Compare the giant melanosomes (thick arrows) of Type 1 melanocytoma cells (1), the small melanosomes (single thin arrows) in Type 2 melanocytoma cells (2), and phagosomes containing melanin granules (double thin arrows) in macrophages (M) (×8,000).

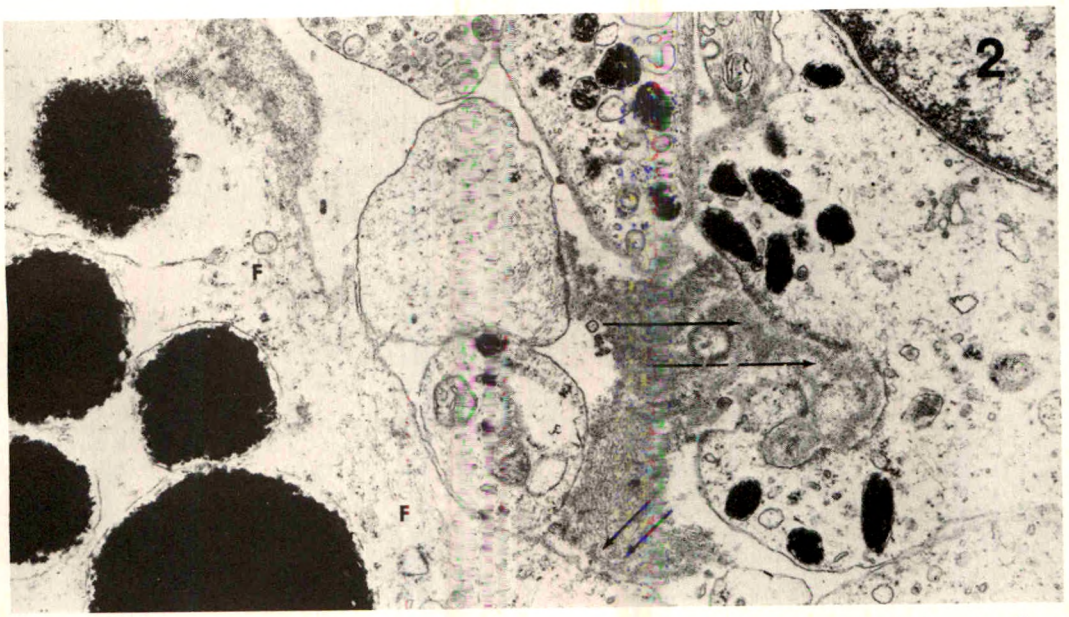


Fig. 7 (Juarez and Tso). Note fine cytoplasmic filaments (F) in Type 1 melanocytoma cells. Occasionally, a linear condensation of extracellular material indicates focal basement membrane formation in both Type 1 (small arrows) and Type 2 melanocytoma cells (2) (large arrows) (×9,600).

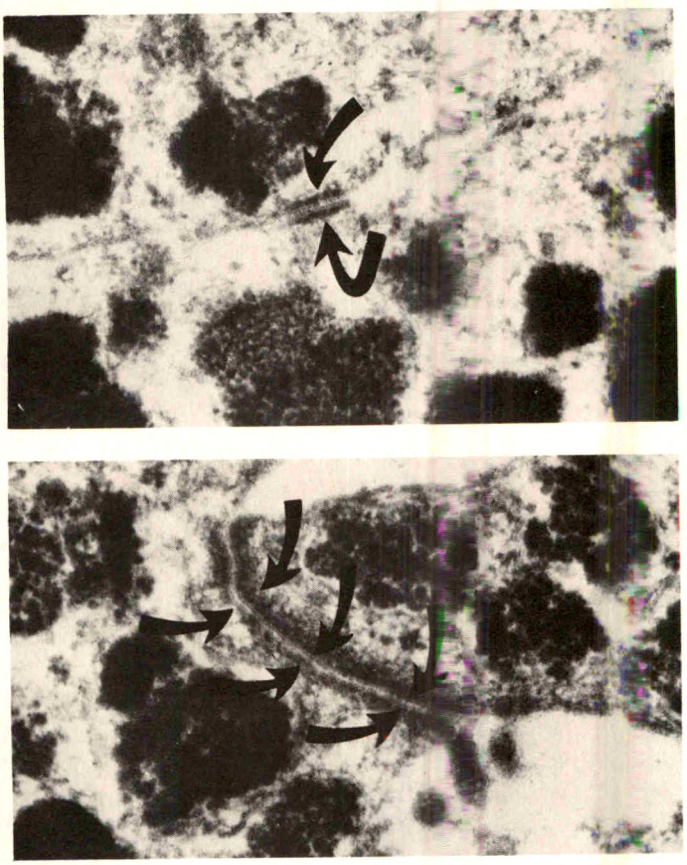


Fig. 8 (Juarez and Tso). Melanocytoma cells are occasionally atached by macula adherens type of cell junctions (arrows) (top, ×12,500; bottom, ×16,000).

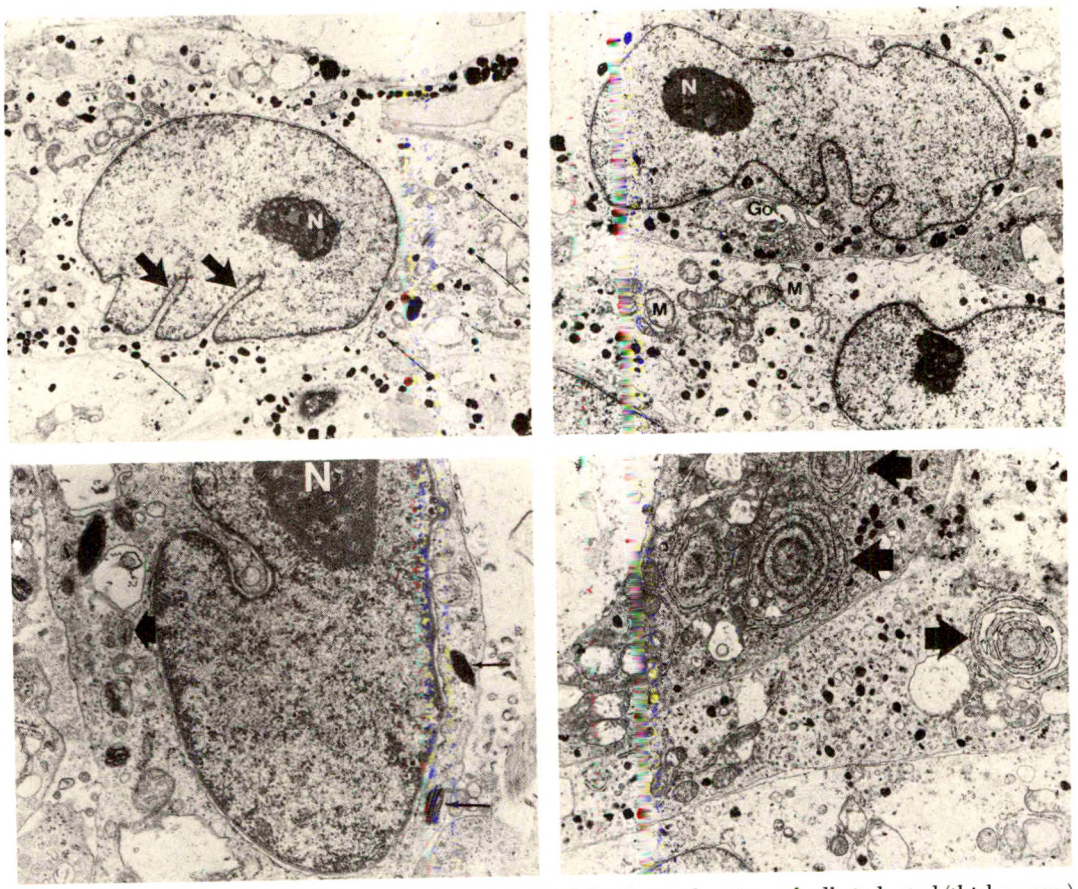


Fig. 9 (Juarez and Tso). Type 2 melanocytoma cell. Top left, The nucleus is markedly indented (thick arrows) with evenly distributed heterochromatin and a nucleolus (N). The latter shows a prominent network pattern. The cytoplasm shows small melanin granules (thin arrows) and numerous organelles (×6,400). Top right, Note the high nuclear cytoplasmic ratio and prominent organelles including many mitochondria (M), small melanin granules and golgi apparatus (Go) (×6,800). Bottom left, The melanosomes show different degrees of melanization. The immature granules (thick arrow) showed a recognizable irregular filamentous substructural matrix. In the more mature granules (thin arrows), the filaments were thick and assumed a rodlike configuration. Nucleolus (N) (×7,420). Bottom right, In the cytoplasm of these cells, the rough endoplasmic reticulum was frequently arranged in whorls (arrows) (×5,900).

in each of the melanocytomas examined, the suspected malignant transformation of this tumor may only be considered as within the spectrum of activity of these benign melanocytoma cells.

Melanocytomas do infiltrate the surrounding ocular tissues.[2] The advancing edge of these tumors is usually feathery on ophthalmoscopic examination and is less pigmented. In sections from these areas, Type 2 melanocytoma cells were more frequently observed. The melanosomes in these cells were few and smaller, and more frequently immature. The metabolically active, lightly pigmented cells cause the feathery advancing edge of melanocytoma.

The morphologic features of the giant melanosomes found in Type 1 tumor cells are distinctive and include the following:

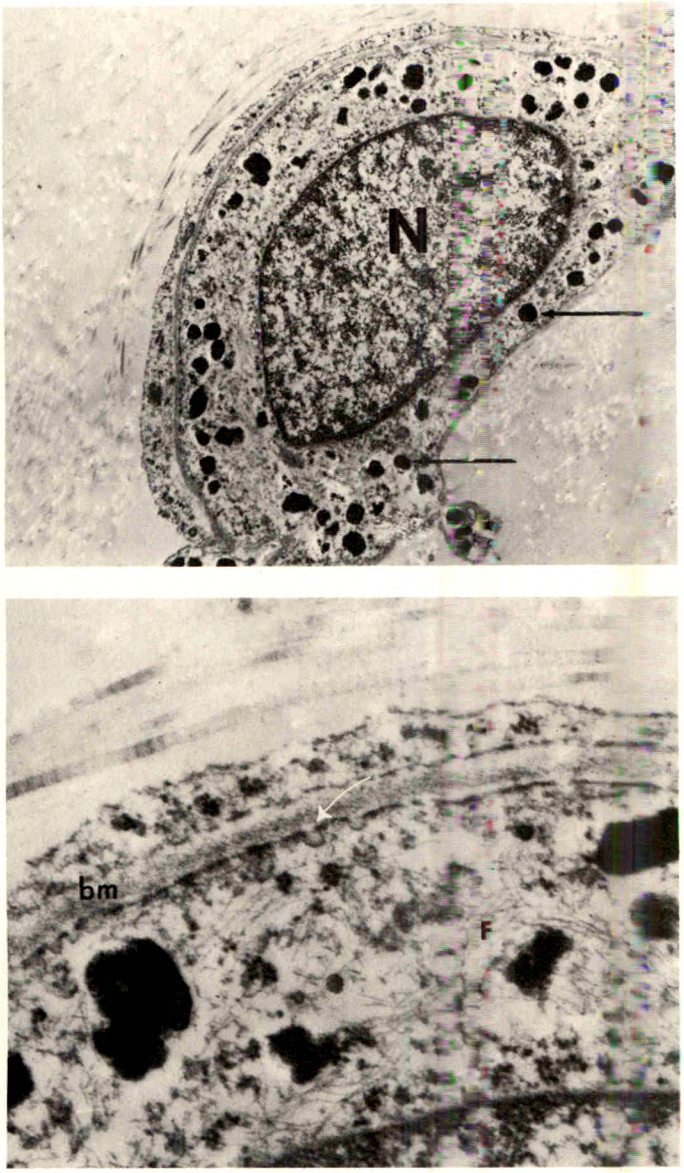


Fig. 10 (Juarez and Tso). An unusual pigmented cell in a ciliary body melanocytoma. Top, The nucleus (N) is centrally placed, containing clumped randomly dispersed heterochromatin. Small melanin granules measuring 1 to 2 μ are scattered in the cytoplasm (arrows). Around the cell a well-formed basement membrane is noted (×4,500). Bottom, haphazardly arranged fine cytoplasmic filaments (F) are noted in the cytoplasm. The plasmalemma shows pinocytotic vesicles (arrow) (bm designates basement membrane) (×10, 900).

1. Their large size. They range from 2 to 5 μ. The melanosomes present in benign pigmented nevi and malignant melanomas of the skin range in size from 0.1 to 0.4 μ.[11] The melanin granules of retinal pigment epithelial cells measure approximately 1 μ and those of the uveal melanocytes 0.3 to 0.5 μ in largest diameter.[15] The largest normal melanosomes of human skin measure up to 1.3 μ.[16] In our study the melanin granules of Type 1 cells are more than ten times larger than those of the Type 2 cells. Giant melanosomes described by others vary considerably in size. Konrad, Honigsmann, and Wolff[17] reported varia-

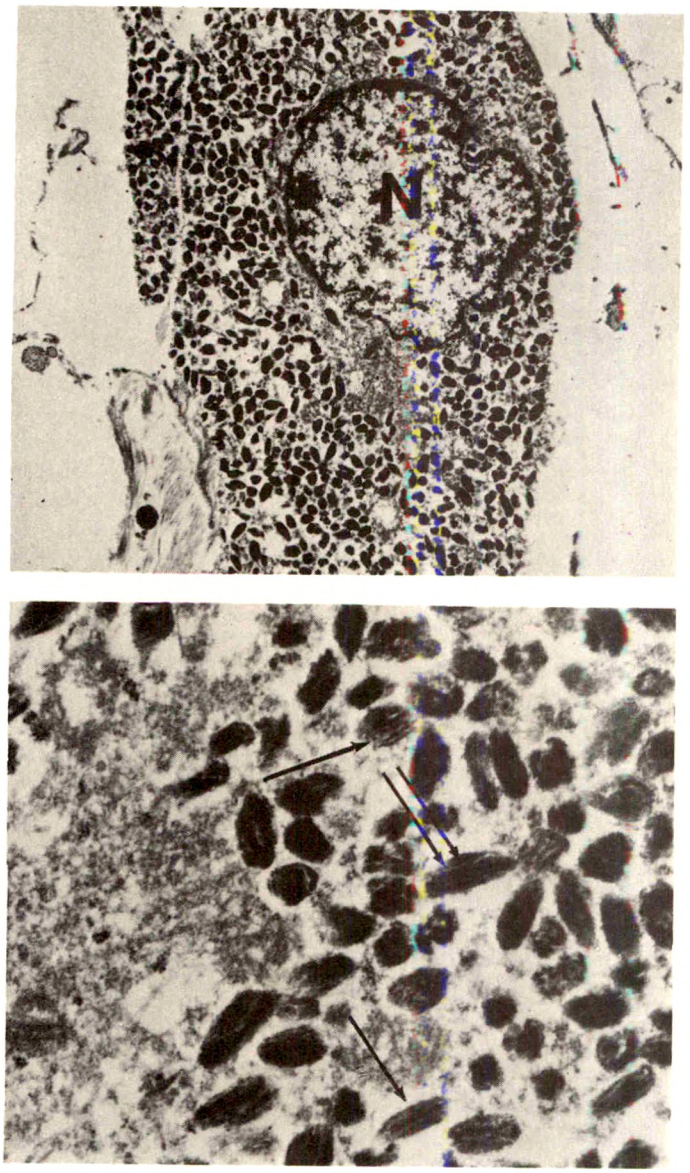


Fig. 11 (Juarez and Tso). Top, A normal uveal melanocyte is shown for comparison with the cells of the melanocytoma. Numerous small melanosomes 0.2 to 0.6 μ and relatively few organelles are present in the cytoplasm (×6,000). Bottom, Ellipsoidal melanosomes with striated rods in a normal uveal melanocyte (arrows) (×16,900).

tions from 1 to 6 μ; Jimbow and Fitzpatrick,[18] from 3.5 to 5 μ; and O'Donnel and associates,[19] up to 12 μ.

2. Microvesicular substructure. These electronlucent microvesicles, 30 to 40 nm in diameter, were concentrically arranged at the periphery of the giant melanosomes. They seemed to be the only substructural matrix in these melanosomes, because no filamentary matrix could be observed. In some giant melanosomes, they were less melanized and the microvesicular substructure could be more easily observed (Fig. 5). These microvesicles had been observed by Tousimis [20] in the normal melano-

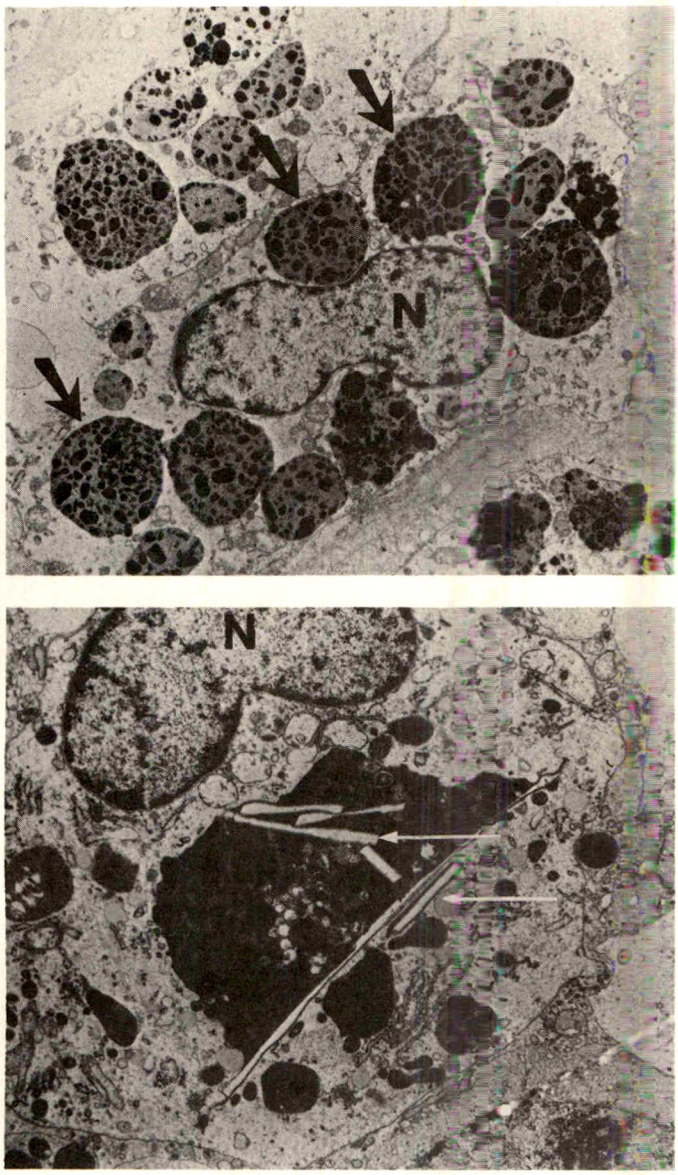


Fig. 12 (Juarez and Tso). Top, A macrophage with eccentric indented nucleus (N). The cytoplasm shows numerous phagosomes (arrows) containing irregular melanin granules (×3,820). Bottom, Crystaloid structures (arrows) and cellular debris from a necrotic pigmented cell observed in a macrophage; nucleus (N) (×6,800).

somes of the iris of rhesus monkeys. Subsequently, similar structures have been shown in the feather melanosomes of *Lamprotornis* (a starling),[21] in the melanosomes of human melanoma cells,[22] and in normal black hair.[23]

3. Plasma membrane envelope. A thin 7-nm thick membrane, consisting of three layers: a central electronlucent layer and outer densely osmiophilic layers surrounded the giant melanosome.

These large melanosomes have been described as "giant melanosomes," "abnormally large melanin granules," and "macromelanosomes." They have been seen in the following entities: café-au-lait

lesions in neurofibromatosis[24]; nevus spilus[17,25]; nevoid pigmentation[26]; leopard syndrome[27]; generalized melanosis secondary to malignant melanoma[28]; x-chromosome-linked ocular albinism[19]; and focal hypertrophy of the retinal pigment epithelium.[29] To explain the formation of these giant granules, Konrad, Wolff, and Honigsmann[25] believe that they originate from an aberrant pathway in the morphogenesis of the melanosomes. The giant melanosomes described in Chediak-Higashi syndrome[30,31] and beige mice[32] appear to differ from the giant melanosomes of the melanocytoma. In these inherited disorders the giant granules are structurally different, because they seem to result from the fusion of individual normally sized granules that are grouped together within a single unit membrane.

The melanosomes of the Type 2 variant of tumor cells were smaller, elongated, and showed different stages of melanization. The immature granules showed a filamentary substructural matrix. These melanosomes appear to be structurally similar to those seen in junctional, compound, and intradermal nevus and in malignant melanomas of skin and uveal malignant melanomas.[33] In the more mature granules, the filamentous matrix attained a rodlike configuration. Irregularities in melanosomes have been suggested by Mishima[10] as being potentially characteristic of malignancy, but in an ultrastructural study comparing the melanin granules in uveal malignant melanomas of different cell types, Iwamoto, Jones, and Howard[12] did not notice any correlation between pigment granules and cell types.

Macula adherens or desmosomelike cell junctions, such as those observed in the melanocytomas, have been previously observed in benign pigmented nevi and malignant melanomas.[10] The description of these types of cell junctions in benign and malignant neoplasms of epithelial, mesenchymal, and neuroectodermal origin are numerous.[34] Likewise, the presence of focal basement membrane production seen within tumor cells has been previously noted in benign and malignant melanocytic tumors.[8-36]

Multiple melanocytomas within the same eye are unusual. Crawford[37] described a case of optic nerve melanocytoma with 47 years of follow-up observation, in which postmortem examination showed a second melanocytoma in the ciliary body. In our Case 1, two melanocytomas were widely separated in the uveal tract and there was little possibility of tumor seeding. The significance of this observation is unknown. There was a third type of cell present in one of the ciliary body melanocytomas. Except for the melanin granules, the presence of fine cytoplasmic filaments, pinocytotic vesicles, focal densification of cytoplasmic membrane and well developed basement membrane, they resembled smooth muscle cells. They resemble somewhat the clump cell Type 2 of Koganei.[38]

Summary

We examined by light and electron microscopy five melanocytomas from four patients. Two types of cells were observed in each tumor. The predominant cell type in most of the tumors studied consisted of plump polyhedral nevus cells that contained numerous giant melanosomes. These cells showed advanced differentiation. They appeared to be metabolically inactive and to have been the cause of the heavy pigmentation and benign nature of these tumors. The second variant of melanocytoma cells were smaller spindle-shaped cells that were lightly pigmented. Other morphologic features of these cells such as

high nucleus-cytoplasm ratio, prominent nucleolus, nuclear membrane infolding, numerous mitochondria, prominent rough endoplasmic reticulum, and free ribosomes, indicated a metabolically active cell, which may explain the infiltrating behavior of these tumors.

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Ophthalmic Miniature

Autophthalmoscopy—The inventions of Prof. Coccius of Leipsic, Dr. Giraud-Teulon, of Paris, Dr. F. Heyman, of Dresden, and N.W. Zehender, of Bern have lately "given us the gift to see ourselves as others see us." But the power of viewing one's own optic disk and retinal vessels is not simply a physiological wonder. It gives the opthalmic student opportunity to study for himself by himself. Indeed, a good method of training himself in ophthalmoscopy is, to become by practice au fait at examining his own fundus oculi. He can also thereby learn what to see and how to see it. Although autophthalmoscopy is but in its infancy, yet we already have several different forms of the instrument, both for the upright and inverted image. It would not be safe to predict the results that may be developed by the autophthalmoscope. One thing let us note. It sprung from philosophic ophthalmologists applying the laws of optics to physiology. It was not stumbled on, but wrought out by patient investigation and experiment.

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OCULAR INFLAMMATION IN REITER'S DISEASE AFTER *SALMONELLA* ENTERITIS

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Ocular inflammation after *Salmonella* enteritis is uncommon. A few cases of iritis, choroiditis, and panophthalmitis of metastatic origin have been noted [1-5] Single cases of conjunctivitis occurring eight to 12 days after the onset of diarrhea as a part of incomplete or complete Reiter's disease have been reported after *Salmonella typhimurium*[6,7] or *S. enteritidis* [8-10] infection, and one case of conjunctivitis, iritis, and arthritis six months after *S. typhimurium* infection.[11] However, there has been little data published regarding detailed ophthalmological findings concerning the ocular inflammation after *Salmonella* infection. We studied the clinical appearance of conjunctivitis and uveitis in Reiter's disease after *Salmonella* infection in eight patients.

SUBJECTS AND METHODS

Eight patients, two men and six women, ranging in age between 18 and 61 (mean, 36) years were included in this study. Bacteriologic confirmation was accepted if *Salmonella* was isolated from the feces at least twice. In serologic tests, the criterion of recent *Salmonella* infection was a maximum agglutinin (Widal) titer of at least 160, combined with a significant rise of titer (at least twofold dilution steps when tested in parallel), or followed by a significant decrease of the titer within six months, or both. The patients underwent a medical and ophthalmologic examination and follow-up. Determination of HLA-antigens using the two-stage microcytotoxicity method[12] was done in all patients.

RESULTS

In all patients the disease was characterized by acute onset with diarrhea lasting from one day up to three weeks. Six patients had prolonged remittent fever with a duration up to six months and a maximum between 37.6 and 40°C (mean 38.6°C). Headache occurred in three patients (Table 1), two of whom (Cases 5 and 6) had marked neurologic and mental symptoms during the acute stage of the infection. One patient (Case 4) had emesis, abdominal pain, and melena; and one patient (Case 5) had

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TABLE 1

SURVEY OF THE CLINICAL PATTERN*

Patient No.,	Age (yrs)	Sex	Diarrhea	Fever (°C)	Headache	Arthritis	Myalgia	Urethritis	SI	Conjunctivitis	AAU
1	61	F	+	39.2	−	+	−	+	−	+	−
2	35	F	+	−	−	+	−	+	+	+	−
3	29	M	+	−	−	+	+	+	+	+	−
4	30	F	+	40.0	+	+	+	+	−	+	−
5	18	F	+	38.5	+	+	+	+	+	+	−
6	29	F	+	37.8	+	+	+	−	−	+	+
7	40	M	+	37.6	−	+	+	−	−	+	+
8	46	F	+	38.7	−	+	+	+	+	−	+

*+designates symptom was present; −, symptom absent; SI, clinical sacroiliitis; and AAU, acute anterior uveitis.

emesis, hematemesis, epistaxis, dyspnea, and convulsions. Another patient (Case 6), had peripheral neuritis affecting the right arm, tinnitus, vertigo, and transient constriction of the visual fields; and one other patient (Case 8) had bilateral pneumonia and nephrotic syndrome with pitting edema and proteinuria during the acute stage of the infection. From five days to one month after onset of the illness, all patients developed acute asymmetric polyarthritis involving usually the larger joints of the lower extremities. Six patients developed myalgia, and six patients had urethritis with dysuria or pyuria (Table 1). During the acute stage of the disease one patient (Case 3) developed carditis with second-degree atrioventricular block. Symptoms of sacroiliitis occurred in four patients (Table 1); one of them (Case 3) developed ankylosing spondylitis during the subsequent five years.

Conjunctivitis occurred in seven patients. Four patients (Cases 1 to 4) had conjunctivitis from one to two weeks after the onset of *Salmonella* infection. One patient (Case 5) with a long remittent active stage of the disease developed conjunctivitis three months after the onset of the illness. One patient (Case 6) with continuing severe symptoms progressing to a chronic disease after the intitial attack had conjunctivitis three years, and acute anterior uveitis 3½ years after the onset of the illness. One patient (Case 7) had conjunctivitis two years, and acute anterior uveitis four years after the initial attack; and one patient (Case 8), with persisting symptoms progressing to a chronic disease, had acute anterior uveitis three years after the onset of the illness.

Conjunctivitis was unilateral in two cases and bilateral in five cases (Table 2). The patients complained of redness and a burning sensation. Except in two patients (Cases 4 and 5), conjunctivitis was mild with conjunctival injection but no chemosis, follicles, or keratitis, and resolved spontaneously in three to seven days. One patient (Case 4) complained ten days after onset of diarrhea of severe smarting in both eyes showing palpebral edema, severe injection, and chemosis in the conjunctiva, and purulent exudate. In this case chloramphenicol eyedrops and ointment did not provide any improvement, and the symptoms subsided within ten days. In five patients (Cases 1

TABLE 2

CLINICAL FEATURES OF CONJUCTIVITIS FOLLOWING *SALMONELLA* INFECTION*

Patient No.	Side	Conjunctival Injection	Chemosis	Follicles	Keratitis, Corneal Erosion	Subsequent AAU	Arthritis	Urethritis
1	B	+	−	−	−	−	+	+
2	U	+	−	−	−	−	+	+
3	B	+	−	−	−	−	+	+
4	B	+	+	−	−	−	+	+
5	B	+	−	+	+	−	+	+
6	B	+	−	−	−	+	+	−
7	U	+	−	−	−	+	+	−

*U designates unilateral; B, bilateral; AAU, acute anterior uveitis; +, symptom present; and −, symptom absent.

to 5) conjunctivitis coincided with arthritis and urethritis, and in two patients (Cases 6 and 7) with arthritis.

An 18-year-old woman (Case 5) became ill in October 1978, with diarrhea, prolonged remittent fever lasting for six months, headache, and polyarthritis. In November 1978, she had dyspnea and convulsions, and in December 1978, emesis, hematemesis, and epistaxis. On Jan. 17. 1979, she developed acute conjunctivitis with severe smarting and redness, and had simultaneous symptoms of urethritis, lower back pain, and polyarthritis. Both eyes showed injection and mucopurulent exudation. The inferior parts of both corneas showed epithelial edema. Chloramphenicol eyedrops were prescribed. Two days later the right cornea was clear; in the left eye the corneal edema had decreased in area and stained only lightly, but three days later the area of the epithelial edema in the left cornea had increased. Severe smarting, lacrimation, redness, mucopurulent exudation, and blepharospasm continued in the left eye. On Feb. 1, 1979, she developed a large erosion taking fluorescein stain in the inferior part of the left cornea. During the treatment with chloramphenicol eyedrops and ointment, the erosion healed in five days and epithelial edema had disappeared ten days later. Lacrimation, smarting sensation, redness, and mucopurulent exudation continued, and hypertrophic follicles and chemosis of the conjunctiva appeared in both eyes. On March 13, 1979, the right cornea showed punctuate epithelial erosions staining lightly. One week later the right cornea was clear. There were no aqueous flare or cells. Conjunctival smears and cultures for bacteria, virus, and fungi were negative. Schirmer test showed bilateral wetting of more than 30 mm/five minutes. Starting March 9, 1979, because of severe painful joint symptoms and lower back pain, she took prednisone orally for six months and, beginning April 4, 1979, she took azathioprine for seven months. During this time she had small conjunctival injection in both eyes but no corneal changes were seen.

Three patients (Cases 6 to 8) developed unilateral acute anterior uveitis from three to four years after the onset of the illness. They complained of redness, photophobia, and pain. One of them had (Case 7) increased lacrimation and two (Cases 6 and 8) had reduced vision. All

patients had pericorneal ciliary injection, aqueous flare and cells, fine keratic precipitates, fibrinous exudation in the aqueous humor, and posterior synechiae in the right eye. One patient (Case 6) showed cells in the vitreous and slight macular edema and slight edema of the disk. The inflammatory signs resolved completely during topical administration of corticosteroids and cyclopegics in five weeks.

Stool cultures yielded *S. enteritidis* in two patients (Cases 6 and 7) and *S. typhimurium* in three patients (Cases 1, 4, and 8) with increased agglutinin titers for *Salmonella* antigens (Widal) in two of them (Cases 1 and 4). Three patients (Cases 2, 3, and 5) had increased agglutinin titers for *Salmonella* O-antigen without positive isolation of *Salmonella* from the stools. Stool cultures for *Shigella*, stool cultures and serologic test titers for *Yersinia*, as well as serologic test titers for *Chlamydia*, were negative in all patients. The peak erythrocyte sedimentation rate values varied between 45 and 150 mm/hr (mean, 98 mm/hr). The highest leukocyte counts were between 7,200 and 11,600/mm^3 (mean, 9,260/mm^3 (Table 3). Latex, Waaler-Rose, and antinuclear antibody titers were negative in all patients. Antistreptolysin O titer was normal in all cases except Cases 5 and 6 with positive antistreptolysin titers of 250 and 400 respectively. HLA-B27 antigen was found in all eight patients.

One patient (Cases 6) provided details of her relatives, three of whom had complete Reiter's disease (Figure).

Discussion

Reiter's disease after *Salmonella* infection occurs rarely; it occured only in one case in an epidemic afflicting 2,800 patients.[10] In this study of eight patients, six had complete and two had incomplete Reiter's disease after *Salmonella* enteritis.

Conjunctivitis occurred in seven cases. Except in one case, it was mild and self-limiting, lasting from three to ten days. One patient with transient episodes of conjunctivitis that lasted for 11 months had transient episodes of keratitis and corneal erosion for two months, but which left no scar or residua. To the best of our knowledge, this is the first case of keratitis or corneal erosion reported after *Salmonella* enteritis.

Three patients developed acute anteri-

TABLE 3

Laboratory findings in patients with *Salmonella* infection and eye inflammation*

Patient No.	Isolation of *Salmonella*	Agglutinin Titer for *Salmonella* (Widal test)	Peak ESR (mm/hr)	Highest Leukocyte Count/mm^3	Latex	Waaler-Rose	ANA	ASO	HLA-B27
1	S. *typhimurium*	160	150	11,600	–	–	–	–	+
2	–	160	67	7,200	–	–	–	–	+
3	–	160	91	7,500	–	–	–	–	+
4	S. *typhimurium*	320	45	11,200	–	–	–	–	+
5	–	320	66	11,000	–	–	–	250	+
6	S. *enteritidis*	–	116	7,200	–	–	–	400	+
7	S. *enteritidis*	–	100	7,800	–	–	–	–	+
8	S. *typhimurium*	–	145	10,600	–	–	–	–	+

*+ designates positive finding; –, negative; ESR, erythrocyte sedimentation rate; ANA, antinuclear antibody titer; and ASO, antistreptolysin O titer (<200).

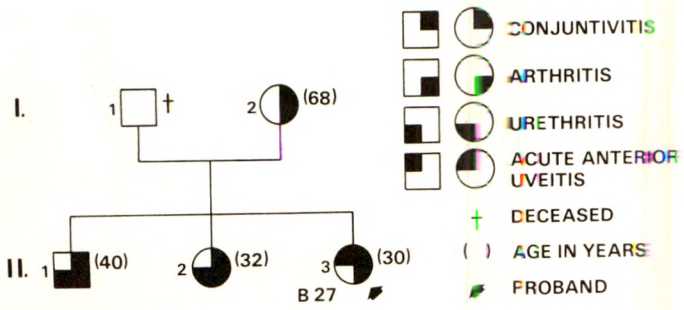


Figure (Saari and associates). Pedigree of family showing proband (Case 6) with HLA-B27 and conjunctivitis, acute anterior uveitis, and arthritis after *Salmonella* infection, and cases of complete or incomplete Reiter's disease in the same family.

or uveitis with clinical characteristics similar to those seen in HLA-B27 associated[13] and familial[14] acute anterior uveitis. In our patients uveitis occurred from three to four years after the onset of the disease. However, in Reiter's disease the eye lesions may not be seen until years after the first attack, suggesting that the initial incomplete form of the syndrome is essentially of the same nature as the fully developed one.[15]

All patients in the study had HLA-B27 antigen, which has been reported in two cases of Reiter's disease after *Salmonella* infection.[7,10] These patients show that Reiter's disease may become manifest after *Salmonella* infection in individuals with a genetically determined predisposition indicated by HLA-B27. However, the presence of HLA-B27 in all cases in this study suggests that both complete Reiter's disease (Cases 1 to 5, and 8) and incomplete Reiter's disease (Cases 6 and 7) may be varying clinical expressions of the same underlying pathogenetic process. HLA-B27 occurs in 76 to 83% of patients with Reiter's disease,[16,17] which shows clear familial tendency.[18]

Four of our patients with Reiter's disease after *Salmonella* infection developed clinical symptoms of sacroiliitis, which was seen also in 12 of 23 patients with ocular inflammation associated with *Yersinia* infection.[19] This suggests that sacroiliac joint involvement may be associated with certain infections in patients with HLA-B27.

Summary

We studied characteristics of ocular inflammation in Reiter's disease after *Salmonella* enteritis in eight patients. After an acute onset with diarrhea, fever in six patients, and headache in three patients, all patients developed arthritis; six patients had myalgia; six patients, urethritis; and one patient, carditis. Sacroiliitis was found in four patients. All patients had HLA-B27 antigen. Conjunctivitis occurred in seven patients. It was mostly mild with no chemosis, follicles, or keratitis, and resolved in ten days. In one case palpebral edema, chemosis in the conjunctiva, and purulent exudate were seen. One patient had transient episodes of keratitis and corneal erosion for two months and episodes of conjunctivitis for 11 months. Three patients developed unilateral acute anterior uveitis with aqueous flare, cells, fine keratic precipitates, and fibrinous exudation from three to four years after the onset of the illness. One of these patients had vitritis and macular and papillary edema.

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VOGT–KOYANAGI–HARADA SYNDROME

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Vogt-Koyanagi-Harada syndrome, the uveomeningeal syndrome, is a bilateral panuveitis associated with meningeal and cutaneous findings.[1] We present our clinical findings in 20 new cases of Vogt-Koyanagi-Harada syndrome treated at a Midwestern ophthalmology center.

SUBJECTS AND METHODS

The following criteria for diagnosing Vogt-Koyanagi-Harada syndrome were presented and discussed at the second annual meeting of the American Uveitis Society, in Kansas City, Missouri, October 1978:

1. The patient should have no history of previous ocular trauma or surgery.
2. One finding from at least three of the following groups of signs should be present:
   a. Bilateral chronic iridocyclitis.
   b. Posterior uveitis, including exudative retinal detachment, forme fruste of exudative retinal detachment (that is, disk hyperemia or edema, subretinal macular edema), and "sunset glow" fundus.
   c. Neurologic signs of tinnitus, neck stiffness, cranial nerve or central nervous system problem, or cerebrospinal fluid pleocytosis.
   d. Cutaneous finding such as alopecia, poliosis, or vitiligo.

All 20 of the patients included in this report fulfilled the above criteria. Patients were examined at least three times by one or both of us. Seventeen of these patients were seen in the Uveitis Clinic here between September 1973 and March 1977. Three additional patients were not registered in the clinic. The data presented are based on a follow-up of at least one year.

Eight (40%) of our patients were male, and 12 (60%) were female. Their ages ranged from 10 to 56 years, with a mean of 39.7 years. The mean age at onset of symptoms was 33.8 years. These data closely correspond to those previously reported.[1-3]

Ancestry—Of our 20 patients, 11 were black, seven were Hispanic, and two were white. Oriental racial type is significant, assuming that American Indian and Hispanic ancestries indicate Oriental forebearers. Seven of the 11 blacks in our series had American Indian ancestry. One black patient was an orphan of unknown ancestry. Seven patients were Latin-Americans from Mexico, Central America, or Puerto Rico. Two of these seven had known American Indian ancestry. Of our two white patients of northern European ancestry, one definitely had American Indian forebearers. No patients in our series had Japanese or Chinese ancestry. Thus, 15 of 20 patients have American Indian or Latin American ancestry. This confirms previous reports that document a predilection for darkly pigmented races or Oriental ancestry.[1-11]

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Discussion

Frequency—Seventeen of the 20 cases reported here represent 3.7% of the 455 referred uveitis patients seen in the Uveitis Clinic between September 1973 and March 1977. Although Japanese authors reported an incidence of about 7% of Vogt-Koyanagi-Harada syndrome in their uveitis clinics,[4,12] our incidence of nearly 4% indicates that the disease may not be as unusual in this country as previously reported.

Cause and pathogenesis—The cause of this disease remains unknown despite reports implicating viruses,[13] bacteria,[14] and fungi.[15,16] A genetic predisposition has been suggested by the finding of Vogt-Koyanagi-Harada syndrome in three siblings.[17] An increased incidence of HLA-type LD-Wa and B (Sa-1) (Japanese version of HLA-Bw22) has been reported from Japan.[12,18] However, these cell surface antigens were not found in nine patients from San Francisco[5] or in 14 of our patients.[19]

Immunologic factors seem to be important. We have shown an antiganglioside antibody in the serum of our patients.[19] Further immunologic data have been shown by investigators from Israel[20] who found cell-mediated immunity to human myelin basic protein in patients with Vogt-Koyanagi-Harada syndrome. Hypersensitivity to neural tissue could represent the common cause of this multisystem disease or it could represent a secondary reaction to nonimmunologically altered neural tissue. Lymphocyte sensitization to uveal antigen or melanin has been shown by several authors.[21-27] We showed increased levels of IgD in patients.[19]

Ocular clinical findings, onset—The incidence of clinical findings is listed in Table 1. Eighteen of our 20 patients had both anterior and posterior inflammation. One patient had only anterior inflammation (Vogt-Koyanagi type) and one patient had only posterior involvement (Harada type), but both developed the typical neurologic and cutaneous findings that complete the diagnosis. Anterior segment disease preceded posterior segment disease by four weeks in one patient. Four patients had disease that began posteriorly and progressed anteriorly throughout a period of one to 11 months. Ten patients had symptoms that began concurrently anteriorly and posteriorly. Most patients had bilateral disease, although inflammation in the second eye was not evident for weeks to years in some cases.

TABLE 1

Incidence of clinical findings in 20 patients with Vogt-Koyanagi-Harada syndrome

Findings	No. of Patients
Exudative retinal detachment or marked macular edema	19
Meningeal signs or symptoms	14
Keratic precipitates	9
Vitiligo	8
Posterior synechiae	8
Cataracta complicata	8
Vascular sheathing	7
Poliosis	7
Madarosis/alopecia	7
Hyperemia or swollen optic disk	5
Koeppe/Busacca nodules	4
Peripheral anterior synechiae	3
Choroidal detachment	1
Phthisis bulbi	1

Anterior segment inflammation—Classically, the syndrome is described as a bilateral panuveitis. Anterior signs may vary from almost no reaction (Harada type) to a severe aqueous cellular and proteinaceous reaction with mutton fat keratic precipitates, iris precipitates and nodules, peripheral anterior synechiae, and posterior iris synechiae (Vogt-Koyanagi type).

Inflammation of the anterior segment tended to persist for several months after posterior disease subsided. Chronic iridocyclitis, which required treatment

with topically and subconjunctivally administered corticosteroids as well as cycloplegics, continued for more than two years in several cases. Posterior subcapsular cataracts probably resulted from prolonged uveitis combined with corticosteroid therapy.

Posterior uveitis—Of our 19 patients with posterior involvement, 14 had exudative retinal detachments. When detachment occurred, the macula was usually involved, resulting in decreased visual acuity to a level of light perception or hand motions (Fig. 1). We believe a forme fruste exudative detachment was present in the remaining five patients. These patients had subretinal fluid in the macular region that caused wrinkling and folds (Fig. 2). Disk hyperemia and edema were also present. Possibly the early high dose of periocular and orally administered corticosteroids prevented frank detachment in these patients. One patient (not included in this study), who was treated with systemic corticosteroids for frank detachment in one eye, developed typical pigmentary changes of old Vogt-Koyanagi-Harada syndrome in the presumed normal fundus of the contralateral eye without an intervening episode of detachment. Alternatively, these cases of forme fruste detachment might represent mild forms of the disease. Manor[28] and G. R. O'Connor (personal communication, May 10, 1977) believe that incomplete forms of Vogt-Koyanagi-Harada syndrome are more common than is generally recognized.

Most patients responded favorably to aggressive periocular and orally administered corticosteroids (prednisone, 100 ± 20 mg/day). Nonrhegmatogenous detachments flattened in less than four months of treatment in 13 cases, with an average time of 2.3 months. Even before detachments had completely resolved, visual acuity improved significantly. Corticosteroid therapy was tapered as the severity of the disease decreased. Permanently decreased visual acuity most commonly was caused by scarring of the macula upon settling of the detachment.

Vasculitis—Vascular sheathing was noted in seven of our patients (Fig. 3). This agrees with one of the original

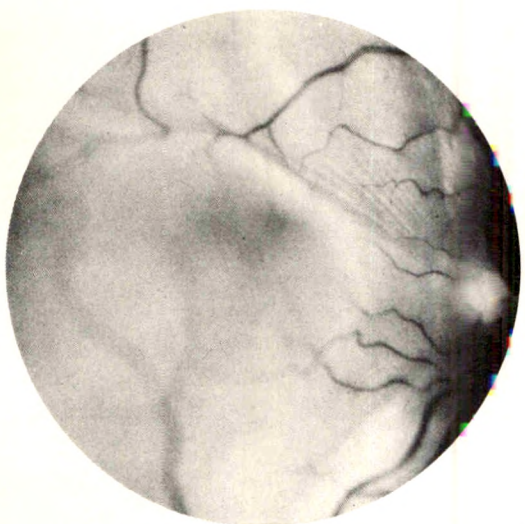


Fig. 1 (Snyder and Tessler). Exudative retinal detachment with macular involvement.

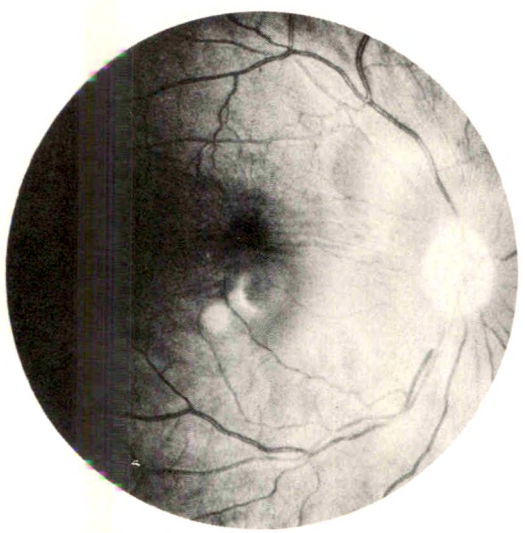


Fig. 2 (Snyder and Tessler). Forme fruste of exudative detachment with subretinal fluid in macula causing wrinkling and folds. White lesions below macula are light reflexes.

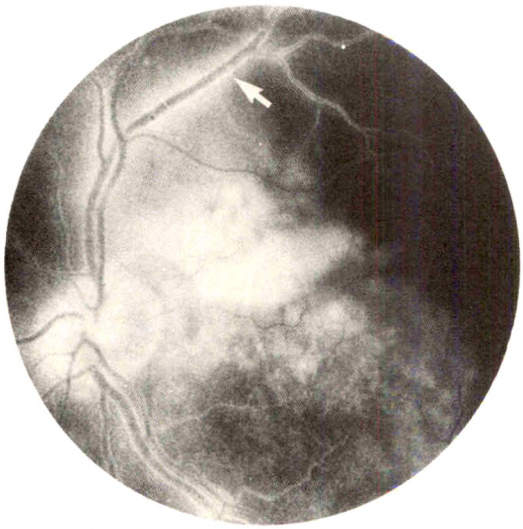


Fig. 3 (Snyder and Tessler). Periphlebitis (arrow) on fluorescein angiogram. Note characteristic subretinal leakage areas in posterior pole.

descriptions of the disease by Okamura,[29] that "in most cases, vessels near the disc show perivasculitis" and "peripheral periphlebitis occurs two to five months" after the onset of disease. This finding is not universal, as Kantor and Goldberg[30] and Shimizu[12] do not mention vasculitis in their reports.

The VDRL test results were positive in three of our patients, and the serum lysozyme level was increased in one.

Subretinal neovascularization—One patient, an 11-year-old black girl, developed a subretinal peripapillary neovascular membrane involving the papillomacular bundle (Fig. 4). Subretinal blood extended to the macular area. A similar finding has been described by Carlson and Kerman[31] and Ronald Smith (personal communication, Oct. 5, 1977).

Effect of diabetes mellitus and other vasculopathies—Three patients had preexisting diabetes mellitus not induced by corticosteroids. An additional four patients had immediate family members with diabetes mellitus. One patient with frank diabetes had background diabetic retinopathy and neovascularization of the disk. When her exudative detachment settled, total regression of the neovascular tissue and background retinopathy occurred. Hemoglobin AS and SC, noted in two patients, had no deleterious effects. This is in contrast to the progression of neovascularization when proliferative diabetic retinopathy or sickle cell retinopathy are combined with inflammatory vasculitides such as sarcoid-

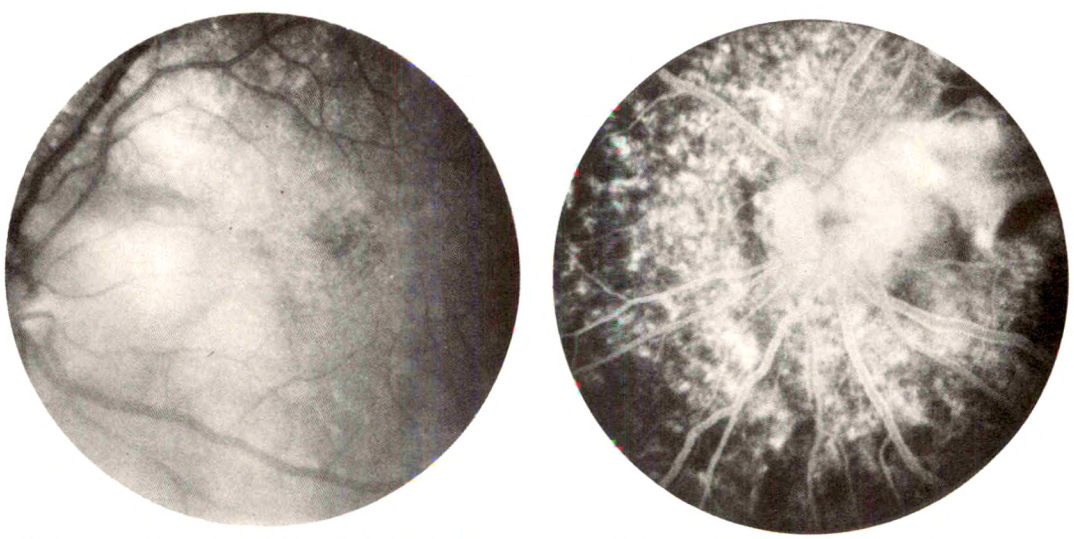


Fig. 4 (Snyder and Tessler). Left, Subretinal neovascular membrane off of disk with resulting intraretinal hemorrhage. Right, Fluorescein angiogram of subretinal neovascular membrane in papillomacular bundle.

osis,[32,33] in which the neovascularization seems to progress faster. The histologic findings in Vogt-Koyanagi-Harada syndrome after settling of the serous retinal detachment are strikingly similar to those found in retinas after laser photocoagulation.[3,34]

Areas of retinal pigment epithelial breakdown with choroidal-retinal fusion may prevent further retinal detachment.[3] Breakdown of the perfusion barrier between retina and choroid possibly precludes progression of neovascularization by allowing oxygen and nutrients from the choroidal circulation to nourish ischemic retinal tissues.[35]

Effect of pregnancy—Three patients in our series became pregnant during the active course of their disease. Two were Latin-American women who went into complete remission and for whom all medication was tapered and discontinued one month before delivery. Both patients had a flare-up of the iridocyclitis, but a detachment of the retina did not recur in the immediate postpartum period. One black woman who initially had visual acuity of 6/6 (20/20) in the left eye at four months of gestation developed minimal macular edema by the eighth month, which subsided before delivery. A flare-up after delivery decreased visual acuity to light perception, owing to serous retinal detachment.

Both cellular and humoral immunity are depressed during pregnancy, especially in the late stages.[36-40] An alpha-globulin in the serum of pregnant women has been shown to suppress cellular immunity.[41,42] Perhaps this alpha-globulin along with endogenous corticosteroids, progesterone, and alpha-fetoprotein caused the late remissions seen in our patients. Upon termination of pregnancy, these factors disappeared, possibly accounting for the postpartum flare-up.

Neurologic findings—The meningeal signs and symptoms that often precede or occur at the onset of the disease (prodromal) are usually of short duration, lasting from a few days to a few weeks. These meningeal signs include severe headache, neck stiffness, and fever. Transient dysacusis with tinnitus, ranging from minimal changes found only on audiometric examination to profound deafness, frequently occur along with nausea and vertigo. Cerebrospinal fluid pleocytosis may corroborate the meningeal findings. Hyperthermia, delerium, convulsions, ataxia, cranial nerve palsies, and coma have been reported in patients with Vogt-Koyanagi-Harada syndrome.[43,44]

Thirteen of our 20 patients had meningeal symptoms and nine patients had dysacusis. One of our patients was undergoing psychotherapy. Any patient with Vogt-Koyanagi-Harada syndrome and neurologic findings should receive a careful examination to eliminate multiple sclerosis, with which retinal vasculitis, choroiditis, optic neuritis, and pars planitis have been associated.[45-47]

Cutaneous findings—Cutaneous signs of poliosis, alopecia, and vitiligo are not constant and usually occur weeks to months after the onset of ocular disease. At least one cutaneous sign was found in 11 of our 20 patients. Vitiligo was present in eight patients. In one patient, vitiligo preceded ocular disease by ten years. Recent speculation indicates that the presence of vitiligo may depend on the melanin of the eye and skin being identical.[48]

Visual outcome—Sixteen of our 20 patients have visual acuity of 6/21 (20/70) or better in at least one eye (Table 2) after follow-up of two to five years. One patient with bilateral visual acuity of light perception developed bilateral phthisis bulbi before she was referred to us from Mexico.

Summary

We studied 20 patients with Vogt-Koyanagi-Harada syndrome. They represented 3.7% of referred patients with

TABLE 2

INITIAL AND LATEST VISUAL ACUITY*

Patient No.	Initial Visual Acuity		Latest Visual Acuity	
	R.E.	L.E.	R.E.	L.E.
1	6/12 (20/40)	6/21 (20/70)	6/21 (20/70)	6/21 (20/70)
2	6/60 (20/200)	6/60 (20/200)	6/15 (20/50)	6/60 (20/50)
3	CF	6/60 (20/200)	CF	6/60 (20/200)
4	6/6 (20/20)	6/6 (20/20)	6/6 (20/20)	6/6 (20/20)
5	6/6 (20/20)	6/30 (20/100)	6/6 (20/20)	6/15 (20/50)
6	HM	HM	LP	LP
7	HM	6/7.5 (20/25)	6/12 (20/40)	6/7.5 (20/25)
8	6/15 (20/50)	6/60 (20/200)	6/90 (20/300)	6/21 (20/70)
9	6/60 (20/200)	6/21 (20/70)	6/21 (20/70)	6/21 (20/70)
10	6/24 (20/80)	LP	LP	Enucleated
11	6/15 (20/50)	6/60 (20/200)	6/7.5 (20/25)	6/7.5 (20/25)
12	6/7.5 (20/25)	HM	6/6 (20/20)	HM/CF
13	6/9 (20/30)	HM	6/9 (20/30)	6/60 (20/200)
14	1.5/60 (5/200)	6/12 (20/40)	6/60 (20/200)	6/9 (20/30)
15	6/24 (20/80)	6/30 (20/100)	6/9 (20/30)	6/12 (20/40)
16	6/9 (20/30)	6/7.5 (20/25)	6/12 (20/40)	6/7.5 (20/25)
17†	LP	LP	6/90 (20/300)	NLP
18	0.3/10 (1/200)	0.9/60 (3/200)	6/7.5 (20/25)	6/7.5 (20/25)
19	6/12 (20/40)	HM	6/6 (20/20)	6/6 (20/20)
20	6/6 (20/20)	6/21 (20/70)	6/6 (20/20)	6/90 (20/300)

*CF indicates counting fingers; HM, hand motions; LP, light perception; and NLP, no light perception.
†This patient had peripheral anterior synechiae and secondary glaucoma.

uveitis. American Indian ancestry appears to be the link to Oriental racial type. Five patients had a forme fruste retinal detachment. After extensive treatment with corticosteroids, detachments flattened within an average of two to three months. Anterior inflammation continued to persist chronically. Seven of our patients had retinal vasculitis. Pregnancy had a beneficial effect on disease activity. Those patients with diabetes mellitus and sickle cell hemoglobin did not have an adverse effect. Visual outcome was good; of 20 patients, 16 had visual ascuity of 6/21 (20/70) in at least one eye.

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FALCIFORM FOLD, RETINAL DETACHMENT, AND NORRIE'S DISEASE

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Norrie's disease (congenital progressive oculo-acoustic-cerebral dysplasia) is a rare, genetically determined syndrome characterized by bilateral congenital blindness associated with varying degrees of hearing impairment and mental retardation. In 1927 Norrie[1] described several cases of a "hereditary atrophia oculi congenita" disorder. Warburg[2,3] has studied and written extensively on the original Danish families noted by Norrie and has established this condition as a specific X-chromosome-linked recessive syndrome. Despite a noncontributory family history of blindness, other diagnoses frequently considered are retinoblastoma, retinal dysplasia, retrolental fibroplasia, and hyperplastic primary vitreous. The disorder is rare and only a few family cases have been reported in the United States.[4-9]

The following case report shows the previously undocumented clinical course progressing from early vitreous hemorrhage and falciform retinal fold to the typical retrolental detached retinal mass and late phthisis bulbi.

CASE REPORTS

Case 1—A 6-week-old boy, the product of a normal term pregnancy, weighed 3,635.2 g at birth. The parents and pediatrician noticed poor vision and a white pupil in the left eye and the patient was referred for examination. The family history showed an X-chromosome-linked congenital blindness occurring in several affected males (Fig. 1).

The patient was examined in the office and under general anesthesia at 8 weeks of age. External ocular examination showed clear corneas measuring 10 × 10 mm (Fig. 2). Schiøtz tonometry was recorded at R.E.: 14.6 mm and L.E.: 12.2 mm. Gonioscopy showed the angle to be open in both eyes. The iris appeared normal and the lens was clear in the right eye. There was iris neovascularization and several iris adhesions to the lens surface in the left eye. Behind the clear lens of the left eye was a vascularized membrane or retinal detachment. Indirect ophthalmoscopy of the right eye showed a dense vitreous hemorrhage and a large nasal falciform fold extending from the disk to the peripheral retina (Fig. 3). No retinal breaks were detected with scleral depression. The fundus was not visible in the left eye. B-scan ultrasonography confirmed the presence of a large retinal falciform fold adjacent to the optic nerve of the right eye and a retrolental membrane or a retinal detachment, or both, in the left eye (Fig. 4).

By age 14 weeks the vitreous hemorrhage cleared and the patient had pupillary light reflexes but poor subjective visual acuity in the right eye. At age 8 months no definite pupillary light reactions could be shown in the right eye. A shallow anterior chamber with iris-lens adhesions and retrolental membrane, believed to be a possible retinal detachment, had developed which was similar to the initial appearance for the left eye (Fig. 5). More synechiae and lens opacification were present in the left eye (Fig. 6).

The patient was readmitted for examination and vitreous and retinal surgery. The clear lens was removed by ultrasonic phacofragmentation by using a pars plana approach to allow access to the folded thickened retrolental membrane or retina. The vascularized membrane was perforated showing choroid through the aperture. The retinal nature of the membrane was then clinically confirmed. Histologic examination of the biopsy tissue showed normal retinal and connective tissue. It was impossible to reattach the shrunken retinal tissue. The operated eye tolerated the surgical procedure well and has not become phthisical during two years of follow-up. The unoperated left eye subsequently became phthisical.

Case 2—A 24-year-old man (uncle of Case 1) was seen because of a history of blindness since birth in both eyes. Ocular examination in this alert man showed visual acuity of light perception. External ocular examination showed phthisis bulbi with band keratopathy. Corneal diameters measured 9 × 10 mm. The anterior chamber appeared flat with some bleeding noted in the left eye. B-scan ultrasonography showed a shrunken globe with retinal detachment in each eye.

Case 3—This man (uncle of Case 1 and brother of Case 2) was followed up elsewhere for severe congenital blindness and mental retardation. Previous diagnoses included bilateral pseudoglioma, and bilateral posterior persistent hyperplastic vitreous. Physical

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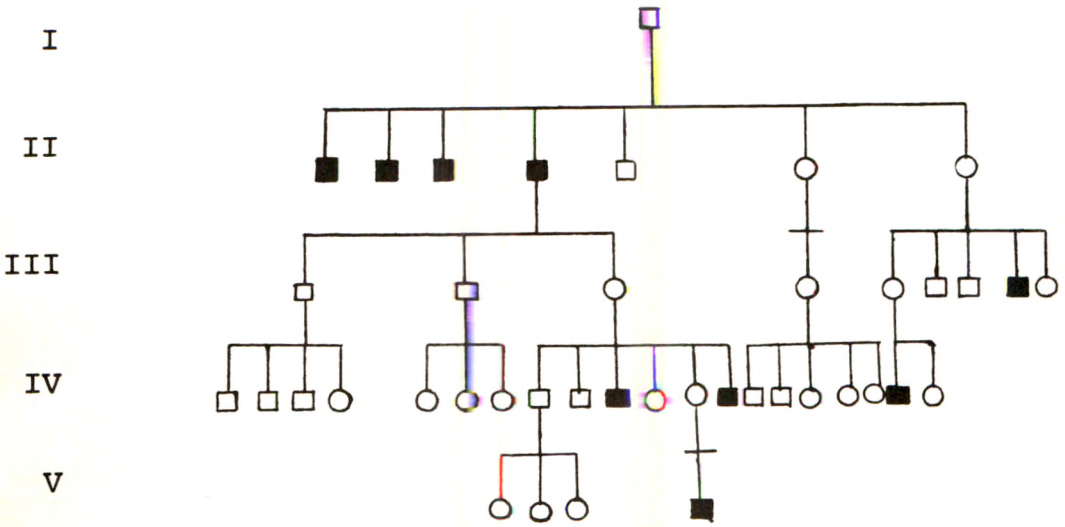


Fig. 1 (Jacklin). Family pedigree of X-chromosome-linked Norrie's disease.

examination showed a severely retarded man with bilateral blindness, endophthalmus, phthisis bulbi, and leukocoria. Ear examination showed thickened tympanic membranes and probable deafness.

Discussion

Congenital retinal detachment and congenital falciform fold occur rarely.[10] They may develop as a sequel to fetal chorioretinitis, retinopathy of prematurity, or as a hereditary disorder usually associated with other ocular or systemic congenital abnormalities, or both.[11-14] In some families, the patients show either falciform folds or retinal detachment, but these features may also occur in the same family. Both autosomal dominant and

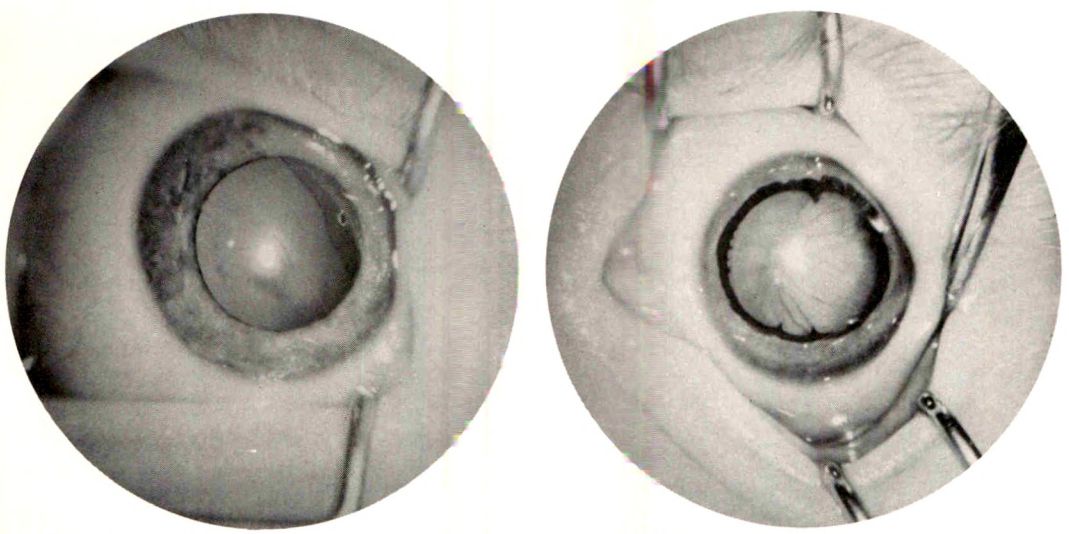


Fig. 2 (Jacklin). Case 1. Extraocular examination at age 6 weeks. Right eye, Nasal retinal falciform fold. Left eye, Vascularized retrolental membrane or retinal detachment.

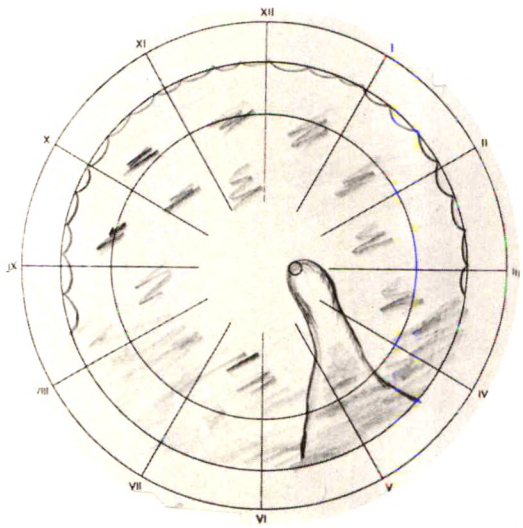


Fig. 3 (Jacklin). Case 1. Retinal drawing of nasal falciform fold of right eye.

autosomal recessive traits have been described. Pruett and Schepens[15] have found nonhereditary congenital falciform folds to result from a posterior form of persistent hyperplastic primary vitreous.[15] In some of their cases minimal congenital vitreoretinal adhesions caused later rhegmatogenous retinal detachment. Congenital nonattachment of the retina and X-chromosome-linked inheritance can occur in the Bloch-Sulzberger type of incontinentia pigmenti and Norrie's syndrome.

The pathogenesis of Norrie's disease is unclear. Warburg[11] believed a genetically determined biochemical defect induces an early arrest in retinal development with associated malformation of the primary and secondary vitreous. Neuroectodermal changes also occur in the cerebral cortex and auditory nerve. The rapid intrauterine and neonatal ocular deterioration has allowed pathologic analysis of a few late specimens consisting of massive vitreous hemorrhage, intraocular osseous formation, and phthisis bulbi. Blodi and Hunter[5] have suggested a link between Norrie's and Coats' disease because of clinical and histopathologic similarities between the two diseases.[5] Apple, Fishman, and Goldberg[9] reported histologic findings in a 3-month-old child. The excised retrolental tissue showed foci of retinal dysplasia, preretinal gliosis, retinal detachment, and organized vitreous hemorrhage and associated persistence of the primary vitreous.

Case 1 shows the previously undocumented early clinical pathogenesis of Norrie's disease from a falciform fold and

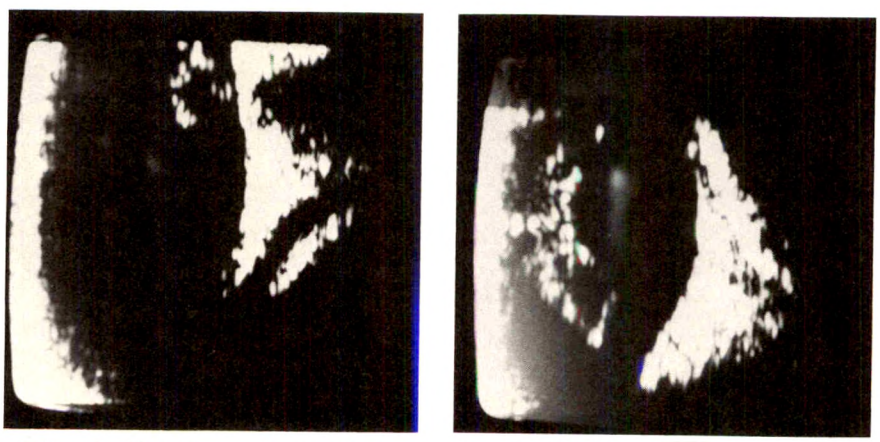


Fig. 4 (Jacklin). Case 1. B-scan ultrasonography. Left, Right eye, falciform fold. Right, Left eye, retrolental tissue.

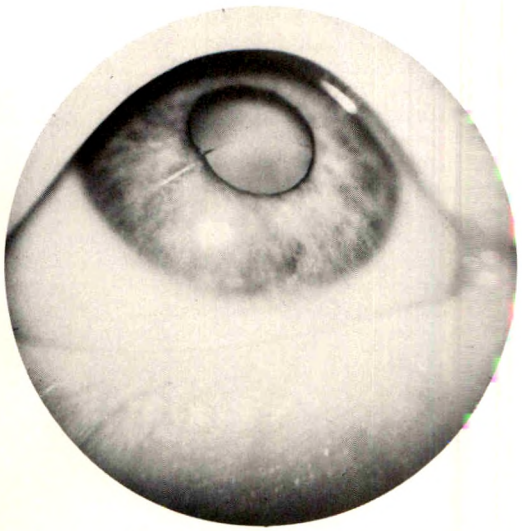


Fig. 5 (Jacklin). Case 1. Extraocular examination at age 8 months shows retrolental membrane or retinal detachment and iris adhesions.

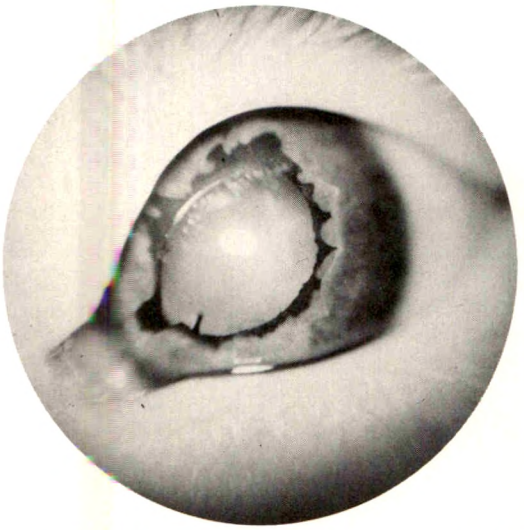


Fig. 6 (Jacklin). Case 1. Left eye, extraocular examination at age 8 months.

recurrent vitreous hemorrhage to the characteristic retrolental membrane and retinal detachment. Surgery in Norrie's disease has previously consisted of extracapsular cataract extraction or late enucleation for old painful blind phthisical eyes. The lens and vitreoretinal surgery performed in Case 1 has thus far prevented phthisis bulbi. Lens removal prevented peripheral and iris lens synechiae and shallowing of the anterior chamber with normal drainage of aqueous into the outflow system. Retinal reattachment was, unfortunately, not possible. Early lensectomy, vitrectomy, and retinal repair should be considered before total retinal detachment and contracture occur.

Summary

A patient with X-chromosome-linked recessive Norrie's disease, characterized by retinal falciform fold, retinal detachment, repeated vitreous hemorrhage, and retrolental membrane formation, was observed from age 6 weeks. The patient showed the clinical progression from these early retinal and vitreous changes to the more typical retrolental vascularized detached retina. Pathologic examination showed normal retinal tissue. Two other male relatives showed the end stages of Norrie's disease with bilateral congenital blindness, with and without mental retardation and deafness. Vitreoretinal surgery prevented phthisis bulbi from occurring in the operated eye but was unsuccessful in reattaching the contracted retina.

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OPHTHALMIC MINIATURE

Witch Two laughs at us. Witch Three scowls at us. Witch sisterhood all stitch, stitch. First Witch has a red circle round each eye. I fancy it like the beginning of the development of a perverted diabolical halo, and that, when it spreads all round her head, she will die in the odor of devilry.

F. G. de Fontaine
The Fireside Dickens
New York, Dillingham, 1888

SURGICAL APPROACHES TO SUBRETINAL STRANDS

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Proliferation of retinal glia or of pigment epithelial cells may result in the formation of retroretinal membranes in cases of retinal detachment.[1,2] This occurs with long-standing nonrhegmatogenous or rhegmatogenous detachments. Subretinal tissue can be so strong that it prevents the retina from reattaching, causing either a clothesline appearance with a subretinal band under the crest of the fold or unexplained folds in detached retina caused by contracted membranes. Detached retinas that are prevented from reattaching by subretinal bands and membranes now can be managed surgically. I successfully removed subretinal strands or membranes surgically in three patients with retinal detachments.

MATERIAL AND METHODS

The purpose of the operation is not to remove the subretinal tissue, but rather to release the traction. Scissors used for cutting in the subretinal space should be as small as possible. They can be straight or angled. The tips of the blades have to be blunt to avoid perforation of the retina and to allow maneuvering within the subretinal space without destroying tissue. The blades should be short to avoid wide gaping of the blades of the limited subretinal space. I use Neubauer vitreous scissors.[3]

The subretinal space can be approached either externally or internally. Initially, the external approach seems to be the most attractive because it is similar to familiar subretinal fluid drainage techniques. Instead of a tiny puncture of the choroid, a cut under an area of high retinal detachment has to be made. A large incision is made in the sclera, and the bulging choroid is carefully diathermized to avoid hemorrhages before the choroid is cut with the same scissors that are to be used in the subretinal space. Quick insertion of the scissors is necessary to avoid loss of subretinal fluid that would prevent manipulation of the scissors in the subretinal space. The exact position of the scissors under the opaque retina is hard to judge. Good intraocular illumination with fiber optics is mandatory in order to follow all manipulations once the scissors are introduced (Fig. 1).

The cutting of the actual strands or membranes must be done carefully to

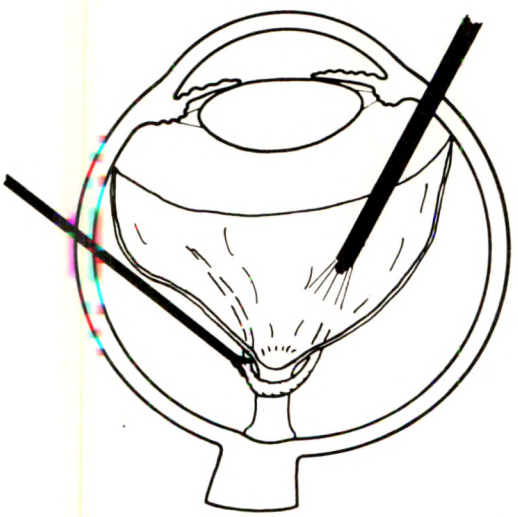


Fig. 1 (Machemer). Schematic drawing of external approach to subretinal strand. Vitreous scissors are introduced into the subretinal space through the sclera.

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avoid damaging the retina. The aim is to get the strand between the two blades of the scissors. Many cuts are often necessary.

Once the subretinal strand is severed the scissors are retracted. Tightening preplaced sutures over the scissors avoids prolapse of the retina through the large opening. Any procedure needed to reattach the retina is added.

Several difficulties may be encountered. The first is loss of subretinal fluid before the scissors are fully introduced or even after introduction of the scissors. Manipulations are easier the more the subretinal space is expanded. The subretinal space may have to be filled with an infusion needle before scissors are re-inserted, or scissors equipped with an infusion tube may be used. Manipulations of the eyeball to reach the subretinal tissue are difficult to perform with an instrument that is inserted into the eye posteriorly; especially when this has to be done on the nasal side. Hemorrhages from the choroid are a great danger, especially with the tangential tilt of the scissors and with the vicinity to large vortex veins.

I prefer an internal approach because it allows for a more controlled procedure, and manipulations of the eye during the cutting maneuvers are much easier than with the external approach. Additionally, subretinal fluid cannot be lost. The procedure consists of cutting a hole into peripheral retina near the quadrant where the subretinal strand will be found, usually after a vitrectomy has been performed. The scissors are inserted through the pars plana in the selected quadrant and guided into the vitreous cavity. They are then used to perform a cut into the retina parallel to the ora serrata as anterior as possible. To facilitate this peripheral cut of the retina, the sclera is indented posterior to the insertion of the scissors by the assistant. The retinal cut can also be performed with the virectomy instrument introduced into the eye on the side opposite to the planned cut. The retinal cut usually takes one to two hours. Hemorrhages are usually not found because the periphery of the retina is not vascularized. The scissors are then guided into the subretinal space (Fig. 2). To avoid uncontrolled tearing of the retina the cut should be enlarged if the hole for the insertion of the scissors seems to be too small. To increase the space for maneuvering of the scissors, the height of the retinal detachment can be increased by aspiration of fluid from the vitreous cavity and infusion of fluid into the subretinal space through the peripheral cut with the vitreous cutter or an infusion needle.

Once the retina is mobilized, the intraocular instruments are removed and the sclerotomy sites are closed. Cryotreatment of the area of the retinal cut and appropriate buckling procedures fol-

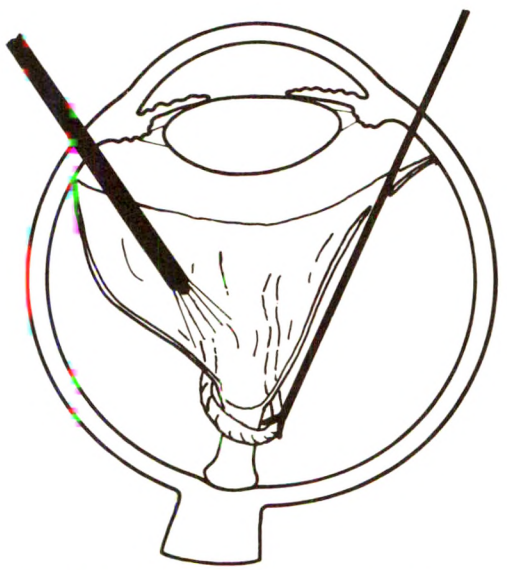


Fig. 2 (Machemer). Schematic drawing of internal approach to subretinal strand. Vitreous scissors are introduced into the vitreous cavity, and are then guided into the subretinal space through a freshly cut peripheral retinal hole.

low. Intravitreal injection of air helps to unfold the retina.

The difficulty encountered with this approach is achieving a good peripheral retinal cut. Indentation should not be too forceful in order to keep the retina from becoming too close to the pigment epithelium. Wide dilation of the pupil is necessary. In phakic eyes the lens may be damaged during the attempt to remove the vitreous base from the planned site of the peripheral retinal cutting. A lentectomy of the preoperatively clear lens may have to be done. If more posterior transretinal cutting is planned (S. Charles, personal communication, 1979), the creation of a posterior retinal hole should be considered. If difficulties are anticipated in closing this hole, this approach should be avoided.

CASE REPORTS

Case 1—An 18-year-old woman had atopic dermatitis for many years. A cataract extraction had been performed four years previously. Visual acuity was 6/7.5 (20/25). One month before examination, a retinal detachment developed in her left eye.

When examined, visual acuity in the left eye was counting fingers at one foot with an intraocular pressure of 4 mm Hg. The anterior chamber had a 2+ flare. Lens remnants were noted behind the pupil. The retina was totally detached with multiple star folds (Fig. 3, left).[4]

A vitrectomy was performed. The vitreous was heavily pigmented. Preretinal membranes could be removed with the help of a hooked needle from a star fold over the macula and from other folds superiorly and inferiorly. It was still not possible to mobilize the retina because of a subretinal strand coming from the superior temporal quadrant, passing around the nasal side near the disk and extending toward the temporal inferior quadrant. This membrane kept the retinal funnel closed like a purse string. I used an external approach to cut the subretinal band on the nasal side with vitreous scissors. The moment the band was severed the ends snapped back, showing a big gap between them. A 5-mm Silastic sponge was placed around the eye after applying a triple row of cryotherapy around the eye. The sponge also covered an inferior peripheral hole. Three milligrams of dexamethasone-alcohol was injected intravitreally together with some air. No subretinal fluid was drained.

Postoperatively the retina settled quickly. Seven months after the operation the retina was well attached with a visual acuity of 6/18 (20/60). A dry retinal fold extended from the disk to the periphery temporally and inferiorly with possible remnants of

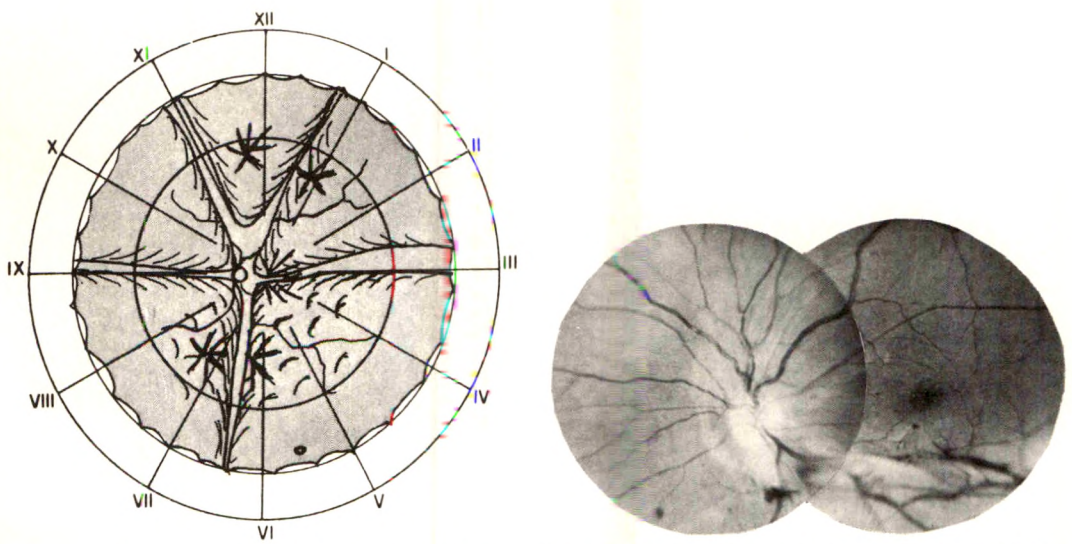


Fig. 3 (Machemer). Case 1. Left, Preoperative fundus drawing. Highly detached retina with fixed folds. Right, Postoperative fundus photograph after vitrectomy and cutting of the subretinal strands. Surrounding the nasal side of the disk and extending temporally inferiorly, remnants of the subretinal strand remain visible underneath the attached retina.

the subretinal strand underneath because of the irregular pigmentation (Fig. 3, right).

Case 2—A 63-year-old man had severe psoriasis and arthritis. Two years previously he had a cataract extraction in his left eye. Visual acuity improved to 6/24 (20/80). Two months later visual acuity decreased to 6/120 (20/400) because of a vitritis. No retinal lesions could be seen. He was treated with periocular corticosteroid injections; however, there was no visual improvement. Two weeks preceding our examination there was further loss of visual acuity in this eye and a retinal detachment was detected.

When the patient was examined his visual acuity was hand movements at two feet. The eye was soft. The aphakic eye had a cellular, hazy vitreous. The retina was shallowly detached except for the nasal inferior quadrant. A retinal fold could be seen extending from the disk toward the temporal periphery at the 2 o'clock position, passing through the macular area. There seemed to be a brown discoloration under the retina in this area (Fig. 4, left).

A vitrectomy was performed. Once the opaque vitreous was removed, a shallow detachment was confirmed. Because of the peculiar sharp fold there was possibly a subretinal membrane under the fold. The internal approach for membrane cutting was applied. Scissors were introduced into the vitreous cavity at the 2 o'clock position in the periphery, and a hole was cut into the fold near the ora serrata. A heavily pigmented membrane was found. This membrane was cut along the fold from the periphery across the macular area to the optic nerve head. The fold opened immediately after this procedure. A 2½-mm wide silicone band was placed around the eye after appropriate cryotherapy of the peripheral retinal tear. No drainage of subretinal fluid was done.

Postoperatively the retina reattached quickly. Seven months after the operation, visual acuity had recovered to 6/120 (20/400). The retina was well attached everywhere. Except for pigmentary irregularities in the temporal superior quadrant no remnants of the membrane could be seen (Fig. 4, right).

RESULTS

I have successfully removed subretinal strands or membranes in three cases. Two were cases of severe massive periretinal proliferation in which after vitrectomy and preretinal membrane peeling the retina could not be mobilized because of a subretinal strand that narrowed the funnel of detached retina. In both cases the technical problems in cutting the strands were solved without complications; one with the external, the other with the internal approach. In Case 1 (Fig. 3), the retina was successfully reattached and visual acuity improved from counting fingers at one foot to 6/18 (20/60). In the second case recurrence of massive periretinal proliferation caused

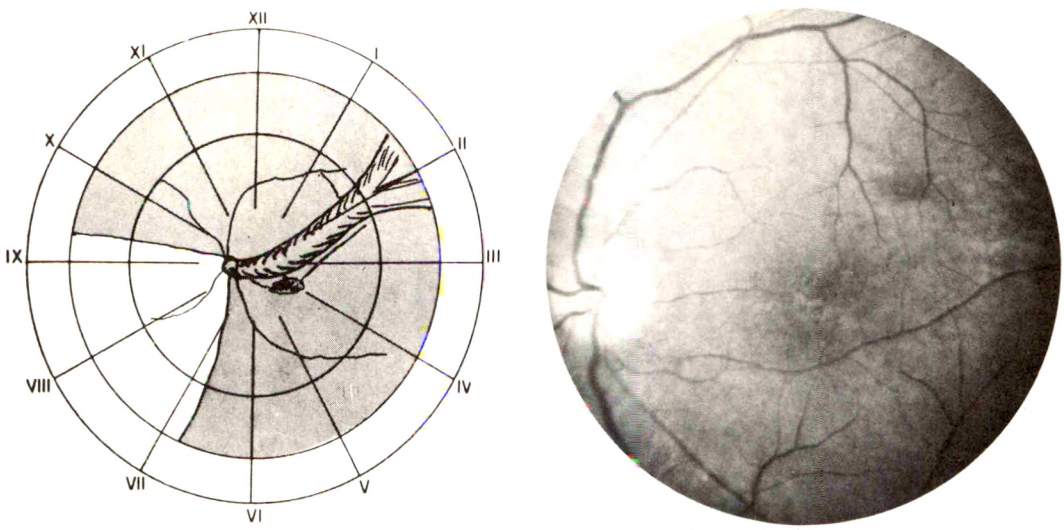


Fig. 4 (Machemer). Case 2. Left, Preoperative fundus drawing. Note the retinal folds passing through the macular area and the shallow traction detachment. Pigmentation is seen inferior to the macular area indicating subretinal proliferation. Right, Postoperative fundus photograph after virectomy and cutting of subretinal membrane. The retina is attached, and the fold has flattened out.

the initially attached retina to detach again. The third case (Case 2, Fig. 4) was a nonrhegmatogenous detachment caused by a subretinal membrane. The patient suffered from chronic uveitis with vitreous opacities combined with severe psoriasis and arthritis. The retina reattached and visual acuity changed from hand movements at two feet to 6/120 (20/400).

Discussion

It is possible to perform surgery within the subretinal space. One may argue that the cutting of the subretinal strands in the two cases of massive periretinal proliferation might not have been necessary despite immediate mobilization of the retina. Stretching of the strand and reattaching of the membranes could just as well have occurred spontaneously after closure of the appropriate retinal hole with buckling procedures, but judgment of the strength of subretinal strands in rhegmatogenous retinal detachment remains subjective. In Case 2, however, the detachment was clearly caused by the subretinal membrane. Because the fold crossed the macular area, any external procedure, if successful, would have resulted in a dry fold crossing the macular area.

Remnants of the subretinal strands were difficult to detect once the retina was reattached. In Case 1 subretinal pigmentation and a slight solid elevation was the only remnant of subretinal tissue. In Case 2, it was impossible to identify remnants of subretinal tissue.

Summary

I successfully removed subretinal strands or membranes surgically in three patients with retinal detachments. Two approaches were used: (1) by cutting through the sclera with vitreous scissors (external approach), or (2) by cutting holes through the retina (internal approach). In two cases of retinal detachment, massive periretinal proliferation occurred, one of which was successfully managed with the external approach. In the other case, recurrence of massive periretinal proliferation caused the retina to detach again. In a third case a nonrhegmatogenous retinal detachment caused by a subretinal membrane was managed successfully with the internal approach.

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SENILE LENS AND SENILE MACULAR CHANGES IN A POPULATION-BASED SAMPLE

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Senile lens and senile macular changes are two commonly encountered ocular findings in older populations. Though both contribute significantly to the morbidity of such populations, little is known of the cause of either.

The Framingham Eye Study[1] is unique in providing data on ophthalmic disease in a population-based sample. In their analysis of the prevalence of cataract and senile macular degeneration in the Framingham Eye Study population, Kahn and associates[2] required that the senile changes be accompanied by a reduction in visual acuity. We report on the presence of senile lens and senile macular changes without consideration of visual acuity. We have used the Framingham Eye Study data to describe some of the epidemiologic characteristics of these senile lens and macular changes to aid in developing hypotheses relating to their cause. The following three aspects will be included: (1) prevalence of the two conditions by age and sex; (2) the association between the two conditions, and (3) the extent to which each of them occurs in neither, one, or both eyes.

SUBJECTS AND METHODS

The data for the Framingham Eye Study were derived from eye examinations performed from 1973 to 1975 on the Framingham Heart Study Cohort.[3] This cohort has been examined biennially, beginning in 1948, for the purpose of identifying factors that affect the risk of cardiovascular diseases. In 1973, 4,045 survivors of the original cohort were listed on the Framingham Heart Study roster, ranging in age from 52 to 85 years. Excluding those who had died before contact (68), 2,940 locally residing subjects and 1,037 nonlocally residing subjects were available (Table 1). A total of 2,477 (84%) of local and 198 (19%) of nonlocal subjects were given a screening examination for eye disorders.

The screening examination was conducted by a second- or third-year resident, according to a written protocol. Pupils were dilated with one drop of 10% phenylephrine HCl and one drop of 1% tropicamide. Additional drops were used if necessary until two thirds of the diameter of the anterior surface of the lens was exposed. A slit lamp was used to identify lens vacuoles, waterclefts, spokes, and lamellar separations, which were identified in the Framingham Eye Study as early senile lens changes. Cortical cuneiform opacities, decreased lucency of the lens nucleus, posterior subcapsular opacities, and miscellaneous other senile lens changes were designated as late changes (Table 2).

Each subject's posterior pole was examined with a direct ophthalmoscope for the presence of pigmentary disturbances, drusen, elevation of the pigment or neurosensory epithelium, or perimacular circinate exudates. In the present study, macular changes are classified into a mild or a moderate to severe category (Table 2). Eyes were classified as having mild changes if any of the

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TABLE 1

FRAMINGHAM EYE STUDY POPULATION

Area	Age (yrs)	No. Eligible	With Screening Examination		Examined After Protocol Change	
			No.	%	No.	%
Local	52-64	1,465	1,293	88	1,139	78
	65-74	941	787	84	652	69
	75-85	534	397	74	318	60
	Total	2,940	2,477	84	2,109	72
Nonlocal	52-64	497	89	18	87	18
	65-74	354	75	21	71	20
	75-85	186	34	18	33	18
	Total	1,037	198	19	191	18

following were observed: (1) subtle macular pigmentary disturbance (though definitely present), or (2) less than ten small macular drusen. Eyes were classified moderate to severe if any of the following were observed: (1) obvious or severe macular pigmentary disturbance; (2) more than ten small macular drusen; (3) large macular drusen; (4) serous, hemorrhagic, or proliferative elevation of the macular pigment or neurosensory epithelium; or (5) perimacular circinate exudates.

TABLE 2

DEFINITION OF SENILE CHANGES

LENS CHANGES

1. Early senile lens changes
   a. Vacuoles
   b. Waterclefts
   c. Spokes
   d. Lamellar separations
2. Late senile lens changes
   a. Cortical cuneiform opacities
   b. Nuclear sclerosis
   c. Posterior subcapsular opacities
   d. Miscellaneous late senile changes

MACULAR CHANGES

1. Senile macular changes—mild
   a. Subtle macular pigmentary disturbance
   b. Small macular drusen (< 10)
2. Senile macular changes—moderate to severe
   a. Obvious or severe macular pigmentary disturbance
   b. Small macular drusen (> 10)
   c. Large macular drusen
   d. Serous, hemorrhagic, or proliferative elevation of the pigment or neurosensory epithelium
   e. Perimacular circinate exudates

A second part of the Framingham Eye Study involved the referral of selected subjects for a definitive examination. All aphakic subjects were referred for this second examination at which time the basis for the aphakia was diagnosed. Those judged to have a history of senile cataract are classified in this report as subjects with late lens changes. Such information on the history of the aphakia is the only portion of the definitive examination used in this report.

Persons with early lens or mild macular changes in either or both eyes, but no more severe changes, were classified in the respective less severe category. Those with more severe changes in either eye, regardless of whether milder changes were present, were classified in the respective more severe category.

Population prevalence estimates of senile lens changes were based on all local subjects examined (2,477) (Table 1). The initial protocol of the Framingham Eye Study, which applied to the first 368 local subjects, did not provide sufficient information on location or severity of posterior pole disease, and macular data about these early subjects were therefore

TABLE 3

PREVALENCE OF SENILE LENS CHANGES BY AGE AND SEX

Age (yrs)	Sex	No. Examined	No. With Adequate Examination	Early Changes No.	%	Late Changes No.	%	Early or Late Changes No.	%
52-64	M	573	548	112	20	96	18	208	38
	F	720	681	140	21	165	24	305	45
	Total	1,293	1,229	252	21	261	21	513	42
65-74	M	318	298	61	20	142	48	203	68
	F	469	434	84	19	249	57	333	77
	Total	787	732	145	20	391	53	536	73
75-85	M	152	136	16	12	104	76	120	88
	F	245	214	22	10	177	83	199	93
	Total	397	350	38	11	281	80	319	91

excluded from analysis. Analysis for associations between the senile macular and senile lens changes used data from both local and nonlocal subjects examined after the protocol revision (2,300).

RESULTS

Table 3 shows the prevalence of senile lens changes, by age and sex, in the local Framingham study population. The percentage of persons with lens changes increased from 42% (513) at age 52 to 64 years, to 91% (319) at age 75 to 85 years. Both sexes showed a steady increase of changes with age, but women had higher rates in every age group. In the oldest age group (75 to 85 years) where the prevalence of lens changes was 91% (319), only 11% (38) had early changes alone.

Table 4 presents the prevalence of senile macular changes, by age and sex. The percentage of persons with such changes increased from 35% (374) in the

TABLE 4

PREVALENCE OF SENILE MACULAR CHANGES BY AGE AND SEX

Age (yrs)	Sex	No. Examined	No. With Adequate Examination	Mild Changes No.	%	Moderate-Severe Changes No.	%	Any Changes No.	%
52-64	M	516	482	92	19	96	20	188	39
	F	623	576	92	16	94	16	186	32
	Total	1,139	1,058	184	17	190	18	374	35
65-74	M	267	242	44	18	68	28	112	46
	F	385	348	83	24	84	24	167	48
	Total	652	590	127	22	152	26	279	47
75-85	M	121	100	13	13	36	36	49	49
	F	197	163	24	15	58	36	82	50
	Total	318	263	37	14	94	36	131	50

TABLE 5

FREQUENCY DISTRIBUTION OF PERSONS BY NUMBER OF AFFECTED EYES BY AGE, FOR EACH TYPE AND SEVERITY OF CONDITION

Condition	Age (yrs)	Eyes Affected: Right Only	Left Only	Both	Proportion Bilateral	Neither
Any lens change	52-64	63	100	350	0.68	716
	65-74	36	42	456	0.85	196
	75-85	7	9	294	0.95	31
	Total	106	151	1,100	0.81	943
Late lens change	52-64	24	40	197	0.75	968
	65-74	22	34	333	0.86	341
	75-85	9	6	257	0.94	69
	Total	55	80	787	0.85	1,378
Any macular change	52-64	59	72	238	0.64	684
	65-74	44	35	194	0.71	311
	75-85	14	12	96	0.79	132
	Total	117	119	528	0.69	1,127
Moderate to severe macular change	52-64	34	46	105	0.57	868
	65-74	17	17	112	0.77	438
	75-85	19	9	57	0.67	169
	Total	70	72	274	0.66	1,475

52 to 64 year group, to 50% (131) in the 75 to 85 year age group. This increase with age was clearly less marked than was that seen for lens changes. With the exception of a lower rate for women in the youngest age group ($P<.05$), no differences were seen in the rates of men and women.

Table 5 shows the frequencies of persons with both eyes affected, for the three age groups, separately for lens and macular changes. When present, each condition usually occurs bilaterally. In persons with moderate to severe macular changes, for example, most have bilateral involvement (274/416). This association is statistically significant, and holds for both diseases and at all ages. The process causing these changes tends to affect both eyes, or neither. The proportion bilaterally affected follows the same pattern as the prevalence. It increases with age and is higher for lens changes than macular changes. Though right and left eyes seem equally susceptible to macular changes, unilateral lens changes are seen 1.5 times as often in the left eye as in the right eye.

Table 6 shows that senile macular and lens changes develop independently. The odds of having lens changes in eyes in which macular changes are present are approximately the same as the odds of having lens changes in eyes in which macular changes are not present. This is shown by ratios of such odds that cluster around 1.00.[4] This is true moreover for men and women, and for both levels of severity evaluated.

DISCUSSION

Comparison with other reports on the prevalence of senile lens and senile macular changes is difficult because of

TABLE 6

ASSOCIATION BETWEEN SENILE MACULAR CHANGES AND SENILE LENS CHANGES, BY SEX AND EYE

Types of Senile Changes	Eye	Average* Odds Ratio	
		Male	Female
1. Any lens change vs any macular change	R.E.	0.86	1.3
	L.E.	0.72	1.3
2. Late lens changes vs moderate to severe macular changes	R.E.	0.68	.99
	L.E.	0.63†	.77

* Odds ratios are "averaged" for the three age groups by method of Mantel and Haenzel.[4]

† Odds ratio significantly different from 1 ($P < .05$).

differences in disease definitions. Unlike many other studies, we did not include any decreased visual acuity criterion. If one does include such a criterion, then some patients with mild lens or macular changes but with visual loss from another cause may be included in the analyses, whereas others with similar or more marked lens or macular changes but without vision changes may be omitted. Moveover, the investigation of associations between conditions such as lens changes and macular changes is harder to interpret when a common criterion such as visual acuity is used.

Imposing a visual acuity restriction of 6/9 (20/30) or worse and excluding those persons with early changes alone, Kahn and associates[2] reported rates for cataracts that increased from 5% at age 52 to 64 years, to 46% at ages 75 to 85 years. In a random sample of older people in Edinburgh, Milne and Williamson[5] found a cataract prevalence of 22% in the 62- to 79-year age group. However, their subjects eyes were neither dilated nor examined with a slit lamp. Similarly, Chatterjee's Punjab study[6] of dry belt Indians showed a cataract prevalence of 22% in a 50- to 59-year age group and 31% in a 60-year and older age group but examinations were conducted without a slit-lamp examination and relied on "diminished fundal glow and diminished visual acuity." Kornzweig, Feldstein, and Schneider's[7] Nursing Home survey showed a 52% cataract prevalence in eyes of subjects younger than age 80 and a 76% prevalence in eyes of subjects older than age 80 years when no visual acuity restriction was used. In a sample of the South Wales population McGuinness[8,9] found cortical opacities in 43% of men at age 40 to 49 years, 57% at age 50 to 59 years, 77% at ages 60 to 69 years, and 84% of those at ages 70 to 74 years. These figures are comparable to those reported here.

We found a higher frequency of lens changes among women. This difference was significant ($P < .02$) for the 52 to 64 and 65 to 74 year age groups. Kahn and associates[2] concluded the increased cataract (with visual acuity of ≤6/9 [20/30]) prevalence he noted in women was "within reasonable limits of chance fluctuation." Chatterjee[6] reported more cataracts in men than women but gave no further details.

Few reports of senile macular disease prevalence are available for comparison with our results. Imposing a visual acuity criterion of 6/9 (20/30), Kahn and associates[2] found a prevalence of senile macular degeneration of 2% at ages 52 to 64 years, 11% at ages 65 to 74 years, and 28% at ages 75 to 85 years. Higher rates for women were significant ($P < .01$). With no visual acuity criterion, our findings for senile macular changes for the three corresponding age groups were 35%, 47%, and 50%. Female prevalence lagged behind that for males for the youngest age group (32% vs 39%) ($P < .05$), but

male and female prevalence figures were comparable for the older age groups. Younger subjects tended to have more subtle pathologic findings so that the prevalence figures for the younger age group could be inflated by false-positives.

The comparison of our findings with those of Kahn and associates[2] suggests that men and women have senile macular changes with approximately equal frequency, but when accompanied by a visual acuity of 6/9 (20/30) or worse, these changes occur more often in women. It should be emphasized that Kahn and associates[2] reported prevalence of visual acuity of 6/9 (20/30) or worse accompanied by, but not necessarily caused by, senile macular degeneration. Possibly the excess prevalence in women reported by Kahn and associates[2] is explained by visual acuity reduction caused by cataract occurring more frequently in women.

The strong associations of senile changes in the two eyes of the same patient suggest changes that impinge on both eyes, as opposed to changes that originate locally. Though the clinician more often than not finds the same disease in the second eye as in the first, many subjects, especially younger ones, have unilateral pathologic findings of each condition.

Though senile macular and lens changes are both associated with aging, the data indicate that they develop independently. Eyes with or without one condition are equally likely to have the other. The question of whether associations between senile lens and macular changes exist among the various subgroups which make up each of these heterogeneous conditions remains unanswered. This question has important implications as we search for the cause of these changes.

Summary

Eye examinations performed on the survivors of the Framingham Heart Study Cohort showed a prevalence of senile lens changes ranging from 42% at ages 52 to 64 years to 91% at ages 75 to 85 years. The prevalence of senile macular changes increased from 35% at 52 to 64 years to 50% at 75 to 85 years. Persons affected with either condition usually had bilateral involvement. No association between the two conditions was shown.

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A CLEAR VIEW TREPHINE AND LAMELLAR DISSECTOR FOR CORNEAL GRAFTING

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In the past few years, many new trephines have been developed for corneal surgery (unpublished data).[1-8] Most corneal surgeons use the operating microscope, and therefore some of the new designs enable the surgeon to see the graft while cutting. Disposable trephines ensure a sharp edge for every graft. Most of the disposable trephines, however, fit onto handles of the cookie-cutter type, which greatly interferes with the surgeon's view during the actual cutting process.

Lieberman[4] described a corneal trephine that consists of two concentric rings with a single blade to cut the cornea. It can be varied to cut from 2.0- to 8.5-mm diameter buttons. Crock and associates[5] described a trephine that also has a rotating cutter and cuts the cornea radially under direct visualization. If corneal buttons stored in McCarey-Kaufmann medium are used as donor material, they would have to be mounted over an artificial chamber to obtain the same edge as that in the recipient's cornea.

Drews[1] described a cone-type trephine that allows the surgeon to view the central cornea while cutting. Donaldson[7] uses a similar design but increases the stability by adding a handle. Varying sizes of disposable trephines can also be used.

The above designs indicate that the following factors are desirable in a corneal trephine:

1. The surgeon should have a clear view of the whole cornea past the corneoscleral limbus while cutting.
2. The instrument should accept different sizes of disposable blades in order to decrease the cost of the instruments needed for different sizes of buttons.
3. The instrument should be comfortable to use and stable during cutting.
4. The trephine should be rotated by the nonfixating hand, preferably by means of a flexible shaft and in a direction perpendicular to the axis of the trephine. The orthogonal relationship between the axis of the drive and the center line of the trephine minimizes their interaction and thereby increases the stability of the trephine during cutting.
5. The instrument must be able to punch disks from the corneal buttons stored in McCarey-Kaufmann medium.

Figure 1 shows a trephine* with these features. The main body of the trephine consists of a ring that houses a small bevel gear. The flexible drive extends from this housing at right angles. There are two notches in this housing so that the trephine may be held either by the right or the left hand. The different sizes of disposable trephines come fitted to a clear plastic disk that can be snapped into

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*Available from the Storz Instrument Co., St. Louis, Missouri.

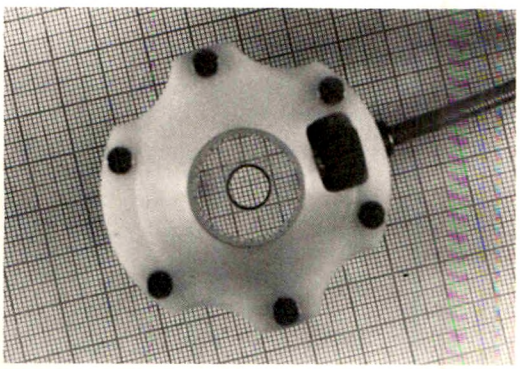


Fig. 1 (Smirmaul and Casey). Unobstructed view through the trephine during surgery. The grid is 10 × 10 mm.

the instrument. The surgeon holds the body of the trephine with one hand and rotates the cutter by means of the flexible shaft with the other. The gear ratio of 4:1 enables the blade to rotate 90 to 120 degrees. An oscillating or continuous rotating motion may be used depending on the preference of the surgeon. Because the hand that holds the housing of the trephine does not move, stability is assured and the direction of the axis can be altered at will during cutting. The trephine cuts along the axial line of the cornea, and corneal buttons stored in McCarey-Kaufmann medium can be used, which may be obtained by means of an endothelial side cutting punch.

A lamellar dissector—Two main techniques are used in lamellar grafting: (1) the open technique; and (2) a closed splitting technique.

In the open technique, the size of the graft is blocked out with a trephine and then, using either a scalpel, a hockey stick, a Dutch blade, or similar cutting instrument, the edge of the button is clasped with fine forceps and the disk is dissected under direct visualization. The cornea is reflected as the dissection progresses.

In the closed or splitting technique, a mark is made on the cornea with a trephine and a perpendicular cut is made to the level desired. The dissection plane is started with a cutting instrument and the cornea is split at that level by means of sweeping strokes with a lamellar dissector. When complete dissection has been achieved, the instrument is withdrawn and a trephine can be used to cut the disk.

The open technique permits direct visualization of the lamellae. The surface obtained, however, is not as smooth as with the splitting technique because the fibers may occasionally be cut. The same plane across the width of the cornea must be used. Another disadvantage of this method is that the recipient bed is open during the process and airborne debris can land on the surface and be trapped when the graft is placed on top. The closed technique is more difficult to perform and because the splitting of the lamellae is not done under direct visualization, perforations are possible. Another problem is that a fair amount of force is needed to split the lamellae with a blunt dissector and the eye has to be held firmly. The force generated by the edge of the lamellar dissector is balanced in only the one direction by the fixation of the eye by means of toothed forceps.

In order to balance this force and thereby reduce the amount of distortion caused to the eye, we designed an instrument* that uses two blades working in opposite directions. The oscillation nature of the blades eliminates any force on the cornea itself because one blade pushes against the other while separating the lamellae. When entering the cornea, after making a plane as in the closed technique, the blades overlap into the slit, and the cornea is split with an oscillating motion. Because the amplitude of the stroke can be varied to a maximum of 11 mm from blade edge to blade edge, the cornea can be split in one

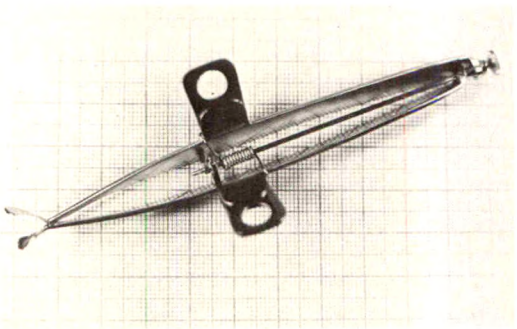


Fig. 2 (Smirmaul and Casey). Lamellar dissector with opposing variable stroke blades.

continuous motion by varying the amplitude. The edges of the blades are rounded and therefore no actual cutting occurs. This ensures that the blades stay within the same lamellar plane. The length of the blades is 12.0 mm and each is 2.5-mm wide (Fig. 2).

Summary

We designed a corneal trephine that permits a full view of the cornea while cutting. The drive mechanism of the trephine enables the surgeon to keep the central cutting axis of the instrument stable while the trephine is rotated. The instrument accepts variable sizes of disposable trephine blades, making it versatile and economical. We also devised a lamellar dissector with two blades to negate the splitting force that occurs during a closed lamellar dissection. It enables the surgeon to perform a closed lamellar dissection with minimal force applied to the globe and minimal risk of perforation.

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ELECTRON MICROSCOPIC STUDY OF RECURRENT REIS-BÜCKLERS' CORNEAL DYSTROPHY

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In 1917 Reis[1] described a dystrophy of the superficial corneal layers characterized by superficial corneal opacity and epithelial erosion. In 1947 Bücklers[2] further described the characteristics of this dystrophy and established the familial inheritance. Further clinical,[1-7] light microscopic,[8-14] and electron microscopic observations[15-21] have been reported. Clinical[6,7] and histologic[12] recurrences of this disease in corneal grafts have been described.

We describe a patient who remained asymptomatic for five years after having a superficial keratectomy, which has been studied electron microscopically.[19] We report the findings in a second keratectomy specimen obtained nine years after the first intervention, which showed curly dense collagen fibrils in the superficial layer similar to those described in the initial specimen. Also evident were profound degenerative changes in the stroma to which the abnormal epithelium was loosely attached. Additionally, a conjunctival biopsy showed ultramicroscopic alterations of the epithelial basement membrane.

CASE REPORT

A 41-year-old woman was first examined on Feb. 9, 1970, because of decreased visual acuity and "recurrent corneal ulcers." These recurrent corneal ulcers, which started in her childhood, occurred from three months to one year apart and healed without difficulty within three to four days. Family history showed four generations of corneal disease. Best corrected visual acuity was R.E.: 6/60 (20/200) and L.E.: 6/18 (20/60).

Results of ocular examination, with the exception of the cornea, were normal in both eyes. Slit-lamp examination of both corneas showed a 6- to 7-mm section of irregular epithelium with diffuse patchy opacities in the area of Bowman's membrane and thickening of Bowman's membrane with multiple nodules. An optical section showed that the opacities had a serrated appearance at the epithelium. The stroma, Descemet's membrane, and the endothelium appeared normal. Both eyes had similar findings. On Feb. 11, 1970, she had a superficial keratectomy in her right eye. The whole epithelium and opaque corneal tissue were removed until a clear stroma was present. Fourteen months later, visual acuity of this eye had improved to 6/9 (20/30) with correction. On Oct. 18, 1970, a superficial keratectomy was performed in her left eye. Eight months later, visual acuity was 6/6 (20/20) with correction.

Postoperatively, no symptoms developed in either eye. However, in February 1975, she noticed a gradual decrease in visual acuity in her right eye. On May 18, 1979, both corneas had begun to show a pattern similar to the preoperative condition. Both corneas were slightly thinner than normal (about 0.48 mm). The epithelial surface in both eyes was moderately irregular. Both corneas had a diffuse ground-glass haziness compromising the anterior one third of the stroma. The cornea showed an area of hypertrophic epithelium and areas of pigment deposits as a Stähli-Hudson's line (Fig. 1). The left eye had a similar appearance but the opacity was less pronounced. Corneal sensitivity was within normal limits. Visual acuity was R.E.: 6/30 (20/100) and L.E.: 6/15 (20/50) with correction.

In May 1979, the patient had a second keratectomy in her right eye. In contrast to the first operation, the whole epithelium came off easily. Then a circular 8-mm section of stroma (0.1 to 0.2 mm deep) was removed. After the operation, the patient wore a soft contact lens. One week later, her visual acuity was 6/15 (20/50) in the right eye. After this second operation the cornea was approximately half as thick as normal. A biopsy of bulbar conjunctiva was taken near the corneoscleral limbus at 2 o'clock.

MATERIAL AND METHODS

The specimens of epithelium, stroma, and conjunctiva were obtained from the

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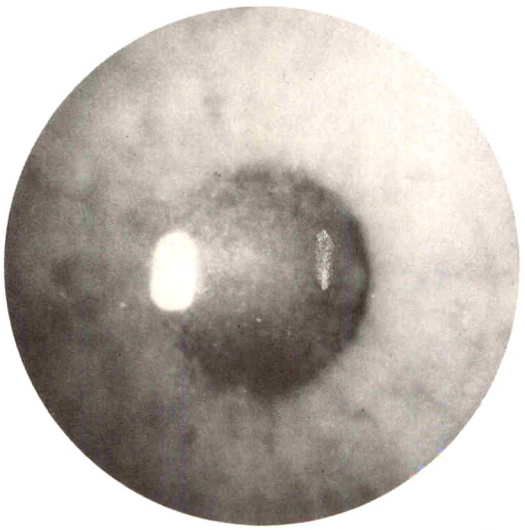


Fig. 1 (Yamaguchi, Polack, and Valenti). Clinical photograph of the right eye before re-operation. The anterior third of the cornea has a diffuse ground-glass appearance.

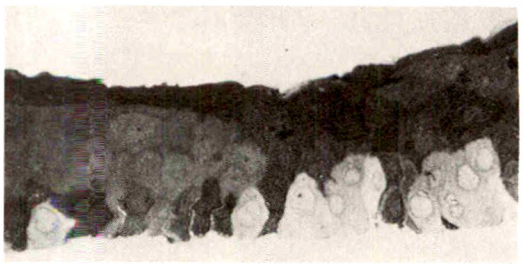


Fig. 2 (Yamaguchi, Polack, and Valenti). Histologic section of removed epithelium shows both dark and clear basal cells. Some of the clear cells are in mitosis (toluidine blue, ×440).

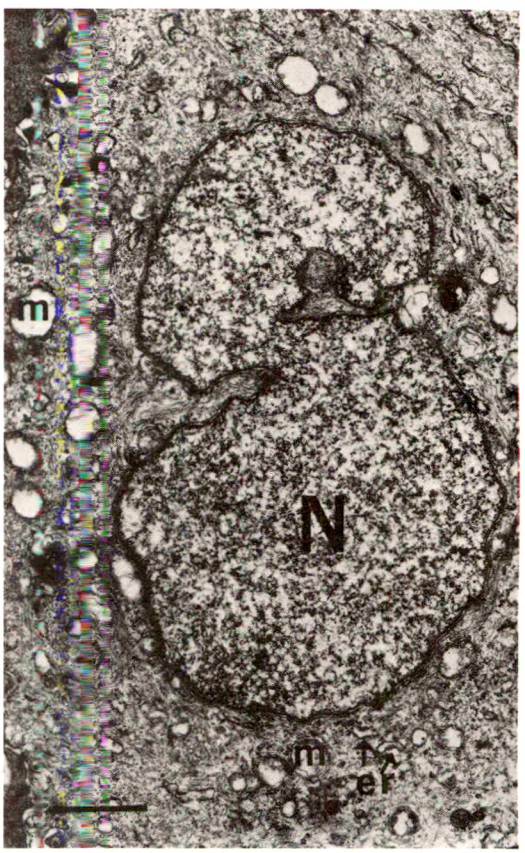


Fig. 3 (Yamaguchi, Polack, and Valenti). Electron micrograph of basal epithelial cells shows distended endoplasmic reticulum (er) and swollen mitochondria (m). Electron dense cell is at left of the picture. Nucleus (N) (×11,817, bar gauge = 1μ).

right eye at the time of surgery. Half of the specimens were fixed in 100% formalin and embedded in paraffin. The paraffin sections were stained with PAS, hematoxylin-eosin, and toluidine blue. The other half was fixed in 3% glutaraldehyde with phosphate buffer (pH 7.2) for 30 minutes, postfixed in cold 1% osmium tetroxide with the same buffer for 90 minutes, dehydrated in graded alcohols, and embedded in Epon.

Embedded tissues were cut with a microtome and stained with uranyl acetate and lead citrate. Electron micrographs were taken.

RESULTS

Light microscopy—The epithelium, which was separated from the stroma, had seven layers and in several areas of the basal layer, there were some dark epithelial cells (Fig. 2).

The removed stroma had a thickness of

70 to 80 μ. There was no Bowman's membrane; however, epithelial cell debris was present on the superficial stroma. This tissue had three distinct layers. The superficial layer was characterized by an almost total absence of keratocytes and a profuse deposition of granular eosinophilic material stained with PAS. The intermediate layer was characterized by vacuolated structures and a few normal keratocytes. The deeper layers had a normal arrangement of collagen lamellae and normal keratocytes. Inflammatory cells were not found. The conjunctival tissue appeared normal.

Electron microscopy—Surface and wing epithelial cells appeared normal. In some areas, cell membranes and hemidesmosomes as well as intercellular spaces of the basal cells were artifactually disrupted (Fig. 3). Desmosomes were well preserved and basal cells contained distended endoplasmic reticulum and swollen mitochondria. Some basal cells were in mitosis. Dark cells were slender in shape with few organelles, and prominent tonofilaments.

The anterior stroma had three characteristic layers (Fig. 4). The superficial layer of the specimen had clumps of curly dense collagen fibrils and a few severely degenerated keratocytes (Figs. 4, left, and 5). These are considered typical changes in this disease. The superficial collagen lamellae in this last keratectomy were disorganized and replaced by dense, short, and curly collagen similar to that described in our first specimen.[19] It appears, however, that the collagen fibrils of the first specimen were of a larger diameter than those of the present case (15 vs 10 nm). Degenerated kerato-

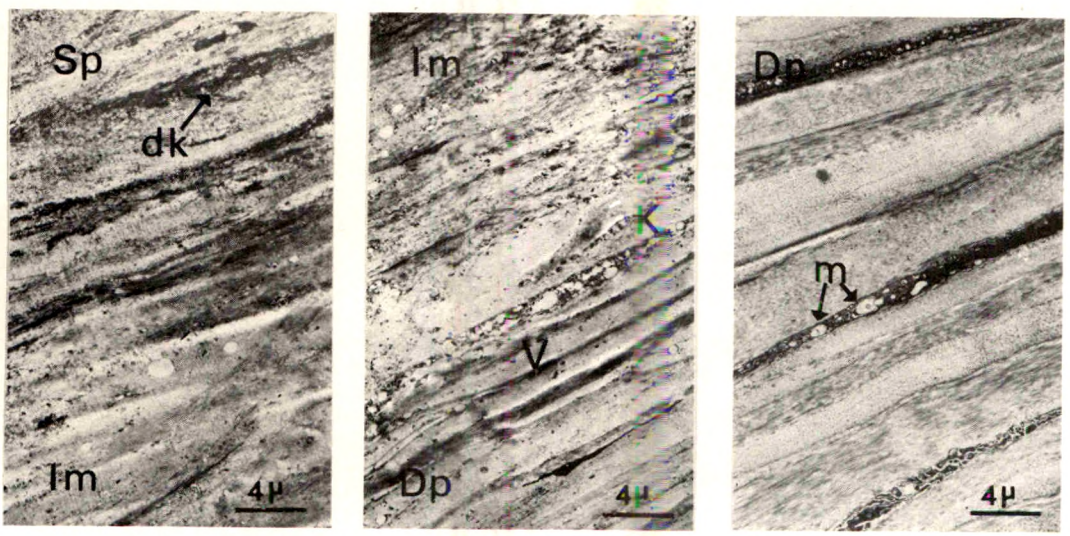


Fig. 4 (Yamaguchi, Polack, and Valenti). Anterior stroma has three characteristic layers. Left, Low magnification photograph of the superficial stroma tissue shows disorganization of the collagen lamellae. The superficial layer (Sp) has few degenerated keratocytes (dk). Intermediate layer (Im) (×2,307). Middle, Low magnification of the intermediate layer (Im) and deeper layer (Dp) of the stromal tissue. Keratocytes (K) of the second layer were degenerated and had many oval-shaped vacuoles (V) (×2,307). Right, The deeper layer has a normal arrangement of collagen lamellae and a normal number of keratocytes, which have distended mitochondria in their cytoplasm (m) (×2,307).

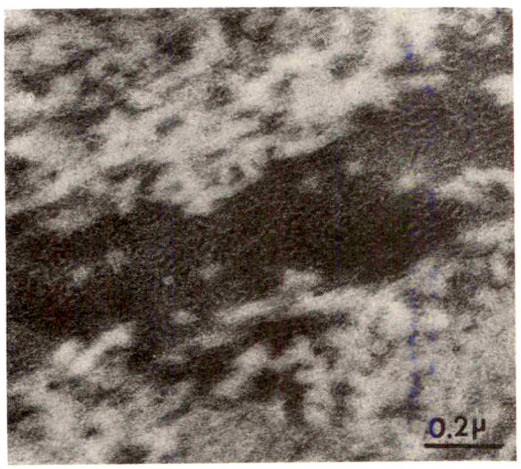


Fig. 5 (Yamaguchi, Polack, and Valenti). High magnification of curly dense collagen fibrils of the superficial layer. They are aggregated in masses and tangled (×48,468).

cytes were often seen surrounded by this curly denser collagen (Figs. 4, left, and 6, top). The lamellae surrounding the curly collagen material was consistently difficult to stain for electron microscopy. Where the superficial and the intermediate layers met, degenerated keratocytes were surrounded by masses of dense collagen fibrils (Fig. 6, middle). The intermediate layer had slightly disorganized collagen lamellae. The keratocytes were degenerated and contained oval-shaped vacuoles but no organelles in their cytoplasm (Fig. 4, center). These vacuoles seemed to be filled with a lipid-like substance (Fig. 7). The deepest layer of the keratectomy specimen had a normal arrangement of collagen lamellae (Fig. 4, right). Keratocytes at this level showed some degree of degenerative changes such as vacuolation, distended mitochondria, and disappearance of plasma membrane (Fig. 6, bottom). Some cells had prominent filaments in their cytoplasm.

The conjunctival epithelium consisted of 12 to 13 cell layers. The cells had normal organelles in their cytoplasm

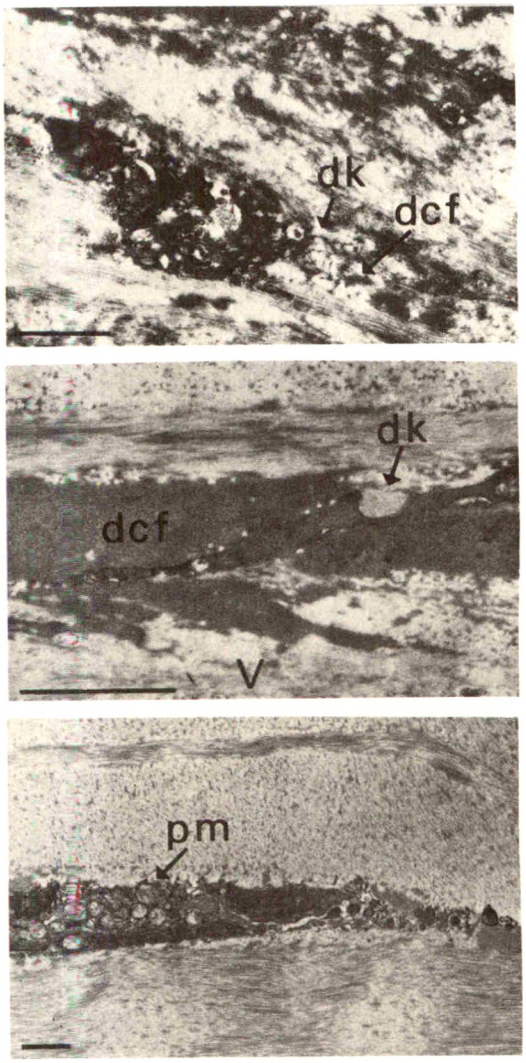


Fig. 6 (Yamaguchi, Polack, and Valebti). Top, Degenerated keratocytes (dk) of the superficial layer. Their plasma membranes have disappeared. Degenerated keratocyte seems to be replaced with denser collagen fibrils (dcf) (×12,073, bar gauge = 1μ). Middle, Degenerated keratocyte (dk) at the area where the superficial and the intermediate layer meet. Degenerated keratocytes surrounded by a mass of dense collagen fibrils (dcf); V (vacuoles) (×13,426, bar gauge = 1μ). Bottom, Keratocyte of the deeper layer. Plasma membrane (pm) has disappeared (×6,154, bar gauge = 1μ).

with normal junctional complexes; however, in some areas the basement membrane was disrupted and duplicated by clumps of short collagen fibers. The

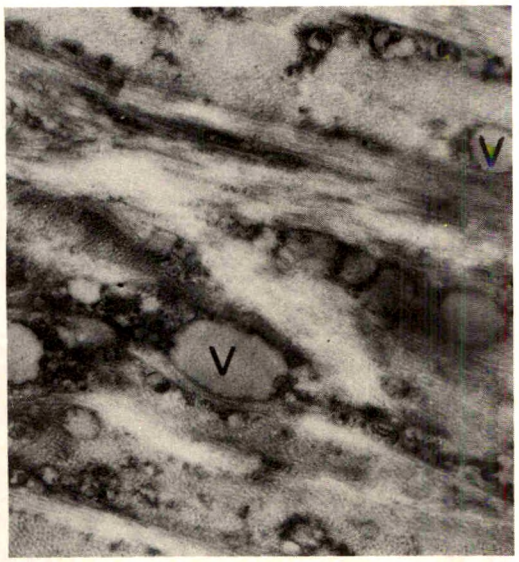


Fig. 7 (Yamaguchi, Polack, and Valenti). Higher magnification of oval shaped vacuoles (V) in degenerated keratocytes of the second layer. These vacuoles seem to be filled with a lipid-like substance (×8,871).

conjunctival stroma contained fibroblasts and macrophages.

Discussion

There are two hypotheses for the cause of Reis-Bücklers' dystrophy. The first is that the primary changes occur in Bowman's membrane with secondary epithelial changes.[10,15-17] The other theory is that an abnormal epithelium leads to degeneration of keratocytes and Bowman's membrane.[12,14,18,21] Even though initial changes are found in Bowman's membrane, as reported by Griffith and Fine,[15] and by Jones and Stauffer,[13] the recurrence in our case nine years after a keratectomy, lends support to the second theory. Degenerative changes were distributed clearly in three levels of the specimen, but were much more pronounced in the superficial stroma. In this layer, keratocytes were completely degenerated, as well as the corneal collagen. The collagen lamellae in this area were completely disorganized. In their place were short, dense curly collagen fibrils. These curly fibrils have been found in all previous electron microscopic studies of this dystrophy[15-21] and appear to be an important diagnostic aspect. The changes were more discrete in the deeper specimen layers, thus suggesting that the keratocytes were not the primary cause of the disease. Some investigators[14,15-18] have suggested that the alterations in keratocytes were regenerative rather than degenerative, and that many of these cells were in an active stage. Hogan and Wood[17] studied the specimen of a 26-year-old man and thought that the new connective tissue was produced by the epithelium and that the increased number of fibroblasts in the gaps or ruptures in Bowman's membrane were associated with the production of new collagen fibers and microfibrils. Jones and Stauffer,[13] by using the light microscope, found few keratocytes in the subepithelial connective tissue layer in the specimen of a 50-year-old patient with recurrence in a graft. In our case there was no evidence of keratocytes participating in corneal regeneration in the superficial layers. When comparing the electron microscopic alterations of the original keratectomy tissue and the present tissue, we found that the degenerative changes were more severe in the latter and that alterations of collagen fibers and keratocytes compromised more corneal layers than it did on the first specimen. This finding raises the question of whether Bowman's membrane had a protective function or acted as a barrier to some substance diffusing from the epithelial layer into the superficial stromal tissue. Teng[22] indicated that dark epithelial cells had a secretory function. Several of these dark cells were found in the epithelial layer and even though no enzyme substance has been found, it could be postulated that if such

an enzyme existed, it could permeate the superficial stroma and eventually cause the degenerative changes we have observed (Fig. 6). However, a disturbance in the production and distribution of basement membrane material apparently exists in the epithelial lining of the globe. This is shown by the reduplication and abnormal appearance of the basement epithelial membrane of the conjunctiva. Even though the abnormal epithelium and Bowman's membrane could be replaced with a corneal graft, these cells would eventually replace the graft epithelium and be participants in the alterations of the connective tissue and keratocytes. In our case, in which the epithelium was completely removed, the cornea was eventually covered by cells of conjunctival origin, which appeared to have a looser adherence to the diseased stroma. Even though this layer readily separated from the resected stroma, no healthy tissue or viable keratocytes were observed in the immediate subepithelial corneal layer.

Summary

We studied the cornea of a 51-year-old woman with recurrent Reis-Bücklers' dystrophy with the transmission electron microscope. The patient had had a superficial keratectomy nine years earlier.

The epithelium removed in a second keratectomy contained distended endoplasmic reticulum and swollen mitochondria. The basal layer showed some cells in mitosis and a slight increase in the number of dark cells. The anterior portion of the removed stroma contained dense collagen fibrils, with the appearance of curly fibrils, which is characteristic of this disease. There were also severe degenerative changes in the stroma. These changes in the collagen fibers and alterations in keratocytes of the deeper layers were less severe than those of the superficial layer. A biopsy of the bulbar conjunctiva showed reduplication of the basement membrane.

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OPHTHALMIC MINIATURE

We find no notice in the Journal whether its appearance will be weekly, monthly, or semi-annually. We trust, if we miss the "brilliant original contributions," we may find copious reports of the actual experience of American practitioners in succeeding issues, and also that the profession will contribute liberally to the subscription list of the American Journal of Ophthalmology.

American Medical Monthly 18:146, 1862

HEIGHT OF THE TARSUS OF THE LOWER EYELID

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The tarsal plate forms the supporting structure, or skeleton, of the eyelids. In performing operations that require a vertical incision through the lower eyelid, we have observed the height of the lower tarsus to be significantly smaller than shown in previous published reports.[1-8] Therefore, we have undertaken a study in both patients and cadavers to document the height of the lower tarsus to aid in eyelid reconstruction.

SUBJECTS AND METHODS

A calibrated Castroviéjo caliper was used to measure the vertical height of the lower tarsus in the middle third of the lower eyelid in 23 eyelids of 20 patients submitting to procedures to correct eyelid notching (one case), perform full-thickness lower eyelid biopsy (one case), correct medial ectropion (four cases), and remove basal cell carcinomas (14 cases). In each case a perpendicular incision was made in the middle third of the lower eyelid with a No. 15 Bard-Parker blade full thickness through the eyelid margin. The tarsus was seen as a white glistening structure, in contrast to the darker layers of orbicularis oculi muscle and the fibroelastic lower eyelid retractors (Fig. 1). If this contrast was obscured by initial bleeding, a cotton-tipped applicator with saline was used to clean any blood from the tarsal plate so that tarsal borders

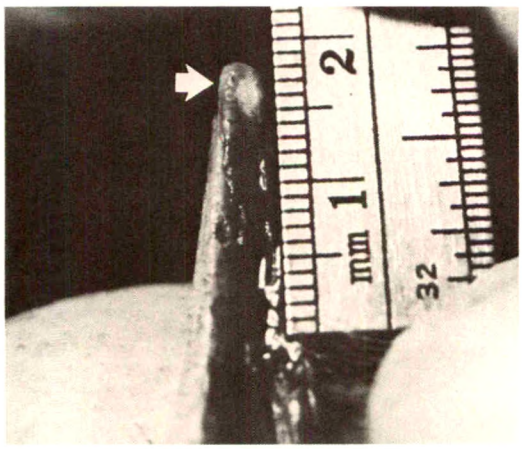


Fig. 1 (Wesley, McCord, and Jones). Vertical incision through lower eyelid after excision of basal cell carcinoma shows vertical height of lower tarsus (arrow).

stood out distinctly from the orbicularis oculi muscle and the fibroelastic tissue (Fig. 2). Initially an operating microscope was used in five cases but the magnification did not add any significant accuracy to the measurements, which were recorded to the nearest 0.1 mm. In each case the vertical incision was made through a section of eyelid, which was clinically normal. In the cases involving basal cell carcinoma, the eyelid tissue was normal also histologically on frozen section. The side of the body, age, sex, and diagnosis of the patients were recorded.

For comparison, measurements of the lower tarsus were performed on 24 lower eyelids of 17 cadavers with vertical sections made through the center of the lower eyelid. Magnification (×4) was used in the cadaver studies to perform the measurements with a Castroviéjo caliper because the tarsus in the cadaver is less distinct from the pale layers of

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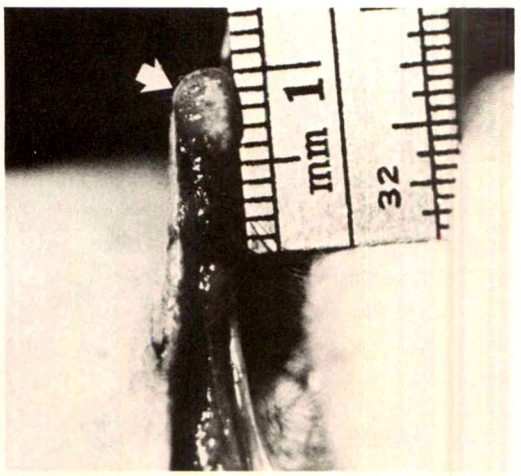


Fig. 2 (Wesley, McCord, and Jones). After blood is sponged from lower eyelid with medial ectropion, vertical height of the tarsus (arrow) is apparent.

orbicularis and lower retractors. Only cadavers with normal appearing eyelids were used in this study.

Results

The average vertical height of the lower tarsus in our patients submitting to eyelid procedures was 3.7 mm (S.D. ± 0.3), which was statistically different ($P < .05$) from the 5- to 7-mm values reported previously.[1-8] The values of the lower tarsus ranged from 3.2 to 5.0 mm, but a lower tarsus greater than 4.0 mm was found in only two of the 23 eyelids measured at surgery. The average value of cadaver lower eyelid tarsus was 3.9 mm with a range of 3.0 to 5.0 mm (S.D. ± 0.5). No average difference was found between right and left eyelids, although one patient with medial ectropion had a right lower tarsus of 4.0 mm and a left lower tarsus of 5.0 mm. In 14 men the value of the lower tarsus was 3.8 mm and the average lower tarsus in nine women was 3.6 mm, which was not statistically different. Additionally, the average age of the women (64 years) was older than that of the men (41 years). There was no statistical correlation of smaller tarsus with advancing years. No differences in tarsal height were found according to diagnosis. In cases in which measurements were made from both lower eyelids, all differences were 0.2 mm or less with the exception of a single cadaver difference of 0.4 mm. One patient with bilateral medial ectropion had a 1.0-mm difference.

The marginal artery in 20 out of 24 cadaver eyelids could be identified lying anterior to the tarsus at approximately 1 mm above the base of the tarsus. The marginal artery was identified in the corresponding location in the lower eyelids of nearly all our patients.

Discussion

In procedures to reconstruct or repair the lower eyelid, sutures placed through the tarsus, or skeleton, of the lower eyelid bear most of the tension as opposed to sutures passed through skin, orbicularis oculi muscle, conjunctiva, or the fibroelastic lower eyelid retractors just below the tarsus. If a tension-bearing suture intended for the tarsus is inadvertently placed through the lower eyelid retractors or orbicularis oculi muscle instead of the tarsal plate, the wound may later separate, which would result in an eyelid notch or unnecessary scarring of the skin.

If a vertical, full-thickness eyelid incision is made through the lower eyelid, the white, glistening tarsus (Figs. 1 and 2) is distinct from the darker orbicularis oculi muscle or lower eyelid retractors. Bleeding after the initial eyelid incision may stain the white tarsus and reduce the contrast with surrounding tissues, but a saline-soaked, cotton-tipped applicator can be used to clean blood from the tarsus to restore contrast. By visualizing the tarsus at surgery, at least two tension-bearing sutures can be placed through the tarsus and a deep stitch

through the gray line (mucocutaneous junction) will pass through the tarsus for additional support.

The average value of the lower tarsus in our patients of 3.7 mm (± 0.3) is significantly different both surgically and statistically ($P < .05$) from the 5 to 7 mm described in previous reports.[1-8] None of these sources indicated how a determination was performed. In all but two of our 23 patients the vertical height of the lower tarsus was 4.0 mm or less. One patient with medial ectropion had a left tarsus of 5.0 mm; the oposite eyelid with a similar clinical appearance measured only 4.0 mm. None of the other patients with medial ectropion had an unusually large tarsus. No correlation of tarsus size by age, sex, or diagnosis was shown.

The average value of 3.9 mm (S.D. +0.5) found in cadavers was not statistically different than that in our patients. The larger variation of measurements in cadavers may be the result of varing states of hydration. Some of the larger estimates of the tarsus found in previous reports may have come from observing cadaver eyelids.[1] This error of overestimating the tarsus in cadaver eyelids is explained by the lack of contrast between the tarsus and other eyelid tissues after death. In our study ×4 magnification was required to delineate tarsus from lower eyelid retractors. Without magnification the entire lower eyelid appeared to be tarsus.

Many operations have been described for correction of ectropion and entropion that show the tarsus to be much larger than we have shown. The success of these operations may result not from the various geometric configurations of full-thickness eyelid excision, but rather from the tightening effect on the lower eyelids and possibly the creation of an internal vertical scar that may strengthen the attachment of the lower eyelid retractors to the tarsus.

Our finding that the marginal artery lies usually 1 mm above and anterior to the base of the lower tarsus supports the concept that the incision in the Cutler-Beard procedure,[9,10] to reconstruct the upper eyelid, should be 5 mm below the lower eyelid margin to preserve the vascular supply to the remaining lower eyelid margin left as the bridge. However, no tarsus will be included in the flap used to reconstruct the upper eyelid, because this incision will be below the base of the lower tarsus.

The height of the upper eyelid tarsus was not measured in this study because the upper tarsal height is obvious when everting the upper eyelid and the measurements reported previously of 8 to 12 mm[1-8] correspond with our results. The size of the lower tarsus is not so easily shown by everting the eyelid; however, the conjunctiva over the lower tarsus has a deeper red appearance than the light palpebral conjunctiva over the lower eyelid retractors.

Although all our measurements were performed in patients with eyelid disease, in each case the vertical full-thickness incision was performed in an area of the eyelid that was clinically normal and histologically benign. Additionally, the insignificant difference between the height of the tarsus in our patients (average 3.7 mm) and the measurements in cadavers with no eyelid disease (3.9 mm) supports our experience that the height of the lower tarsus, or skeleton of the lower eyelid, is usually 4.0 mm or less.

Summary

The vertical height of the lower eyelid tarsus of 20 patients submitting to lower eyelid resections and the lower tarsus of 17 cadavers was 3.7 mm (± 0.3) and 3.9 mm (± 0.5), respectively. The average value found in our patients was statistically different ($P < .05$) than the values of

5 to 7 mm reported previously. The lower tarsus should be clearly identified in procedures involving eyelid repair or eyelid reconstruction so that tension-bearing sutures can be placed through the tarsus.

ACKNOWLEDGMENT

David Myrich, of Storz Instrument Co., provided instrumentation and assistance in this study.

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ORGANIC NITRATE THERAPY IN GLAUCOMA

ANGELIKA J. S. WIZEMANN, M.D., AND VOLKER WIZEMANN, M.D.
Giessen, Germany

In 1915 Wessely[1] observed that amyl nitrite causes an increase of intraocular pressure (IOP) in cats. Since then the use of organic nitrates has been considered to be contraindicted in glaucoma. Although the studies on amyl nitrite showed controversial results concerning the effect of that substance on IOP[2-8] no case of nitroglycerin causing glaucoma had been reported[9-14] and some authors warned of the use of organic nitrates in the presence of glaucoma.[15,16]

Because coronary heart disease as well as glaucoma occur more frequently in the elderly the application of organic nitrates is of interest clinically. Specific doses of nitroglycerin given intravenously by perfusion technique and high doses of isosorbide dinitrate administered orally were used. We describe herein the effect of organic nitrates on IOP.

SUBJECTS AND METHODS

Three categories of patients were selected for this study: (1) Ten volunteers with normal IOP, open angles, and no history of glaucoma; (2) 25 patients with open angles and borderline or increased IOP without cupping of the disk, in whom antiglaucomatous treatment had not yet been started; (3) 20 patients with narrow angles and borderline or increased pressure without glaucomatous cupping, not under treatment when tested. Intraocular pressures were determined with a Goldmann applanation tonometer. All measurements in each patient were made by one person. Arterial blood pressure was recorded every 30 minutes.

The following three studies were performed: (1) Dose response: by means of a perfusion system, nitroglycerin was given intravenously in doses of 9.7 to 194 μg/minute. Intraocular pressure was measured every five minutes. A dose-response curve was obtained by changing the infusion speed. Beginning with the lowest dose of nitroglycerin, the dose was increased when a plateau of IOP was reached (usually after 20 to 30 minutes). (2) By administering 97 μg of nitroglycerin per minute, changes in IOP were recorded in five- to 20-minute intervals. (3) Ten volunteers and 44 patients took 40 mg of isosorbide dinitrate orally at 8 o'clock in the morning. Intraocular pressure was measured at 30- to 60-minute intervals thereafter.

In three additional patients with open-angle glaucoma, antiglaucomatous local therapy was interrupted and 2 × 40 mg of isosorbide dinitrate was given orally for three weeks after a two-day interval. Arterial blood pressure was measured at 30-minute intervals when nitroglycerin was infused. In patients undergoing an isosorbide dinitrate therapy, arterial blood pressure was recorded three times a day. In ten patients taking 80 mg of isosorbide dinitrate per day for at least two weeks, blood levels of methemoglobin were determined by using the spectrophotometric method of Evelyn and Malloy.[17]

RESULTS

Nitroglycerin administered intravenously led to a dose-dependent decrease

From Justus Liebig-Universität, 63 Giessen, FRG, Universitäts-Augenklinik, Friedrichstrasse (Dr. A. Wizemann), and Medizinische Universitätsklinik, Klinikstrasse (Dr. V. Wizemann).

Reprint requests to Angelika J. S. Wizemann, M.D., Univ.-Augenklinik, D-63 Giessen, Germany.

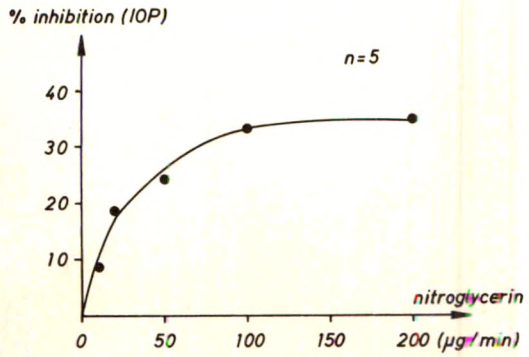


Fig. 1 (Wizemann and Wizemann). Dose response relation between intravenously administered nitroglycerin and IOP.

in IOP. At a dose of 9.7 µg/minute, IOP was decreased by 9%. Doubling of the dose resulted in an inhibition of IOP by 19%; 50 µg/minute of nitroglycerin inhibited by 24%. The maximum effect occurred at a dose of 100 µg/minute. A further increase in dosage of nitroglycerin did not exceed the maximum effect (Fig. 1).

When nitroglycerin was infused with a constant dose of 97 µg/minute, a decrease of IOP was recorded in patients with healthy eyes and patients with open and narrow angles. Patients with open angles responded with a maximum decrease in IOP of 50% after 30 minutes of infusion. The effect was constant during the infusion time of 120 minutes. In patients with narrow angles the action of nitroglycerin on IOP was less pronounced and during infusion there was a tendency of an increasing IOP (Fig. 2). Persons with normal IOP and no history of glaucoma showed a similar inhibition pattern under nitroglycerin infusion. A comparable result was obtained when 40 mg of isosorbide dinitrate (Sequels) was given orally twice daily. A decrease of IOP was detected in all 25 patients with open-angle glaucoma after 30 minutes. A 40% decrease in IOP lasted for five to six hours (Fig. 3). During the next

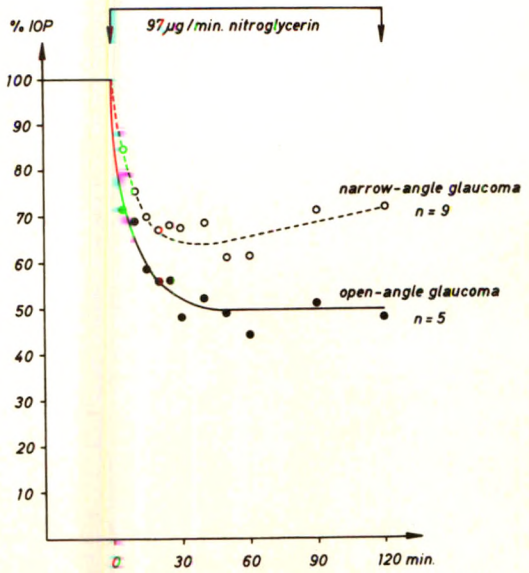


Fig. 2 (Wizemann and Wizemann). Effect of 97 µg of nitroglycerin per minute on IOP.

hours IOP increased slowly. The ten patients with normal IOPs and the 20 patients with narrow-angle glaucoma responded with a decrease in IOP of 25%. In three patients who responded poorly to topical treatment the therapy was interrupted for two days. When isosorbide dinitrate (2 × 40 mg/day) was given, IOP returned to normal (Fig. 4).

Approximately 20% of the patients who took isosorbide dinitrate orally complained of headaches. When the therapy was continued, headaches disappeared

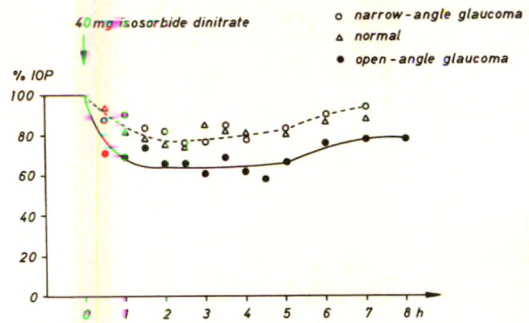


Fig. 3 (Wizemann and Wizemann). Reaction of IOP after 40 mg of isosorbide dinitrate (Sequels) administered orally.

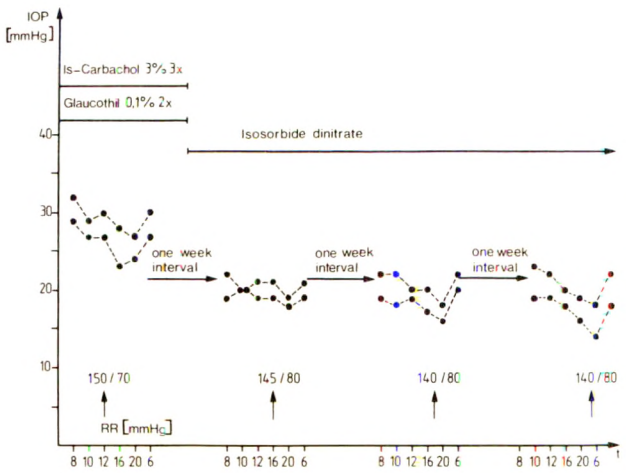


Fig. 4 (Wizemann and Wizemann). Intraocular pressure of a patient with open-angle glaucoma after isosorbide dinitrate monotherapy.

except in one patient who was omitted from the study. Systemic blood pressure was least influenced by the doses used when orally administered. Supermaximal doses of 194 μg/minute of nitroglycerin intravenously decreased mean arterial blood pressure by 10 to 20 mm/Hg. When the infusion was withdrawn, blood pressure returned to normal within one hour in all cases.

In 16 patients taking isosorbide dinitrate orally for more than six months, no signs of cardiovascular or neurologic disorders could be detected. An escape phenomenon concerning the decreasing effect on IOP has not been seen yet. Methemoglobin concentrations in blood averaged 0.9% of total hemoglobin (range 0 to 2.7%). In no case could a pathologic value of more than 3.0% methemoglobin be measured.

Discussion

Organic nitrates such as nitroglycerin or isosorbide dinitrate have a direct relaxing action on the smooth muscles of the blood vessels, mainly the large arteries and the venous capacity system.[18] This leads to a venous pooling and a decrease of myocardial preload. Consequently, those drugs are used in the therapy of angina pectoris and congestive heart failure. In recent years organic nitrates have been used in the therapy of acute myocardial infarction.[19] The cellular action of organic nitrates is still unknown.

Nitroglycerin inhibits the calcium influx in potassium depolarized coronary smooth muscle.[20] Because there is a specificity of this action to coronary vessels it seems unlikely that side effects of nitrates, as on the blush areas of the skin, the meningeal and intracranial vessels[21] and the observed effect of the IOP can be explained by calcium antagonism.

The decrease of IOP after intravenous or oral administration of nitrates did not correlate with the pattern of systemic blood pressure, thus indicating that systemic hemodynamic changes do not influence the effect on IOP. The dose of nitrates used in our experiments exceeds those of previous studies. We believe that the ineffectiveness of nitrates on IOP described by others[7,10-14] is the result of the comparable low dose administered.

With the exception of minor headaches no side-effects occurred. It seems unlikely that nitrate-induced methemoglobin is responsible for the headaches,

because this symptom disappeared when the therapy was continued.

In approximately 200 patients who had acute myocardial infarction, and who were treated by intravenous organic nitrates in 1979, no neurologic disorders have been seen by us. Because organic nitrates increase cardiac output in congestive heart failure, cerebral symptoms often improved.

Because about one third of all elderly men suffer from coronary heart disease, organic nitrates can exert only beneficial cardiac side-effects, except in patients with mean arterial pressures below 80 mm/Hg. Our preliminary study shows a potential new indication for a drug that has been used for other purposes for more than 100 years.

Summary

We determined the effect of organic nitrates on normal volunteers and patients with open-angle and narrow-angle glaucoma. Nitroglycerin lowered intraocular pressure in all patients when administered by the perfusion technique. Isosorbide dinitrate given orally (2 × 40 mg per day) has a potent decreasing effect on intraocular pressure, which cannot be explained by the influence on arterial blood pressure.

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NOTES, CASES AND INSTRUMENTS

THE USE OF DISPOSABLE CAUTERY TO SCRAPE CORNEAL ULCERS

IRENE OLIJNYK, M.D.,
ANTHONY L. MARCHESE, PH. D.,
AND JAMES E. MCDONALD, M.D.
Maywood, Illinois

To scrape corneal ulcers for smear, culture, or both, we have had good results with a disposable cautery. The unit we have used is the Concept Accu-Temp handheld disposable cautery (Figure). This cautery is a "hot cautery" by virture of its ability to achieve a hot temperature upon activation. (Most handheld cauteries may be used if they are two-cell instruments with alkaline 1.5 V batteries.) Our measurements show that the tip reaches a maximum of 2,300°F (1,260°C) as specified by the manufacturer. Although it is designed as a single-use instrument, this refers to the sterility of the entire instrument. According to our measurements, a temperature of about 1,560°F (850°C) is necessary to make this particular tip glow red. As long as the instrument achieves a red glow when activated, the tip is sterile.



Figure (Olijnyk, Marchese, and McDonald). Close-up of the Concept Accu-Temp handheld cautery for use in the diagnostic scraping of corneal ulcers.

The cautery tip is heated by depressing the activating button until the tip has a red-hot color. The tip is then allowed to cool. The result is a sterile, sturdy, and well-defined tip. The tip can then be used to scrape the borders of a corneal ulcer and the scrapings applied to a slide for smear or inoculated onto culture media. This process can be repeated many times as long as the tip achieves a red-hot color upon activation.

SUMMARY

We used a handheld disposable cautery for the diagnostic scraping of corneal ulcers. Sterility of the tip is obtained by activating the unit until the tip reaches a red-hot color and then allowing it to cool. The unit has a sturdy, well-defined tip, and is self-sterilizing, reusable, readily available, and simple to use.

From the Department of Ophthalmology, Loyola University Medical Center, Maywood, Illinois.
Reprint requests to James E. McDonald, M.D., Department of Ophthalmology, Loyola University Medical Center, 2160 S. First Ave., Maywood, IL 60153.

OCULAR EXAMINATION CART FOR THE OPERATING ROOM

PAUL E. ROMANO, M.D.
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At my hospital, the ophthalmology service does not have its own operating room. Moreover, I often perform ocular examinations while children are under

From the Division of Ophthalmology, Children's Memorial Hospital, and the Department of Ophthalmology, Northwestern University Medical School, Chicago, Illinois.
Reprint requests to Paul E. Romano, M.D., Children's Memorial Hospital, 2300 Children's Plaza, Chicago, IL 60614.



Figure (Romano). Operating room ocular examination cart.

anesthesia for surgery on other services. To provide the necessary convenience and mobility, I designed a mobile cart to have all ocular examination equipment available (Figure). It has served all the desired functions with little or no additional modifications over the years. The original cart was an adaptation of a unit of refracting lane equipment available from Surgical Mechanical Research.

The modifications and features include a folding extension table at the left side, a single power cord for unit and accessories, multiple extra power outlets at rear, extra drawers, and oversized lockable casters.

The cart contains the following equipment: skiascopy racks (plus and minus), trial lens set, prisms for measuring deviations, eyedrops, ointments, pneumatic tonometer, sterile instrument sets for measuring and manipulating the globe, retinoscope, direct ophthalmoscope, indirect ophthalmoscope, handheld slit lamp, gonioscopy lenses, and gonioscope.

SUMMARY

I modified an office refraction and treatment cart in order to have mobile and convenient ocular examination equipment easily available in the operating room.

ACKNOWLEDGMENT

E. Grenz assisted in the design and construction of the mobile cart.

MEETINGS, CONFERENCES, SYMPOSIA

EDITED BY THOMAS CHALKLEY, M.D.

ASSOCIATION FOR RESEARCH IN VISION AND OPHTHALMOLOGY 1980 SPRING MEETING

The Association for Research in Vision and Ophthalmology held its 52nd meeting in Orlando, Florida, May 4-9, 1980. Of the more than 1,300 abstracts received, 1,146 presentations were chosen for the meeting. Some of these were in the form of posters. The poster sessions have become popular. There was a slight increase in the number of preregistered individuals. Participants from 20 countries registered for the meeting and 126 presentations were given from countries other than the United States including 36 from Canada, 21 from Germany, 20 from the United Kingdom, 50 from Japan, six from Australia, and five each from Israel and Italy.

Highlights of the meeting included the presentation of the Friedenwald Award to Arnall Patz of Wilmer Institute and Johns Hopkins Hospital. Dr. Patz's lecture was entitled (1) "Studies on retinal neovascularization" and (2) "Jonas S. Friedenwald, man of science." The Proctor Medal was awarded to Bernard Becker of Washington University School of Medicine who lectured on "Vanadate and aqueous humor dynamics."

The officers of the Association are Gunter K. von Noorden, president; Claes Dohlman, vice president; Paul Henkind, secretary-treasurer; Paul O'Brien, president-elect; Nigel W. Daw, vice president-elect; and Alan Laties, editor-in-chief of "Investigative Ophthalmology and Visual Science."

The annual spring meeting for 1981 will be held in Sarasota, Florida, April 26th to May 1st.

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PREVENTION OF BLINDNESS IN THE AMERICAS

The World Health Organization and the Pan American Health Organization (PAHO) are developing a program for the worldwide prevention of blindness. The goal of the program is to eliminate avoidable blindness by reducing national blindness rates to less than 0.5% with no more than 1% in individual communities. The strategies are as follows:

1. To identify communities with a high prevalence of avoidable blindness and the causes of this blindness.

2. To design intensive programs for those communities which are most heavily affected. These would include simple treatment, both preventive and therapeutic, such as correction of malnutrition, control of infections, and surgery for cataract.

3. These measures would then become an integral part of primary health care.

4. The intermediate and central ophthalmic services would be strengthened as required to provide an adequate referral system and supervision and training of peripheral health workers.

Although most of the technology is available for these programs, research in some areas is needed. These research efforts should include the entire clinical field. Continued research is necessary so that the appropriate technology can be developed and applied.

Research is also needed to find the best means of collecting relevant information on the nature and magnitude of the problems of large areas. This information is needed for national planning of health services. The methods to be developed and field tested should be simple, reliable, economical, and reasonably standardized to permit paramedics to work under rugged field conditions.

Epidemiologic research should include (1) systematic collection of data on selected eye diseases; (2) multidisciplinary studies on the etiological factors of specific eye diseases and conditions; (3) surveillance of eye diseases; and (4) the development of realistic forms to collect data at the three principal levels.

The PAHO is interested in identifying academic and other centers in North America with individual investigators or

teams interested in participating in these types of research in Latin America and the Caribbean. The PAHO has had considerable success in promoting this type of collaborative research between "sister institutions" in North America and South America. If your institution is interested, the individual investigators and the areas of clinical and epidemiologic research in which they are interested should be specified. It would also be helpful to know whether they have specific contacts in Latin America or the Caribbean with whom further collaborative efforts would be appropriate. Funds are available through the PAHO for the initial visits between investigators, which could be used to design projects for further funding.

PAHO has asked Dr. Chandler Dawson, Director of the WHO Collaborative Center for Prevention of Blindness, the Francis I. Proctor Foundation, San Francisco, California, to collect and coordinate this information.

A. EDWARD MAUMENEE

CORRESPONDENCE

Letters to the Editor must be typed double-spaced on 8½ × 11-inch bond paper, with 1½-inch margins on all four sides, and limited in length to two manuscript pages.

Photohazards of Intraocular Lens Implants in Aphakia

Editor:

An important deficiency of intraocular lenses (IOLs) for use in aphakic eyes has been neglected. Such lenses, composed of polymethylmethacrylate, enhance the light-transmission properties of the anterior eye by transmitting and focusing blue and near-ultraviolet light on the retina, a situation that can impair vision and can enhance radiant energy damage to the retina. These statements are derived from recent research on altered eye and light interactions in individuals with aphakia and on wavelength-dependent damage to the retina. My colleagues and I[1-6] have stressed that the natural ocular lens of humans is pigmented and strongly absorbs shortwave blue and longwave ultraviolet light, filtering these wavelengths and preventing most of this radiant energy from reaching the retina. Mainster[7,8] has shown that the retinas of aphakic and pseudophakic (that is, IOL-containing) eyes receive large amounts of blue and near-ultraviolet light, thereby establishing a greater risk of retinal damage by these radiant energies. According to Ham and associates,[9-11] it is blue and near-ultraviolet light that is the most damaging to the retina.

These findings underscore the importance of providing individuals who have IOLs with photoprotective eyewear that absorbs light with wavelengths shorter than 450 nm, or to modify IOLs to absorb shorter wavelengths of light energy. This provides better visual acuity by eliminating chromatic abberation and protects the retina from shortwave-length light. Use of photosensitizing drugs such as epinephrine increases this hazard.[12]

Radiant energy thus represents another potential hazard of IOLs. Those already recognized include possible damage to the cornea (endothelium) and the iris, and many dangerous intraocular injuries that could result from the surgery or the loosening of the IOL from its site. I hope that general approval for large-scale use is delayed until all of these hazards are eliminated.

In my opinion, the use of IOLs introduces greater hazards to the eyes of people with aphakia than conventional visual aids. While vision in aphakic eyes is more improved by implantation of IOLs than by the use of spectacles or contact lenses, these other devices are

safer and now offer better retinal protection against radiant energy. Research and development aimed at improving spectacles and contact lenses for aphakia, as is being done with IOLs, may provide adequate visual acuity without these additional dangers.

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11. Ham, W. T., Jr., Mueller, H. A., Ruffolo, J. J., Jr., and Guerry, D., III: Solar retinopathy as a function of wavelength. Its significance for protective eyewear. In Williams, T. P., and Baker, B. N. (eds.): Effects of Constant Light on Visual Processes. New York, Plenum Publishing Corp, 1980.
12. Zigman, S.: Effects of near-UV light photoproducts of epinephrine on aqueous humor proteins. Invest. Ophthalmol. 13:127, 1974.

Large-Print Consent Forms

Editor:

Obviously, most individuals with bilateral cataracts have visual difficulties. One method of assisting these patients is the use of large-print materials. Most of the handouts used in ophthalmology offices, however, are typewritten or printed in regular-sized print.

In addition to the many large-print materials and other aids provided for the individuals with poor visual acuity, the National Association for the Visually Handicapped (NAVH) has made available to ophthalmologists the minimum standard informed consent form, in the official 18-point NAVH type, for patients requiring cataract operations, implantation of intraocular lenses, or both.

The form is printed on 8½ × 11-inch paper and contains 11 pages of information; it includes the patient's consent form and a form approving surgery on behalf of a minor or a disabled person. The form has been reviewed by two Food and Drug Administration groups (the Ophthalmic Device Classification Panel and the Bureau of Medical Devices) for compliance with the Administration regulations protecting human subjects.

This material does not contain any information about the NAVH nor is there a logo indicating its source. This form, which costs 25¢ per copy, is extremely useful. I suggest that ophthalmologists write or call the National Association for the Visually Handicapped, 305 E. 24th St., New York, NY 10010; telephone (212) 889-3141, to learn how the NAVH can assist us and our patients.

We should, of course, also remember the many other vision-oriented agencies that can assist in our practices.

FRANK J. WEINSTOCK, M.D.
Canton, Ohio

Corticosteroids and Varicella Disciform Keratitis

Editor:

We read with interest "Varicella dendritic keratitis" by Y. Uchida, M. Kaneko, and K. Hayashi (Am. J. Oph-

thalmol. 89:259, 1980), and wish to compliment the authors on their use of immunofluorescence to document the presence of varicella virus.

We note that corticosteroids were used in the therapy of their two patients and a recurrence of disciform keratitis ensued. We have described a patient with disciform keratitis who was not treated with corticosteroids.[1] He had no recurrence of the condition. Our patient's visual acuity improved from 6/30 (20/100) to 6/6 (20/20) within ten weeks. We wish to emphasize that corticosteroids may not be needed and may encourage relapses and recurrences in disciform disease secondary to varicella.

HOWARD H. TESSLER, M.D.
BURTON M. KRIMMER, M.D.
Chicago, Illinois

REFERENCE

1. Tessler, H. H., and Krimmer, B. M.: Disciform keratitis induced by varicella virus. Eye Ear Nose Throat Mon. 54:311, 1975.

Reply

Editor:

We think similar immune mechanisms play a role in producing disciform stromal disease in herpes simplex and herpes zoster as well as in varicella. Once herpetic stromal disease has been treated with corticosteroids, it is difficult to withdraw these drugs without recurrence of the disease, even if tapering regimens are undertaken. Our two patients with varicella disciform keratitis had been receiving corticosteroids before coming to our clinic. Recurrences encountered on cessation of these drugs led us to prolong administration. We think that we should have discontinued the corticosteroids despite the relapse. It is accepted in the treatment of disciform keratitis that when a patient has not been given corticosteroids previously and his disease is mild, these drugs should be avoided.

Drs. Tessler and Kimmer are correct, but we are still uncertain whether varicella disciform keratitis is always benign and can be cured by the use of cycloplegics alone.

YUKIO UCHIDA, M.D.
Tokyo, Japan

Pars Plana Vitrectomy Techniques—Correction

Editor:

We wish to correct an error in our article "Pars plana vitrectomy techniques for relief of epiretinal traction by membrane segmentation" (Am. J. Ophthalmol. 89:408, 1980). The instrumentation and technique for endophotocoagulation should have been referenced to Steve Charles who devised the Charles endophotocoagulator. The original manuscript accepted by THE JOURNAL contained this credit as a footnote, but this was inadvertently dropped. We believe this is one of the most important of Dr. Charles's many innovative contributions to vitreous surgery and regret that proper credit was not given in the published article.

TRAVIS A. MEREDITH, M.D.
HENRY J. KAPLAN, M.D.
THOMAS M. AABERG, M.D.
Milwaukee, Wisconsin

BOOK REVIEWS

The Ophthalmological Society of the United Kingdom. A Hundred Years of History. By Frank W. Law. Published by the Society, 1980, 72 pages. $6.50

Frank Law, ophthalmic surgeon, Knight of the Order of St. John, Hon-

orary Treasurer of the Ophthalmological Society of the United Kingdom (OSUK) for the past 33 years, the President of the Society in 1960 and 1961, author of Volume 2 of the "History and Traditions of the Moorfields Eye Hospital" (the first volume of which was written by Treacher Collins), wrote this engaging history to appear during the Centenary celebration of the OSUK. He combed the Transactions of the Society and the Council minutes to bring forth an intriguing history.

The history of this Society is more than the history of British ophthalmology for the Society now numbers more than 1,500 members, the majority of whom are nonresidents of the British Isles. The first American to be named an honorary member was Elkenah Williams in 1880. Williams was professor of ophthalmology at Miami Medical College and president of the International Congress of Ophthalmology in 1876.

In the appendices are listed the original rules of the Society with the rule changes as revised up to 1979, the presidents of the Society, the Bowman lecturers and their topics, and the recipients of the Society prizes, and the Royal Eye Hospital prize which the Society administers. This is a fine volume to commemorate the Centenary.

FRANK W. NEWELL

Practical Ophthalmic Microsurgery. By A. S. M. Lim. S. Karger, 1980. Hardcover, 88 pages, index, appendices, 70 color figures, 13 black and white figures.

This 85-page volume is designed for the ophthalmic surgeon already trained who has had no experience with the operating microscope. It comprises eight chapters, which reflect the author's own experiences and preferences in its use and describes his means of adapting to the microscope, and it describes some common errors in the use of the instrument. There are short sections on types of microscopes and microsurgical instruments, needles, and sutures. The section on basic techniques of suturing is particularly well done and the practical points made are well taken. Little information is included which cannot be found in other more extensive texts on the same subject and no attempt is made to describe in detail all microsurgical procedures. For this reason the volume will not serve as a reference. It will be helpful in organizing workshops and introducing the beginner to operating with the microscope.

The book is well written and easy to read. The illustrations are superb. The volume will be of interest to all who use the operating microscope and will be particularly useful to those who are just beginning.

SAMUEL D. MCPHERSON, JR.

Pathologie des Auges. By G. O. H. Naumann. Berlin, Springer-Verlag, 1980. Clothbound, 994 pages, index, 546 black and white figures, 192 tables. $380.80

This is a magnificent volume in the best European tradition of the all encompassing "Werk." Yet, it has a concise practical approach, sympathetic to American readers, which reflects Professor Naumann's association with the Armed Forces Institute of Pathology and the collaboration of David Apple. The book is one of 12 volumes in a series on pathologic subspecialities and is directed as much to the general pathologist doing ophthalmic pathology as it is to the ophthalmic pathologist or ophthalmologist.

It is this broad didactic purpose that

makes the volume so valuable. The text is unique both as a generalized reference work, complete in the differential diagnosis of ophthalmic disorders, and as a detailed exposition of attendant pathologic findings. Each condition is well illustrated with superb clinical photographs correlated with excellent light and electron micrographs and supplemented by some 118 tables of differential diagnoses and 115 schematic drawings of the histopathologic and anatomic processes involved.

Professor Naumann has been able to draw upon the clinical and histopathologic collections of the universities of Hamburg and Tübingen and the Illinois Eye and Ear Infirmary. Of special interest is the collection of the late Professor Stock (Tübingen 1921-1953) providing material, which as Professor Naumann notes, "would rarely lead to enucleation today."

Chapter organization is traditional and covers the various ocular structures as well as trauma, inflammation, and systemic diseases. New organizational concepts include Chapter 12 on the correlation of retinal pathology and ophthalmoscopic findings. Chapter 14 is devoted to glaucoma and the "hypotony-syndrome." The masterful combination of detailed yet concise text with clinical, histopathologic, and electron microscopic photographs, differential diagnostic tables and schematic drawings makes as good a chapter on glaucoma as can be found anywhere. Chapter 17 deals with ocular changes from drugs.

The index is complete, with good cross references. It allows full use of the book in either a clinical or pathology laboratory setting. There is a separate index for the differential diagnostic tables as well as one for the schematic drawings. The references given with each chapter are extensive, current and complete with respect to the American bibliography. This book would be a pleasure for any ophthalmologist to own. I highly recommend it to any colleague who reads German.

GERHARD W. CIBIS

Allergy and Immonology of the Eye. By Mitchell H. Friedlaender. Philadelphia, Lippincott/Harper, 1980. Hardcover, 338 pages, preface, index, 146 black and white figures. $27.50

This scholarly text covers all pertinent aspects of ocular immunology to date. It is divided into 12 chapters. The first two chapters deal with the basics of immunology such as the nature of the immunoglobulins, what they do, the subsets of lymphocytes, and how tissue is injured in immune interactions. These two chapters have general referencing consisting of excellent basic-type texts on general immunology.

Chapter 3 (18 pages, 125 references) contains a distillate of papers on ocular aspects of the immune response. This chapter is arranged somewhat by anatomic area and by characteristics of various immune responses such as cellular immune response, cytotoxic reaction and anaphylactic reactions.

The remaining 237 pages of text, divided into nine chapters, are arranged not by anatomic area but by topic. These topics range from subjects such as the "Eye and the Skin" (Chapter 4) to "Neurologic and Endocrine Diseases in the Eye" (Chapter 10). Within each chapter are the various diseases. Each disease is taken up as to the known and expected immunopathology, clinical features of the disease in general, ocular findings, differential diagnosis, and treatment. The chapters are well referenced (1,083 references for these nine chapters).

This book contains valuable information on the growing body of inflammation we call ocular immunology. It is the most comprehensive compilation to date of ocular immunologic information.

It is recommended for those at any level or in any discipline who would like to know what ocular immunology is and would like to have one book that contains all, or nearly all, of the pertinent references for the field of ocular immunology.

MATHEA R. ALLANSMITH

Oculoplastic Surgery. By Charles E. Iliff, W. Jackson Iliff, and Nicholas T. Iliff. Philadelphia, W. B. Saunders Co., 1979. Hardcover, 348 pages, index, 174 black and white figures, 535 color figures. $110.

This text is a summary of ophthalmic plastic and reconstructive surgery, based on the 30-year experience of Dr. Charles Iliff, with significant contributions by his two sons. All the important areas of oculoplastic surgery are covered. These include wound closure, skin grafts, basic flap preparation, blepharoptosis repair, blepharoplasty, ectropion, entropion, tumors and diseases of the eyelids and orbit. Attention is given to lacrimal tract surgery, rehabilitation of patients with thyroid disease, various congenital defects, and trauma. Extensive color photographs show both the disease states and the surgical procedures used to correct them. The photographs are supplemented by excellent line drawings, which clarify the surgical principles of repair. The text is clear and well correlated with the illustrations.

The number of references is small, and when presented they are found at the bottom of the page rather than at the end of the chapter. For the most part the author gives his procedure for each situation and does not discuss alternative methods of repair. Little attention is given to complications. Anyone interested in this field will find many useful points to incorporate into practice even though one may not wish to use all the procedures presented. I have no reservations about recommending this book to residents, fellows in ophthalmic plastic surgery, and practicing ophthalmologists and oculoplastic surgeons. To ignore this book is to deny oneself the benefit of Dr. Iliff's vast experience.

FREDERICK MAUSOLF

Cecil Textbook of Medicine, 15th ed. Edited by Paul B. Beeson, Walsh McDermott, and James B. Wyngaarden. Philadelphia, W. B. Saunders Co., 1979. Hardcover, 2,478 pages, index, 226 black and white figures, 9 color figures. Single volume, $46; 2-volume set, $56.

Medical students may choose between three major textbooks of medicine: "Cecil Textbook of Medicine" (1979); "Harrison's Principles of Internal Medicine" (1977); and Harvey, Johns, McKuzick, Owens, and Ross's "The Principles and Practice of Medicine" (1980). The last is the Johns Hopkins descendant of Osler's "Practice of Medicine" and is written by the faculty of Johns Hopkins School of Medicine. Harrison depends heavily, but not exclusively, upon physicians in New England states. The contributors to Cecil Textbook are more dispersed throughout the country. Some authors contribute sections to two of the textbooks, but I could find no instance of any author providing chapters to all three.

These volumes have become so large that some reviewers give their weight before describing their contents. Cecil contains some 2 million words written by 237 authors. There are 2,350 pages of text and 119 pages of index. Harrison was

prepared by 206 authors and consists of approximately 1.75 million words, 2,100 pages of text, and 102 pages of index. The Hopkins book was prepared by 94 authors and contains about 1.3 million words of text in 1,510 pages of text and 66 pages of index.

Each volume is encylcopedic and comprehensive with no deference to students or the novice. Morris B. Landers of Duke provides a 23-page section on "Diseases of the Eye" in Cecil; whereas Neil Miller, Lawrence Hirst, and Arnall Patz provide a 15-page section and one color plate on ophthalmology in "Principles and Practice." Harrison does not contain a separate section on the eye, but devotes two color plates to fundus changes in various diseases. Their quality is so poor, however, one would be hard put to recognize the disorder from its picture.

The student will search in vain for mention of Horner's syndrome in pulmonary lesions, in all except "Principles and Practices in Medicine." There is no mention of cotton-wool patches in lupus erythematosus in Cecil and Harrison. Harrison has a fine section on chlamydial eye disease by Dawson. Jawetz discusses trachoma inclusion conjunctivitis in Cecil. Generally though the level of ophthalmic awareness is disappointing. It reflects the de-emphasis of ophthalmology in the medical school curriculum and the inability of many physicians to recognize even the simplest ocular signs of systemic disease.

FRANK W. NEWELL

SYMPOSIA

Topics in Neuro-Ophthalmology. Edited by H. Stanley Thompson, Robert Daroff, Lars Frisén, Joel S. Glaser, and Michael D. Sanders. Baltimore, Williams and Wilkins, 1979. Hardcover, 378 pages, list of contributors, index, over 200 black and white figures.

FRISÉN, L: **The cornerstones of perimetric strategy**

FRISEN, L.: **Kinetic perimetry. Techniques and strategies**

BURDE, R. M.: **Static perimetry**

HEDIN, A.: **Color in peripheral vision**

FRISEN, L.: **Simple visual field tests. Introduction**

FRISÉN, L.: **The tangent screen**

BURDE, R. M.: **Confrontation testing**

HEDIN, A.: **Color confrontation testing**

FRISEN, L.: **Topical diagnosis of visual field defects. Introduction**

FRISÉN, L: **Choriorentinal folds**

BURDE, R. M.: **Acute ischemic optic neuropathy**

FRISÉN, L.: **Optic nerve hypoplasia**

THOMPSON, H. S.: **Binasal field loss**

HEDIN, A.: **Temporal field defects of pre-chiamsal origin**

SAVINO, P. J., SCHATZ, N. J., CORBETT, J. J., AND ORR., L. S.: **Visual field defects in optic tract disease**

FRISÉN, L.: **The lateral geniculate body**

Glaucoma Update. Edited by G. Peter Halberg. Birmingham, Alabama, Inter-Optics Publications, Inc., 1980. Paperbound, 145 pages, list of contributors, 98 black and white figures, 7 color figures, 59 tables.

BLODI, F. C.: **The chamber angle in glaucoma**

SHAFFER, R. N.: **Otto Barkan and goniotomy**

KOLKER, A. E.: **Ocular hypertension**

KITAZAWA, Y., AND AOKI, S.: **Introduction of decision-making in the early diagnosis of primary open-angle glaucoma**

FRANÇOIS, J., AND DELAEY, J. J.: **Fluorescence angiography of the disc and the peripapillary choroid in glaucoma**

CRISTINI G., AND GIOVANNINI, A.: **Rheography in early stages of glaucoma**

SOUTHREN, A. L., WEINSTEIN, B., GORDON, G., AND DUNN, M. W.: **Lymphocyte receptors in glaucoma**

CINOTTI, A. A.: **Optic nerve and visual field in glaucoma**

TOWNSEND, W.: **The pathogenesis of secondary congenital glaucoma**

KARA, G. B.: **Pathogenesis of glaucoma in intraocular tumors**

HERSCHLER, J.: **Role of the ciliary processes in malignant (ciliary block) glaucoma**

TREDICI, T. J.: **The United States Air Force screening and management program for glaucoma. Evaluation after ten years**

KROHN, D. L., AND BREITFELLER, J. M.: **Transcorneal flux of pilocarpine**

ZIMMERMAN, T.: **Summary of clinical research with timolol**

KATZ, I. M.: **Timolol in aphakic patients**

KEATES, E. U.: **Extended evaluation of timolol combination therapy in chronic open-angle glaucoma**

NEWELL, F. W., AND STARK, P.: **Nabilone: A synthetic cannabinoid analogue in open-angle glaucoma in man**

KITAZAWA, Y., HORIE, T., AND TAKAHASHI, O.: **On the ocular hypotensive effect of topical atenolol**

HARRISON, R.: **Clonidine in glaucoma therapy**

CRISTINI, G., AND MEDUIR, R.: **The revascularization of the optic nerve in terminal glaucoma**

WORTHEN, D. M.: **Laser treatment for open-angle glaucoma**

LEE, P. E.: **Transpupillary laser photocoagulation of the ciliary processes in the management of aphakic glaucoma**

CORDERO-MORENO, R., AND DE PIFANO, I.: **Trabeculectomy. Clinicopathologic correlations**

D'AMICO, R. A.: **Glaucoma and keratoplasty**

HALBERG, G. P.: **Subscleral filtering. An eclectic procedure**

BURRIS, J. E., JR.: **An operation for co-existing open-angle glaucoma and senile cataract**

RIZZUTI, A. B., AND RIZZUTI, R. A.: **Cataract extraction combined with trabeculectomy**

DREWS, R. C.: **Glaucoma and lens implantation**

WEINSTOCK, F. J.: **Intraocular lenses in relation to intraocular pressure**

ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

American Journal of Epidemiology

THE EPIDEMIOLOGY OF MYOPIA. Angle, J., and Wissmann, D. A. (Dept. Sociology, Univ. Arizona, Tucson, Ariz.). Am. J. Epidemiol. 111:220, 1980.

Many people have reduced unaided vision because of myopia, a spherical error of refraction. The biological theory of myopia views myopia as the result of genetically determined characteristics of eye tissues, whereas the use-abuse theory views myopia as the result of habitual use of the eye at a near focal length, near-work. The use-abuse theory implies that myopia is preventable whereas the biological theory does not. Myopia varies over age, gender, race, ethnicity, level of education, social class and degree of urbanization. The explanation of the epidemiology of myopia in the use-abuse theory is that some types of people do more near-work than others. Using data from the Health Examination Survey of 12- to 17-year-olds conducted by the U.S. Public Health Service from 1966-1970, this paper finds that the use-abuse theory can explain at least some of the variance of myopia and much of the socially patterned variance. This finding raises the possibility that at least some of the myopia extant in a population is preventable. (3 tables, 36 references) — Authors' abstract

Archives of Ophthalmology

GLAUCOMA DUE TO ENDOTHELIALIZATION OF THE ANTERIOR CHAMBER ANGLE: A COMPARISON OF POSTERIOR POLYMORPHOUS DYSTROPHY OF THE CORNEA AND CHANDLER'S SYNDROME. Rodrigues, M. M., Phelps, C. D., Krachmer, J. H., Cibis, G. W., and Weingeist, T. A. (Clinical Branch, Natl. Eye Inst., Bethesda, Md.). Arch. Ophthalmol. 98:688, 1980.

Posterior polymorphous dystrophy and Chandler's syndrome are separate ocular diseases with certain clinical features in common. Both may cause endothelial dystrophy, corneal edema, iridocorneal adhesions, and glaucoma. Differences between the two disorders include the morphology of the endothelial dystrophy, hereditary transmission, laterality, and rate of progression. Histopathologic examination of trabeculectomy and iridectomy specimens from two patients with posterior polymorphous dystrophy and one patient with Chandler's syndrome disclosed a common basic pathologic process—endothelialization of the anterior chamber angle. Ectopic corneal endothelium and abnormal Descemet's membrane extended across the trabecular meshwork and onto the anterior surface of the iris. The appearance of the endothelial cells, however, was strikingly different in the two conditions. The endothelial cells in posterior polymorphous dystrophy had ultrastructural characteristics of epithelial cells. Those in Chandler's syndrome were degenerated but retained ultrastructural features of endothelial cells. (9 figures, 1 table, 19 references)—Authors' abstract

CHARACTERISTICS OF INFANTILE ESOTROPIA FOLLOWING EARLY BIMEDIAL RECTUS RECESSION. Hiles, D. A., Watson, B. A., and Biglan, A. W. (Dept. Ophthalmol., Univ. Pittsburgh School of Med., Pittsburgh, Pa.). Arch. Ophthalmol. 98:697, 1980.

A homogenous group of infants with esotro-

pia whose eyes underwent surgical alignment before one year of age were studied. Three groups emerged: those who remained stable following their initial early alignment, those who were well aligned and remained stable for prolonged periods of time and then decompensated, and those who were unstable throughout the observation period. Preoperatively, no factors were identified that predicted into which group each patient might enter. By the end of the first decade of life all patients were able to achieve satisfactory ocular alignment. However, there is need for constant monitoring and repeated medical and surgical therapy. While most patients achieve normal and equal visual acuities, the fusion responses remain at diminished levels. (8 tables, 22 references)—David Shoch

RECLASSIFICATION OF INTRAOCULAR RETICULUM CELL SARCOMA (HISTIOCYTIC LYMPHOMA): IMMUNOLOGIC CHARACTERIZATION OF VITREOUS CELLS. Kaplan, H. J., Meredith, T. A., Aaberg, T. M., and Keller, R. H. (Dept. Ophthalmol., Medical College of Wisconsin, Milwaukee, Wisc.). Arch. Ophthalmol. 98:707, 1980.

Systemic and orbital reticulum cell sarcomas have been shown to be derived from lymphocytes, not reticulum cells or histiocytes, by immunologic and cytochemical characterization. Unfortunately, until now it has not been technically possible to study intraocular reticulum cell sarcomas. The authors have been able to immunologically characterize a primary intraocular reticulum cell sarcoma in a patient with chronic lymphocytic leukemia (e.g., Richter's syndrome), establishing its origin from B lymphocytes. A review of the clinical course of such tumors indicates that primary intraocular non-Hodgkin's lymphoma is usually a manifestation of a systemic neoplasm of the immune system. (2 figures, 34 references) — Authors' abstract

Australian Journal of Ophthalmology

MEASUREMENT OF DYNAMIC STEREOACUITY AND GLOBAL STEREOPSIS. Dunlop, D. B., Neill, R. A., and Dunlop, P. (66 King Street, Newcastle, Australia). Aust. J. Ophthalmol. 8:35, 1980.

A new test uses dynamic random-dot test patterns generated by inexpensive computer-controlled integrated electronics. Global stereopsis is delineated instead of central stereopsis. The speed of stereopsis is also measured so that both temporal and spatial aspects can be assessed. It is capable of providing a new type of grading for stereoscopic deficiencies. (19 figures, 38 references)—Ronald Lowe

British Journal of Psychiatry

COMPLEX VISUAL HALLUCINATIONS IN PARTIAL BLINDNESS DUE TO EYE DISEASE. White, N. J. (Nuffield Dept. Clin. Med., John Radcliffe Hosp., Headington, Oxford). Br. J. Psychiatry 136:284, 1980.

Visual hallucinations related to primary eye disease are usually unformed and consist of simple flashes of light or zigzag lines, whereas more complex visual hallucinations are associated with diseases of the cerebral peduncles or the cerebral hemispheres. However, the three patients described here experienced complex, formed hallucinations during progressive visual failure from eye disease. The hallucinations began abruptly, were brightly colored stereotyped figures, animals, or objects, and appeared to be provoked by light. As blindness progressed, the clarity, frequency, and duration of the hallucinations faded. The patients had no abnormalities other than their eye diseases, which in two cases was macula degeneration and in the third choroideremia. (12 references) —David Shoch

Cancer Treatment Report

OCULAR ASSESSMENT OF PATIENTS TREATED WITH TAMOXIFEN. Beck, M., and Mills, P. V. (Dept. Ophthalmol.,

University Hosp. of Wales, Cardiff). Cancer Treat. Rep. 63:1833, 1979.

Tamoxifen is a specific estrogen antagonist used in the treatment of breast cancer. In a previous study, corneal and retinal changes were reported in four patients receiving high-dose tamoxifen therapy for less than one year. In this study, 19 patients treated with tamoxifen for periods of three months to four years at normal dose levels were studied and no ocular changes attributable to the drug were observed. The patients in this study all had histologically proved breast carcinoma with signs of recurrence and all were treated with tamoxifen in doses not exceeding 20 mg twice daily for periods of between three months and four years. The patients in the previous paper in whom ocular toxicity was reported received 120 to 160 mg twice daily for over a year. Therefore, the ocular toxicity appears to be directly related to the dosage level and patients on high doses of tamoxifen must have regular ocular examinations. (1 table, 7 references)—David Shoch

Diabetologia

PLATELET SHAPE CHANGE ABNORMALITIES IN DIABETIC RETINOPATHY. Porta, M., Hilgard, P., and Kohner, E. M. (Depts. Med. and Haematol., Hammersmith Hosp., London). Diabetologia 18:217, 1980.

There have been numerous reports regarding changes in the platelet aggregation in diabetics. This appears to occur more commonly in those diabetics with advanced proliferative retinopathy. However, not all authors have been able to confirm this finding. The present article evaluates an early change in platelet aggregation, that is, a change in shape. This shape change occurs in the early phase of platelet aggregation and is related to energy-requiring mechanisms. The authors studied the in vitro platelet aggregation in 29 normal subjects and 35 diabetic patients with retinopathy and evaluated both conventional aggregation and platelet shape change. There was significant differences between the controls and the diabetics and between the controls and the subgroup of patients with proliferative retinopathy as regards the platelet shape change. Aggregation itself did not show a significant difference between normal and diabetic subjects. No conclusion can be drawn as to whether the platelet shape change is causative in the production of proliferative retinopathy or whether it is simply a reflection of the progression of the disease. (3 figures, 3 tables, 16 references)—David Shoch

International Journal of Radiation Oncology•Biology•Physics

THE RADIOTHERAPEUTIC MANAGEMENT OF OPTIC NERVE GLIOMAS IN CHILDREN. Danoff, B. F., Kramer, S., and Thompson, B. (Thomas Jefferson Univ. Hosp., Philadelphia, Pa.). Int. J. Radiation Oncology Biol. Phys. 6:45, 1980.

Optic nerve gliomas represent one to five percent of all intracranial tumors in children. The management of these tumors remains controversial. From 1956 to 1977, 18 children with optic nerve gliomas were treated by the authors using external beam radiotherapy. All children presented with decreased visual acuity and five of eighteen were blind in one eye. No patient was found to have involvement of a single optic nerve. In eight patients, the chiasm was involved, in ten patients, tumor had extended to the frontal lobes and/or hypothalamus. Initial surgical management included biopsy only in seven patients, inspection of tumor in two patients and partial excision in seven patients. Two patients were treated with radiotherapy based on radiological findings. A tumor dose of 5000-6000 rad was given in 5.5–6.5 weeks. Stabilization of visual impairment or improvement in vision was noted in 78 percent of patients who were evaluable. The ten year survival was 73 percent. The authors state that radiotherapy is indicated in the treatment of children with optic nerve gliomas who have poor prognostic signs (e.g., chiasmal and/or hypothalamic involvement and progressive visual loss). (2 figures, 8 tables, 27 references) —Authors' abstract

TREATMENT OF ORBITAL PSEUDOTUMOR

(IDIOPATHIC ORBITAL INFLAMMATION) BY RADIATION THERAPY. Donaldson, S. S., McDougall, R., Egbert, P. R., Enzmann, D. R., and Kriss, J. P. (Stanford Univ. School of Med., Stanford, Calif.). Int. J. Radiation Oncology Biol. Phys. 6:79, 1980.

This report describes experience with orbital radiotherapy in the treatment of five patients with orbital pseudotumor, three with bilateral involvement, two with unilateral lesions. Four patients with computed tomography scans had characteristic findings of pseudotumor; four had the diagnosis confirmed by biopsy. There was no evidence of an alternative diagnosis or systemic disease in any of the patients. Therapy consisted of 2000 rad of fractionated radiotherapy over ten days with a 4 MeV linear accelerator. Each patient experienced dramatic and rapid improvement in symptoms and signs. Exophthalmometric measurements improved by eight, eight, six and five millimeters respectively in those with proptosis. There has been no recurrence after a mean follow-up of 30 months and no complications of treatment. Orbital radiotherapy may be a preferable alternative to surgical or corticosteroid treatment for pseudotumor. Because of the variability in presentation, radiotherapy must be individualized so that orbital lesions are treated effectively while adjacent normal structures are shielded. (8 figures, 1 table, 15 references)—Authors' abstract

Laryngoscope

PALLIATIVE ORBITAL DECOMPRESSION FOR METASTATIC MELANOMA TO THE ORBIT. Sobol, S., Druck, N. S., and Wolf, M. (Dept. Otolaryngol., Washington Univ. School of Med., St. Louis, Mo.). Laryngoscope 90:329, 1980.

Although relatively rare, metastatic carcinomas to the orbit are the second most common orbital neoplasm in the adult. There are only ten cases reported in the literature of cutaneous melanoma metastatic to the orbit and, of these, only two had disease involving both orbits. This paper presents the third case of bilateral orbital melanoma; however, in this case, the primary site was the uveal tract in the first eye, with contralateral involvement being a metastasis of the orbital primary. The patient developed severe unilateral proptosis in his only seeing eye with progressive loss of vision. A palliative orbital decompression was performed which successfully restored visual acuity for an extended period of time. (2 figures, 9 references) — Authors' abstract

Medical Journal of Australia

PREVALENCE AND CAUSES OF BLINDNESS IN AUSTRALIAN ABORIGINES. Taylor, H. R. (Wilmer Ophthalmol. Inst., Johns Hopkins Hosp., Baltimore, Md.). Med. J. Aust. 1:71, 1980.

A descriptive epidemiological survey of monocular and binocular blindness in Australian Aborigines was undertaken. A case-control study was also performed using age and sex matched sighted controls to allow investigation of personal and environmental factors which may contribute to blindness. The prevalence rate of binocular blindness in the Australian Aboriginal males and females was 11/1000; and the monocular blindness rates were 24/1000 for males and 18/1000 for females. The prevalence of blindness was strongly age-related; over the age of 60 years, 21.5% of males and 17.5% of females were binocularly blind, and 25.5% of males and 18.5% of females were monocularly blind. Half of the blindness was caused by cataracts, one-third by corneal disease, and one-twelfth by the loss of the globe. Trachoma and trauma were positively associated with blindness. Employment seemed to reduce the likelihood of blindness, with the exception of the Aboriginal males who had been employed as stockmen and who had a high rate of blindness. Less than 10% of blindness was attributable to degenerative or retinal disorders. (9 tables, 34 references)—Author's abstract

Ophthalmology

UNILATERAL FOUR-MUSCLE SURGERY FOR LARGE-ANGLE EXOTROPIA. Raab, E. L. (Dept. Ophthalmol., Mount

Sinai School of Med., City Univ. Of New York, N.Y.). Ophthalmol. 86: 1441, 1979.

Eight patients with large-angle exotropia and unimprovable poor vision in one eye were treated with conventional maximum horizontal rectus recession/resection plus weakening of both obliques of the poorly seeing eye. This appears to be a useful alternative to "supermaximal" recession/resection procedures, which are more likely to lead to deficient or restricted rotations. The effect is probably due to the release of additional sites of contracture that occur in the oblique muscles. The principal disadvantage is a small postsurgical hypertropia of the operated eye, for which routine infraplacement of both horizontal recti can compensate adequately. (4 figures, 1 table, 10 references) — Author's abstract

CLINICAL OBSERVATIONS IN CYCLODEVIATIONS. von Noorden, G. K. (Cullen Eye Inst., Baylor College of Med., and Dept. Ophthalmol., Texas Children's Hosp., Houston, Tex.). Ophthalmol. 86:1451, 1979.

Cyclodeviations differ from other strabismus forms in several aspects. Adaptive mechanisms other than suppression cause the patient to remain asymptomatic in spite of ophthalmoscopically demonstrable torsion of the globe around the sagittal axis. These mechanisms include cyclofusion and a monocular sensory adaptation causing the images seen by the paretic eye to appear vertically and horizontally aligned even though they do not fall on the vertical and horizontal anatomic-geometric retinal meridians. Pure excyclotropias, e.g., those not associated with vertical deviations, respond well to anterior and lateral displacement of the anterior portion of the superior oblique tendon. The operation eliminated the cyclodeviation in four eyes (three patients); however, a partial recurrence was observed in one case. (5 figures, 1 table, 24 references)—Author's abstract

SUCTION OPHTHALMODYNAMOMETRY IN THE DIAGNOSIS OF CAROTID STENOSIS. Zaret, C. R., Sacks, J. G., and Holm, P. W. (Dept. Ophthalmol., Northwestern University, Chicago, Il.). Ophthalmol. 86:1510, 1979.

In a prospective evaluation of 39 patients who had arteriography for suspected carotid stenosis, suction ophthalmodynamometry correctly identified 86% of the patients who had radiographically demonstrable unilateral or asymmetric obstructions. This compares favorably with carotid compression tonography, oculoplethysmography, and Doppler flow studies. The false-positive rate was 27%, a figure higher than the latter two noninvasive tests. (2 tables, 11 references)—Authors' abstract

CORNEAL EDEMA IN ESSENTIAL IRIS ATROPHY. Shields, M. B., McCracken, J. S., Klintworth, G. K., and Campbell, D. G. (Duke Univ. Eye Ctr., Durham, N.C.). Ophthalmol. 86:1533, 1979.

In a clinical study of the essential iris atrophies, corneal edema occurred in 49 of 90 patients, and a characteristic hammered-silver appearance of the posterior cornea was visible in 54 cases. Intraocular pressure in the eyes with corneal edema ranged from normal to extreme elevation with glaucomatous damage, and lowering the pressure relieved the corneal edema in many cases with penetrating keratoplasty being effective when other measures failed. Electron microscopy of two keratoplasty specimens revealed extremely few, distinctly abnormal cells lining a collagenous layer posterior to Descemet's membrane. (9 figures, 5 tables, 20 references) — Authors' abstract

Transactions of the American Ophthalmological Society

PREMATURE CATARACTS ASSOCIATED WITH GENERALIZED LENTIGO. Howard, R. O. (Dept. Ophthalmol., Yale Univ. School of Med., New Haven, Conn.). Trans. Am. Ophthalmol. Soc. 77:121, 1979.

Generalized lentigo (leopard syndrome) is an

autosomal dominant trait characterized by lentigo, sensorineural deafness, retarded growth (below 25%), ocular hypertelorism, mandibular prognathism, pectus carinatum or excavatum, dorsal kyphosis, winging of the scapulae, valvular pulmonary artery stenosis, electrocardiographic conduction defects, and genitourinary defects. Ocular evaluations of patients with generalized lentigo have revealed the appearance of multiple small white punctate and comma-shaped opacities in the cortex and nuclei of the lenses of affected patients. On the basis of age of the patients examined, it would seem that the corneal opacities first appear in the third decade. Although the opacities may be extensive, the lens opacities do not appear to impair visual function until approximately twenty years after they first appear. (5 figures, 23 references)—Author's abstract

PROGNOSIS FOR LIFE IN PATIENTS WITH DIABETES: RELATION TO SEVERITY OF RETINOPATHY. Davis, M. D., Hiller, R., Magli, Y. L., Podgor, M. J., Ederer, F., Harris, W. A., Long, J. W., and Haug, G. A. (Dept. Ophthalmol., Univ. Wisconsin, Madison, Wisc.). Trans. Am. Ophthalmol. Soc. 77:144, 1979.

In a group of 709 individuals with diabetes diagnosed prior to age 50 and followed for five to 13 years a strong inverse relationship was demonstrated between the severity of the retinopathy at the initial visit and survival. Survival in patients with no retinopathy or with microaneurysms only was a little different from that of the general population (five-year rate 99%). The five-year survival rate for patients with more severe nonproliferative retinopathy, characterized by the presence of hemorrhages and/or exudates, but without new vessels or vitreous hemorrhage (Class B_2) was 81% and that for patients with proliferative retinopathy was 56%. After adjustment for age at diagnosis of diabetes, duration of diabetes and sex, the differences in survival between these three groups were highly statistically significant. Impairment of visual acuity was also shown to be inversely related to survival. The five-year survival rate for patients with visual acuity of 20/200 (6/60) or worse in each eye was 42%. (4 figures, 8 tables, 18 references) — Authors' abstract

UVEITIS ASSOCIATED WITH SARCOIDOSIS AND ANGIOTENSIN CONVERTING ENZYME. Weinreb, R. N., and Kimura, S. J. (Clin. Res. Ctr. for Eye Disease, Dept. Ophthalmol., and Francis Proctor Found., Univ. California, San Francisco, Calif.). Trans. Am. Ophthalmol. Soc. 77:280, 1979.

Sarcoid uveitis is usually a presumptive diagnosis based on the simultaneous presence of uveitis and clinicoradiographic or histologic findings of sarcoidosis. The elevation of serum angiotensin converting enzyme in some patients with granulomatous uveitis is strongly presumptive of sarcoid uveitis even in the absence of these findings. Serum angiotensin converting enzyme may prove to be a useful indicator of ocular sarcoid in the absence of otherwise undetectable systemic disease. (2 figures, 2 tables, 14 references)—Authors' abstract

THE ASSOCIATION OF VIRUSES WITH UVEAL MELANOMA. (Thesis) Albert, D. M. (Dept. Ophthalmol., Harvard Med. School, Massachusetts Eye and Ear Infirm., Boston, Mass.). Trans. Am. Ophthalmol. Soc. 77:367, 1979.

Electron microscopic examination of 57 ocular melanomas (54 human, two feline and one canine) revealed the presence of viral particles in six specimens. Herpesvirus particles were observed in one human specimen and were passed in human fibroblasts (WI-38), where they gave rise to intranuclear inclusions. A-type oncornavirus particles (oncogenic RNA virus) were observed in a second case, both in cells of tumor directly removed from an enucleated eye as well as in cells grown in tissue culture. In three human specimens and one feline specimen, togavirus particles were observed. Rubella is a member of this group, and the possibility that the presence of togavirus in these tumors is the result of latent ocular infection by rubella virus is raised. Herpes virus and RNA tumor viruses are widely considered as having a possible etiologic role for certain human cancers. The observation of togavirus is unexpected, as this virus has not been previously implicated in human or animal tumors.

Injection of an RNA tumor virus (Gardner strain feline sarcoma virus) into the anterior chamber of newborn kittens resulted in the development of iris and ciliary body melanomas, many of which showed invasion and, in one instance, metastasis. This is the first animal model of a viral-induced uveal melanoma, and the histology and ultrastructure are described. These results emphasize the need for the continued investigation of the role of these viruses in uveal melanoma. (23 figures, 255 references) — Author's abstract

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EDITED BY THOMAS CHALKLEY, M.D.

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For adequate publicity, notices of postgraduate courses, meetings, and lectures must be received at least four months before the date of occurrence.

XIII PAN AMERICAN CONGRESS OF OPHTHALMOLOGY

The XIII Pan American Congress of Ophthalmology will be held May 10-15, 1981, in Acapulco, Mexico. For further information, write XIII Pan American Congress of Ophthalmology, Apartado Postal No. 11-566, Mexico 11, D. F. Mexico.

FIRST INTERNATIONAL INTRAOCULAR LENS SEMINAR

The First International Intraocular Lens Seminar will be held Aug. 28 to Sept. 1, 1980, in Southampton, Bermuda. The registration fee is $275. For further information, write Mrs. Verna Lloyd, 7800 York Rd., Baltimore, MD 202014.

SYMPOSIUM ON HUMAN HERPES SIMPLEX INFECTIONS

A symposium on human herpes simplex infections and their therapy will be held in Brest, France, March 27 and 28, 1981. For further information, write Dr. Colin, Laboratoire de Virologie, Faculté de Médecine, B. P. 815, 29279 Brest, Cedex, France.

UNIVERSITY OF MICHIGAN MEDICAL SCHOOL: OCULO-PLASTIC SURGERY SYMPOSIUM

The University of Michigan Medical School will sponsor an Oculo-Plastic Surgery Symposium, Aug. 14 and 15, 1980. The fee is $200 for practicing physicians, and $125 for physicians-in-training. For further information, write Floyd C. Pennington, Director, Office of Continuing Medical Education, The Towsley Center, University of Michigan Medical School, Ann Arbor, MI 48109.

AMERICAN COLLEGE OF VETERINARY OPHTHALMOLOGISTS: ANNUAL MEETING

The annual meeting of the American College of Veterinary Ophthalmologists will be held Nov. 1-3, 1980, at the Radisson Hotel, Chicago, Illinois. The program will include an Immunology Workshop Nov. 1; and a Scientific Program Nov. 2 and 3. Individuals who wish to present a paper should submit an abstract to Charles Martin at the College of Veterinary Medicine, University of Georgia, Athens, GA 30602. There will be a special program for residents on Nov. 2. Residents who wish to present a paper should submit an abstract of the paper to Allen Brightman at the University of Illinois Veterinary College, Urbana, IL 61801. There will be no charge for attendance for Diplomates of the ACVO; nonmembers, $100; residents presenting papers, no charge; residents not presenting a paper, $35.

UNIVERSITY OF MIAMI SCHOOL OF MEDICINE: 1980 NEURO-OPHTHALMOLOGY COURSE

The University of Miami School of Medicine will hold the 1980 Neuro-Ophthalmology Course Dec. 10-13, 1980, in Miami, Florida. Registration fee is $275 for practitioners and $150 for residents. For further information, write Continuing Education in Ophthalmology, Inc., P.O. Box 610326, Miami, FL 33161.

NEW YORK UNIVERSITY POST-GRADUATE MEDICAL SCHOOL: BASIC SCIENCES IN OPHTHALMOLOGY

The New York University Post-Graduate Medical School will hold a course, "Basic Sciences in Ophthalmology," Sept. 3 to Dec. 20, 1980, Monday through Saturday. The fee is $1,000. For further information, write NYU Post-Graduate Medical School, 550 First Ave., New York, NY 10016.

PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY: NEW OFFICERS

New officers elected to the Pennsylvania Academy of Ophthalmology and Otolaryngology at the Academy's 36th annual meeting are as follows: president; Jerome Dersh; president-elect, Donald B. Kamerer; first vice-president, William H. Annesley, Jr., second vice-president, John M. Penta; and third vice-president, John F. Connole. Reelected were the following: treasurer, William K. Grove; editor of Transactions, Thomas B. Souders; and secretary for instruction, Donald T. Burns.

Fight For Sight Award

Three National Institutes of Health scientists and a University of Pennsylvania veterinarian have been cited for outstanding achievement in basic research by Fight for Sight, Inc. The award-winning study of enzyme activity in the eyes of certain dogs with a hereditary blinding disorder may provide clues to the development of a treatment for early-onset retinitis pigmentosa in humans. The award cites Gerald J. Chader and Leo Liu of the Laboratory of Vision Research, National Eye Institute; Gopal Krishna of the Laboratory of Chemical Pharmacology, National Heart, Lung and Blood Institute; and Gustave Aguirre of the University of Pennsylvania School of Veterinary Medicine, for their research on "Cyclic GMP phosphodiesterase activator. Involvement in a hereditary retinal degeneration."

The four investigators received embossed scrolls and will share $500 awarded by Fight for Sight, Inc., in cooperation with the Association for Research in Vision and Ophthalmology. The award was presented at the Association's 1980 meeting in Orlando, Florida, May 4-9.

National Advisory Eye Council: New Members

Richard N. Lolley, of Los Angeles, California, and Thomas J. Tredici, of Brooks Air Force Base, Texas, have been appointed ex officio members of the National Advisory Eye Council, the principal advisory body to the National Eye Institute (NEI).

Dr. Lolley will represent the Veterans Administration at Council meetings and Dr. Tredici will be the Department of Defense liaison. Drs. Lolley and Tredici will advise the NEI director and staff on the awarding of grants for vision research training. They will also provide guidance on NEI policy development and program planning.

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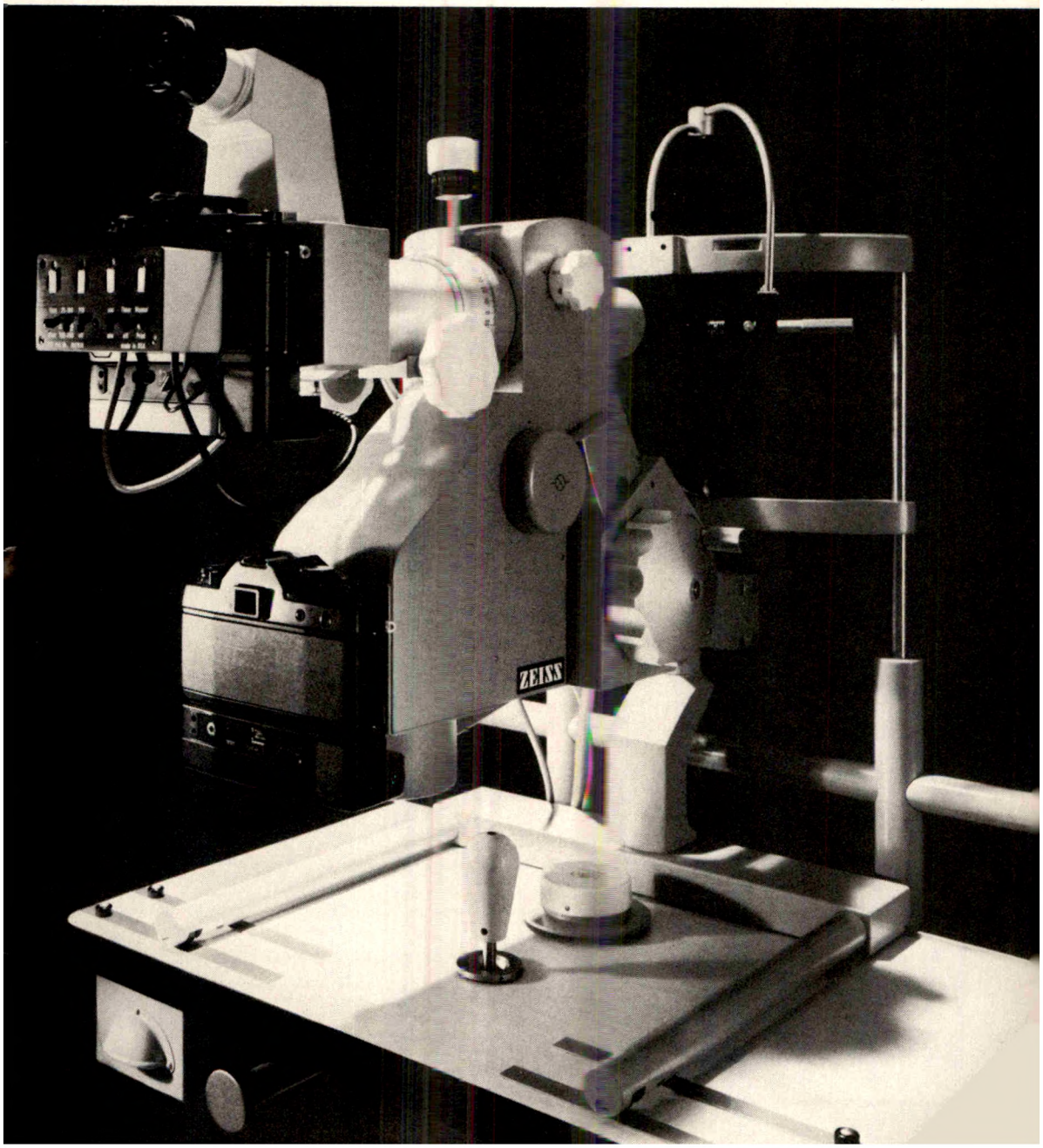
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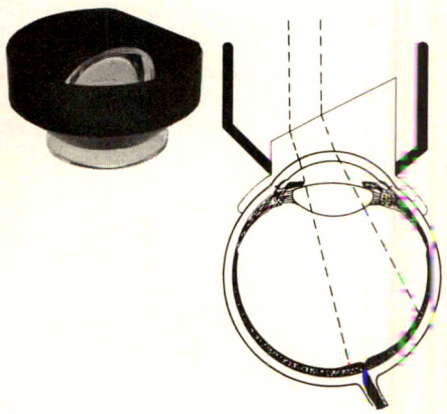
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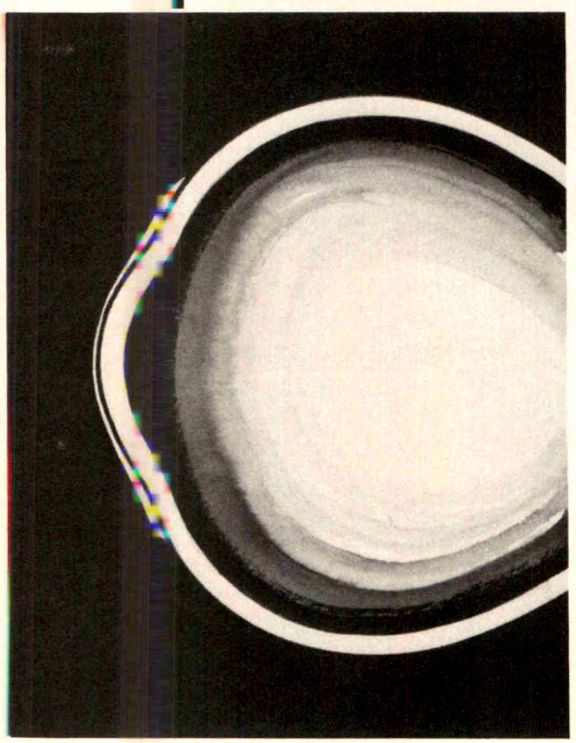
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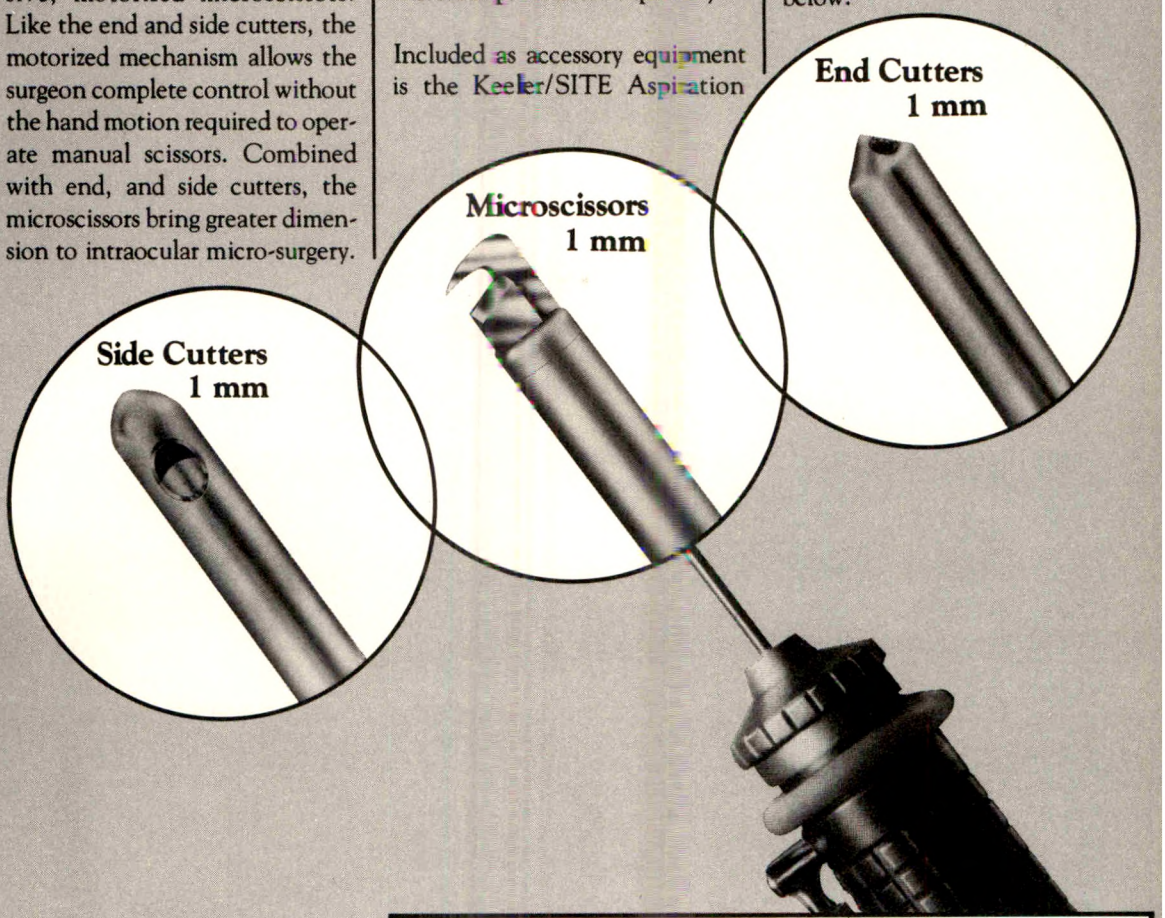
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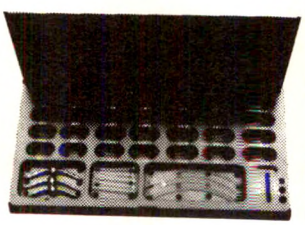
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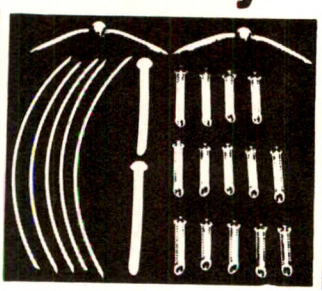
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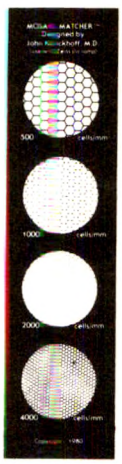
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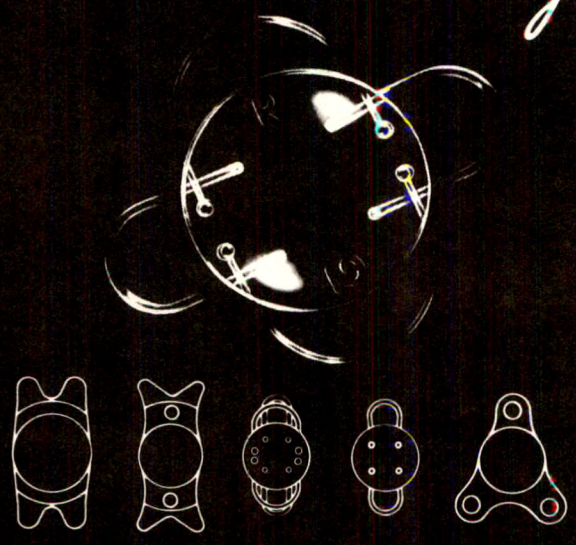
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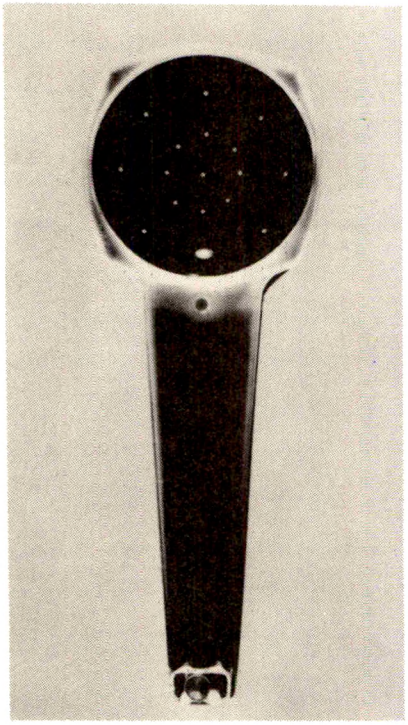
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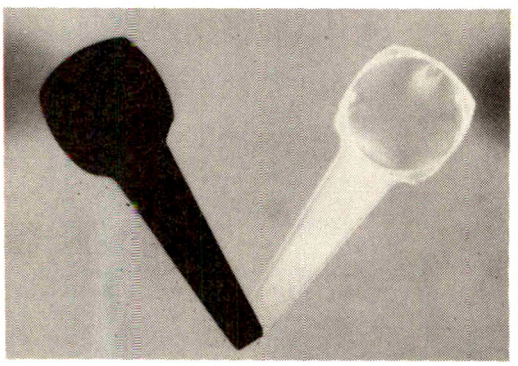
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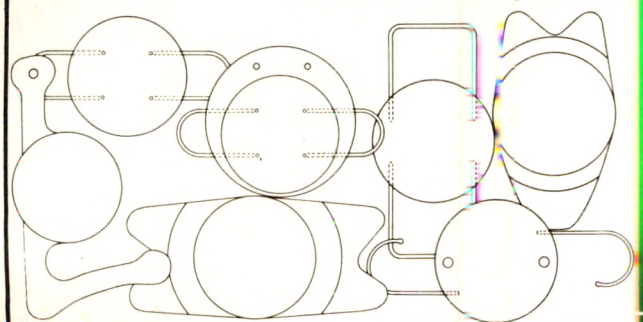
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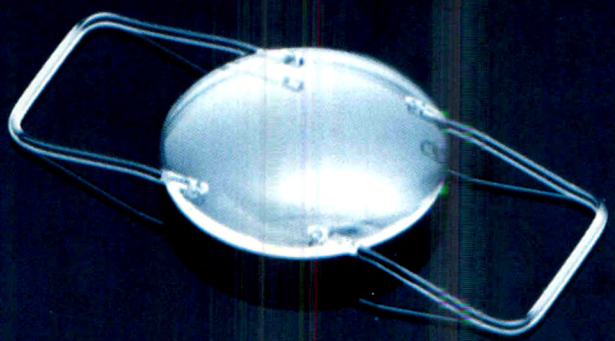
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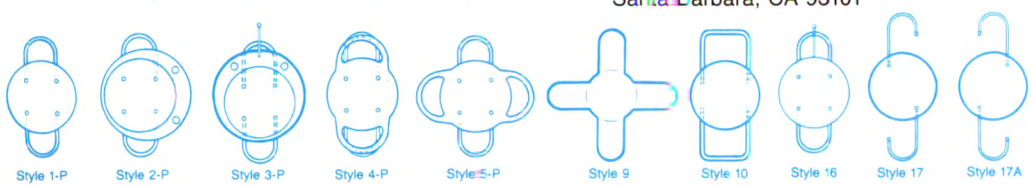
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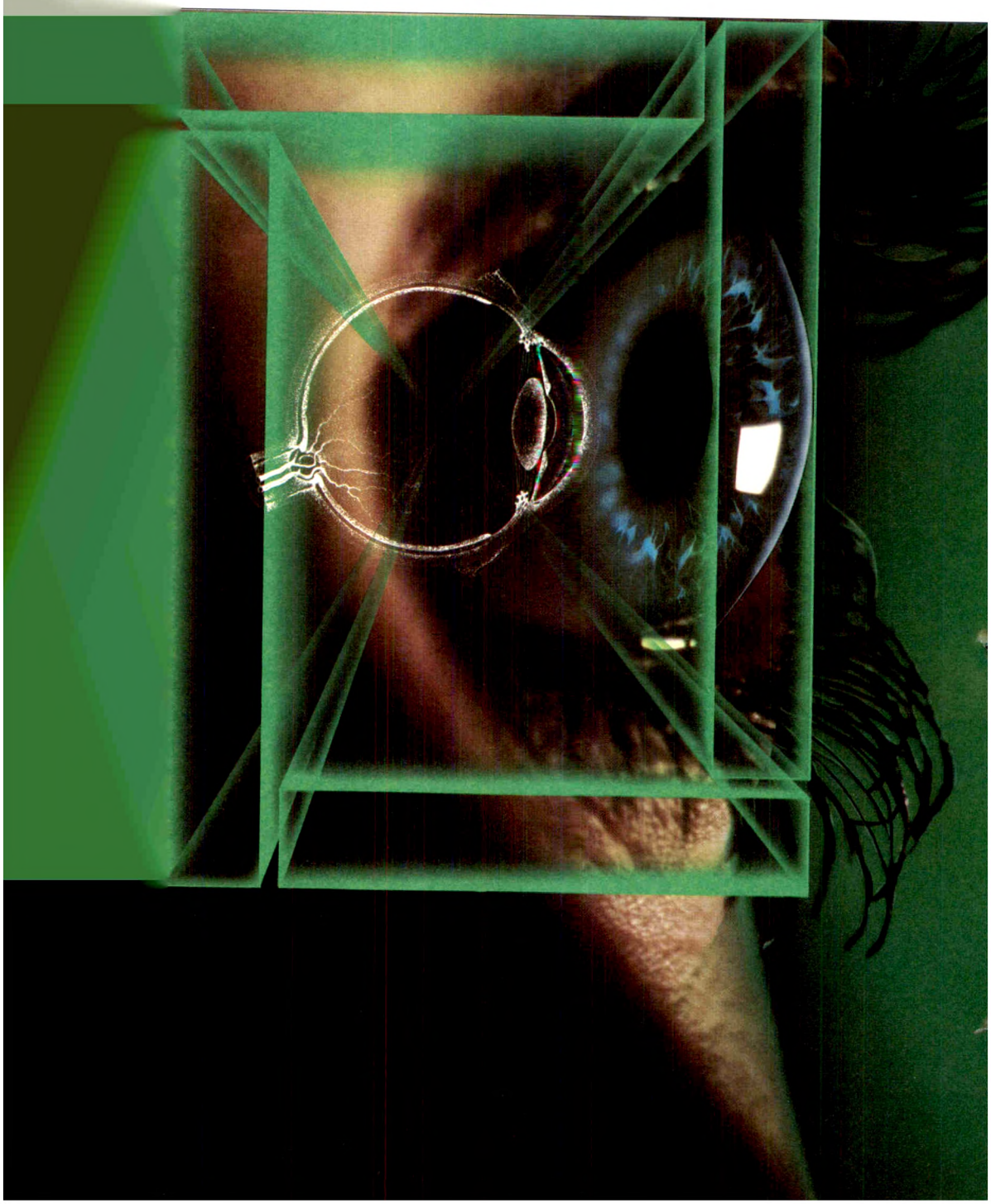
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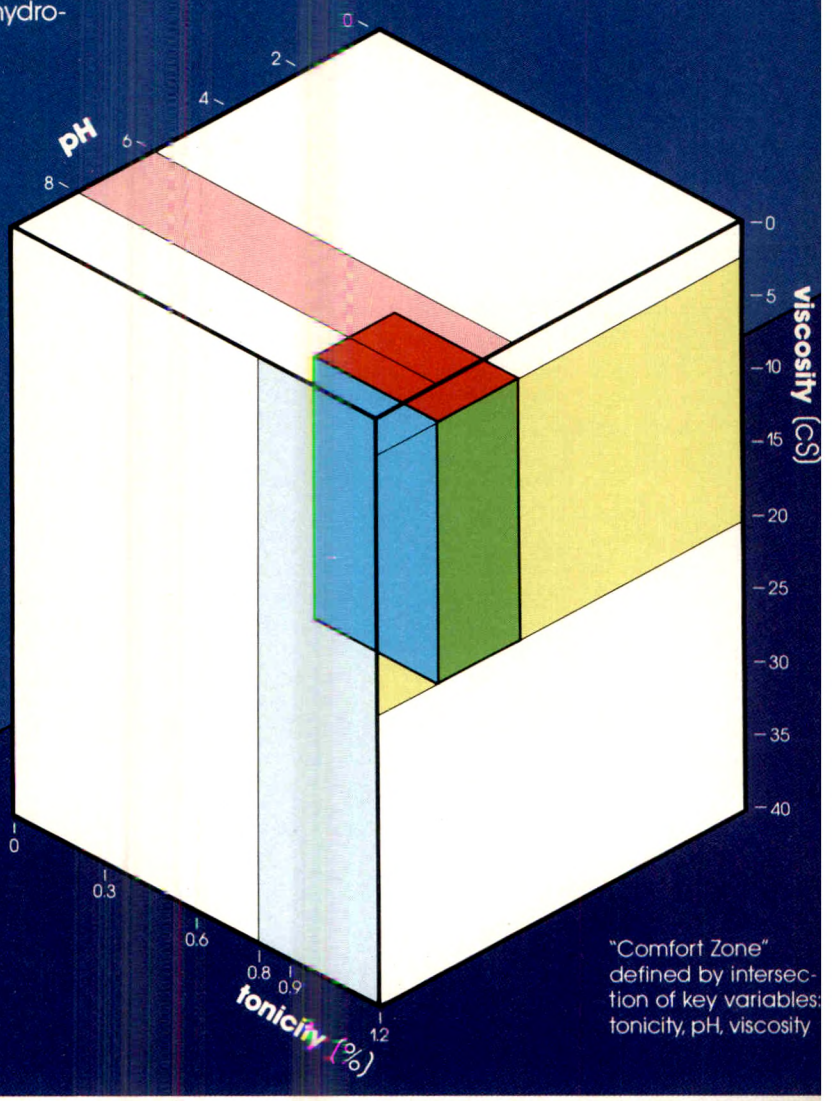
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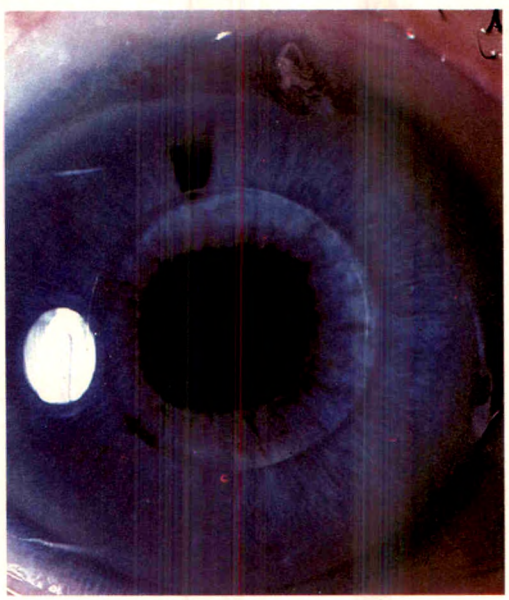
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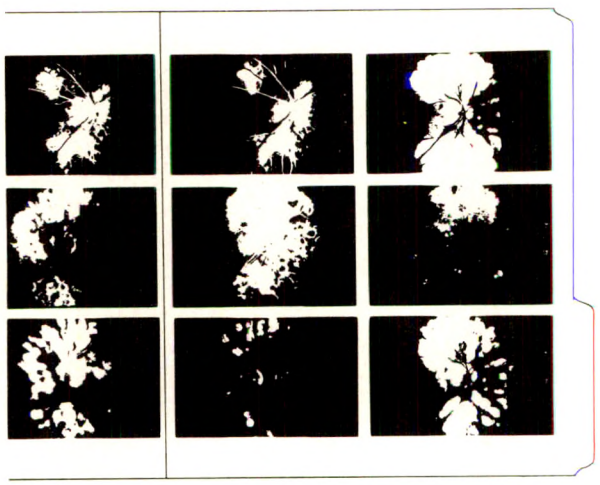
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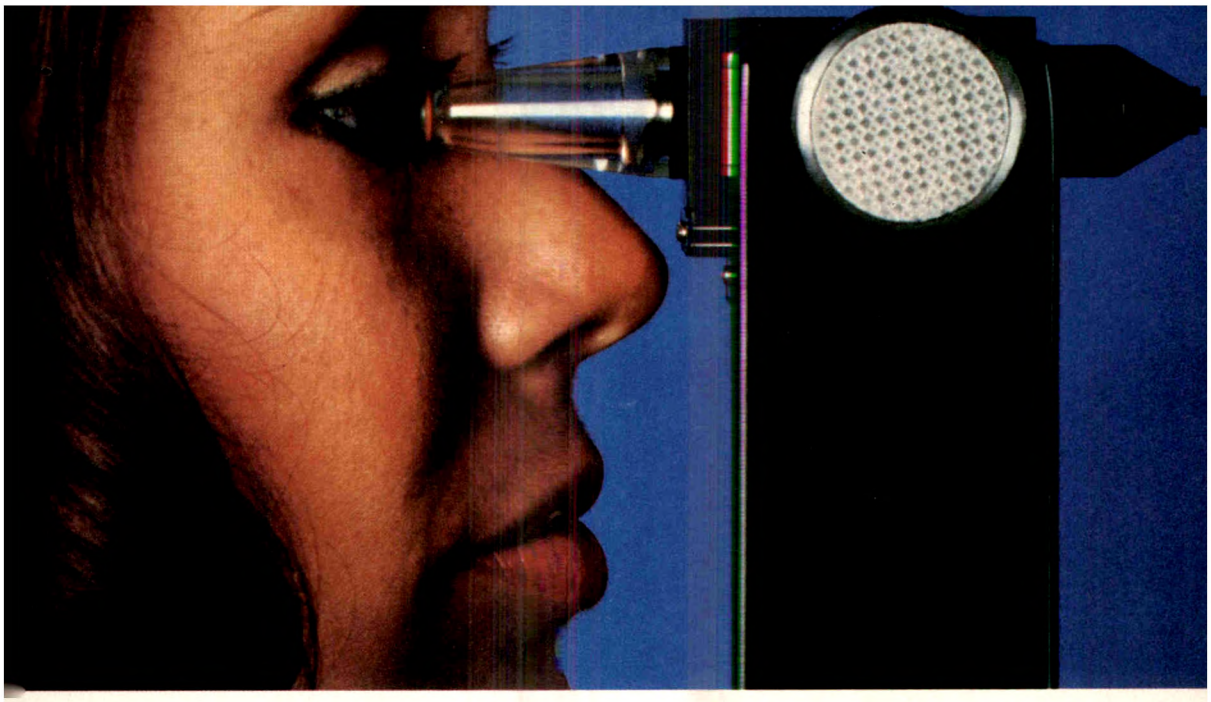
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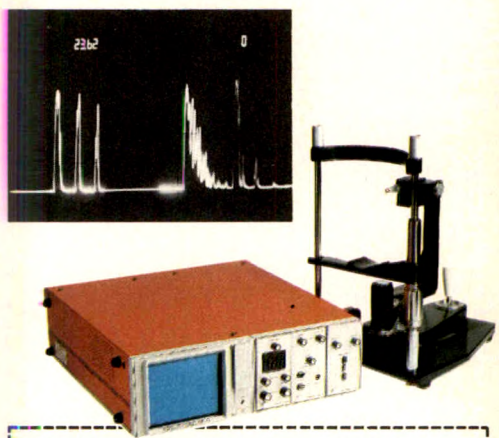
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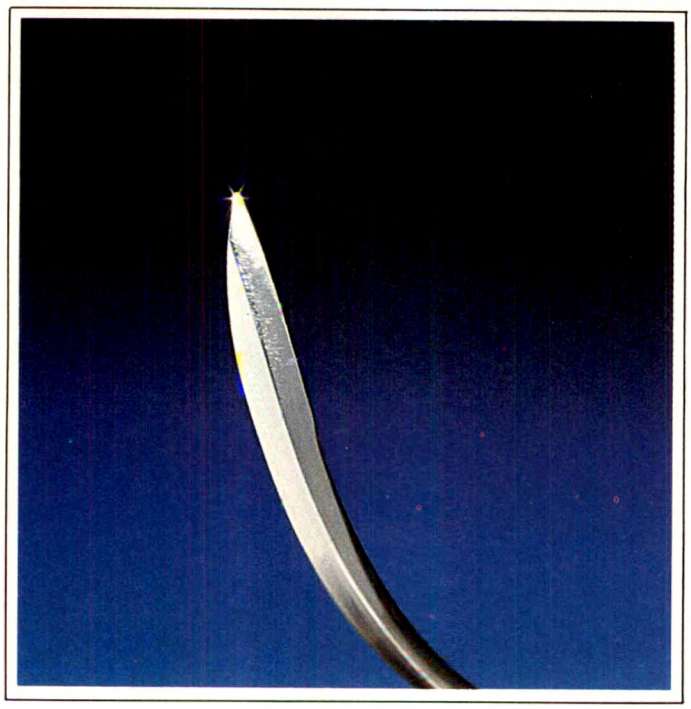


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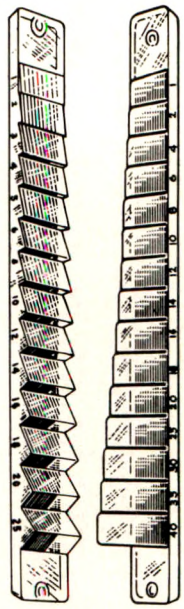
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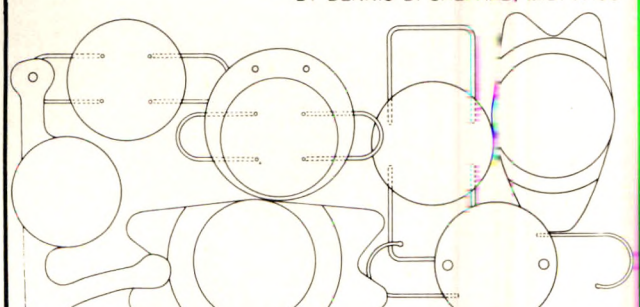


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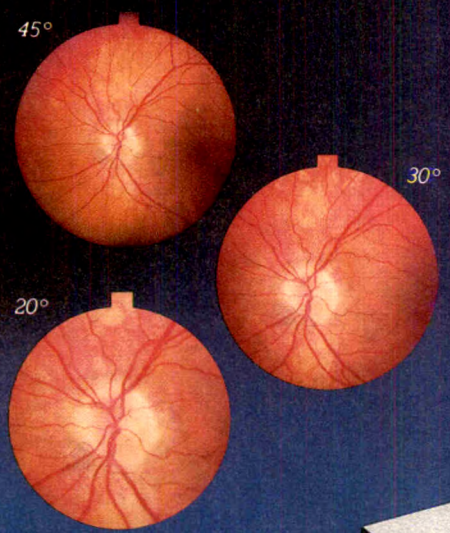
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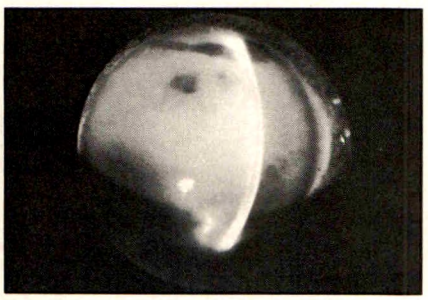


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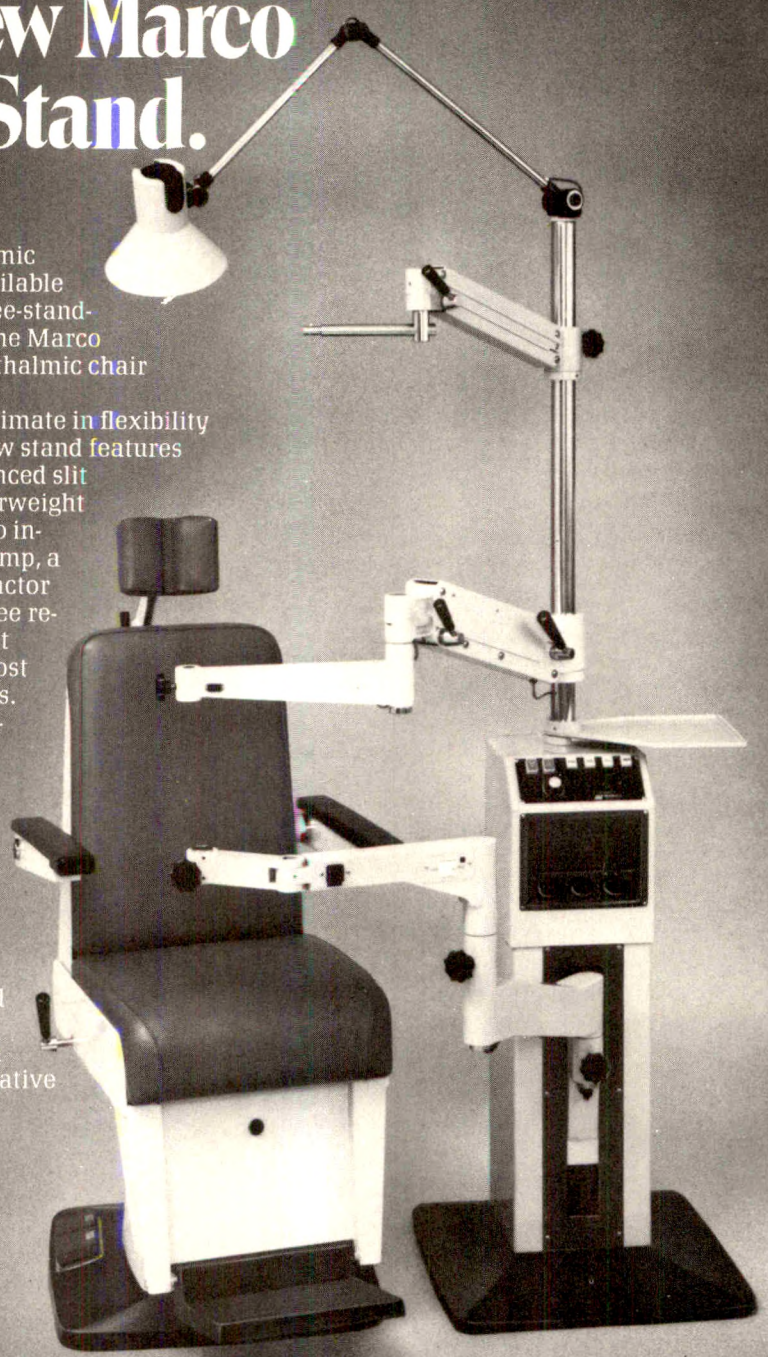
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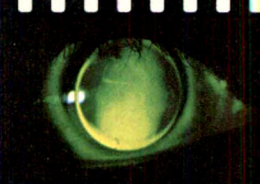
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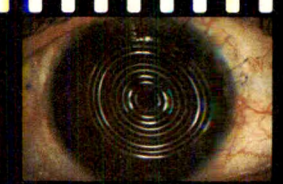
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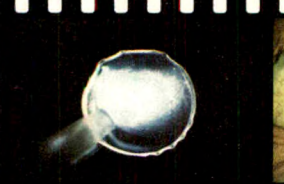
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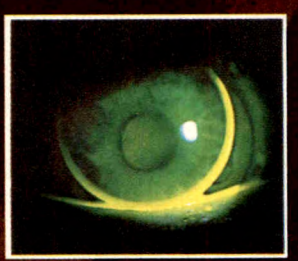
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WARNINGS
(1) If the lens becomes less comfortable to the wearer than when first placed on the wearer's cornea, a foreign body may be present. The patient should remove the lens immediately. If discomfort persists, an eye care practitioner should examine the patient as soon as possible to determine if any corneal abrasion, ulceration, irritation or infection is present.

(2) Aphakic patients should not be fitted during the postoperative period until the surgeon determines that the eye has healed completely.

(3) Only use Preflex® Sterile Cleaning Solution, Flex-Care® Sterile Solution, and Adapettes® Sterile Lubricating Solution with the POLYCON lens. Store ONLY in Flex-Care® Sterile Solution. Patients must adhere to recommended daily lens care regimen. FAILURE TO DO SO MAY RESULT IN DEVELOPMENT OF SERIOUS OCULAR INFECTIONS.

(4) Wearers must not use any medicaments or ophthalmic solutions other than Adapettes® solution prior to inserting the lens or while the lens is on the eye.

(5) Remove before sleeping or in the presence of noxious and irritating vapors.

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1-80

References

1 Multi-clinic study of 418 patients, data on file, Syntex Ophthalmics, Inc.

2 Korb JE, Finnemore VM: Corneal edema with polymethylmethacrylate vs. gas-permeable rigid polymer contact lenses of identical design. To be published.

3 Williams CE: New design concepts for permeable rigid contact lenses. *JAOA* 50:331-336, 1979.

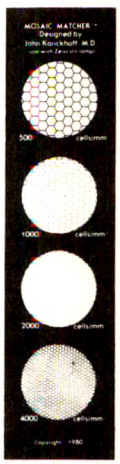
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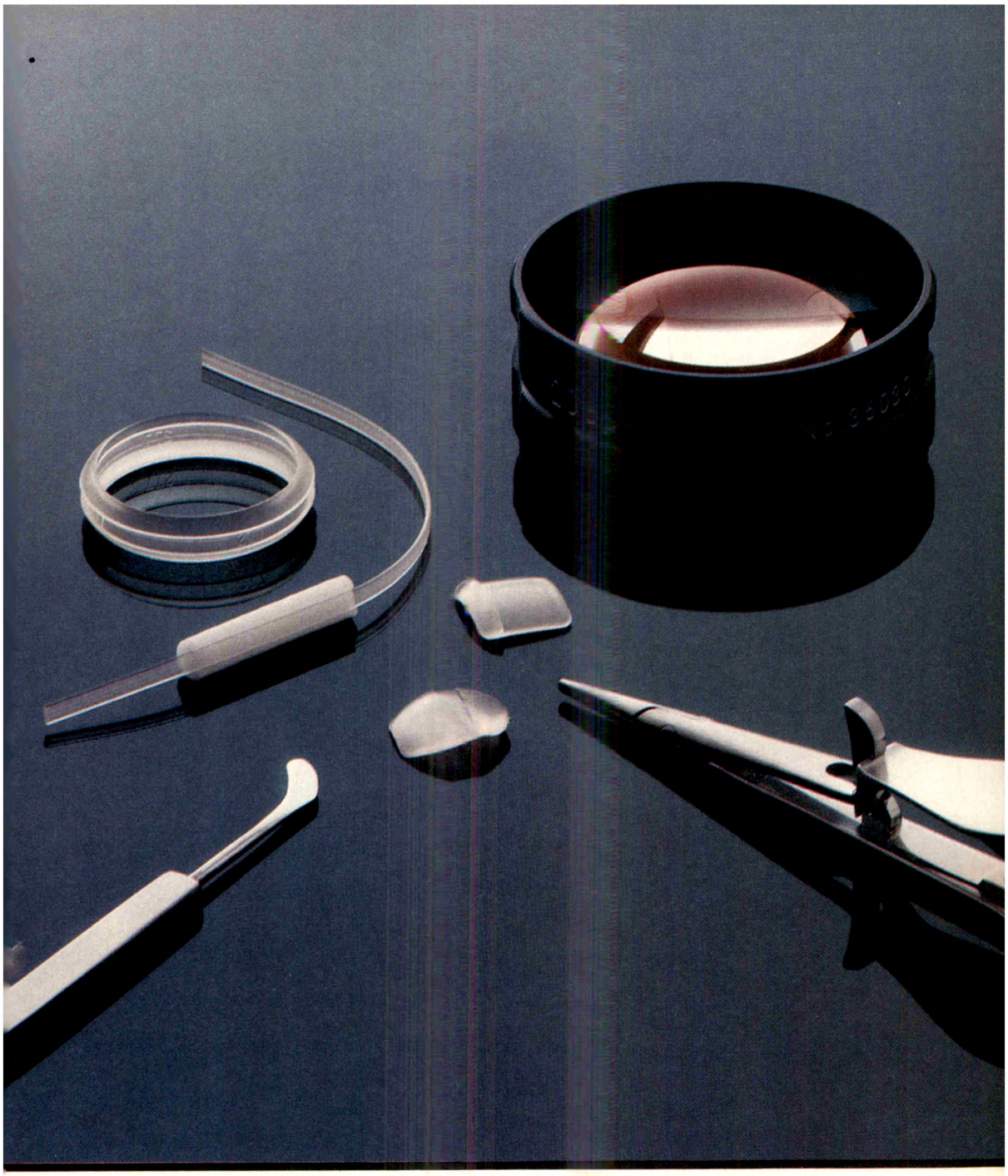
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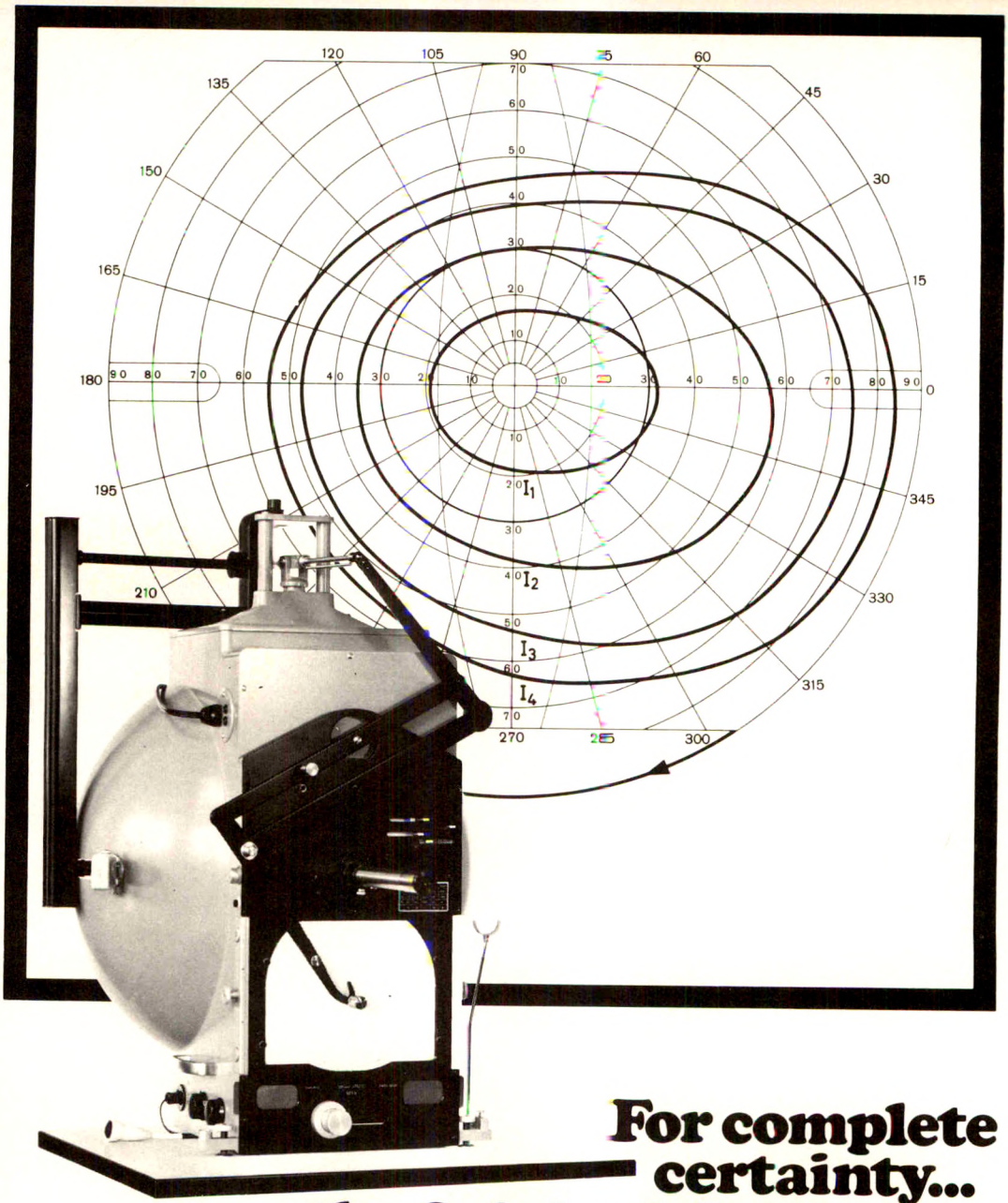
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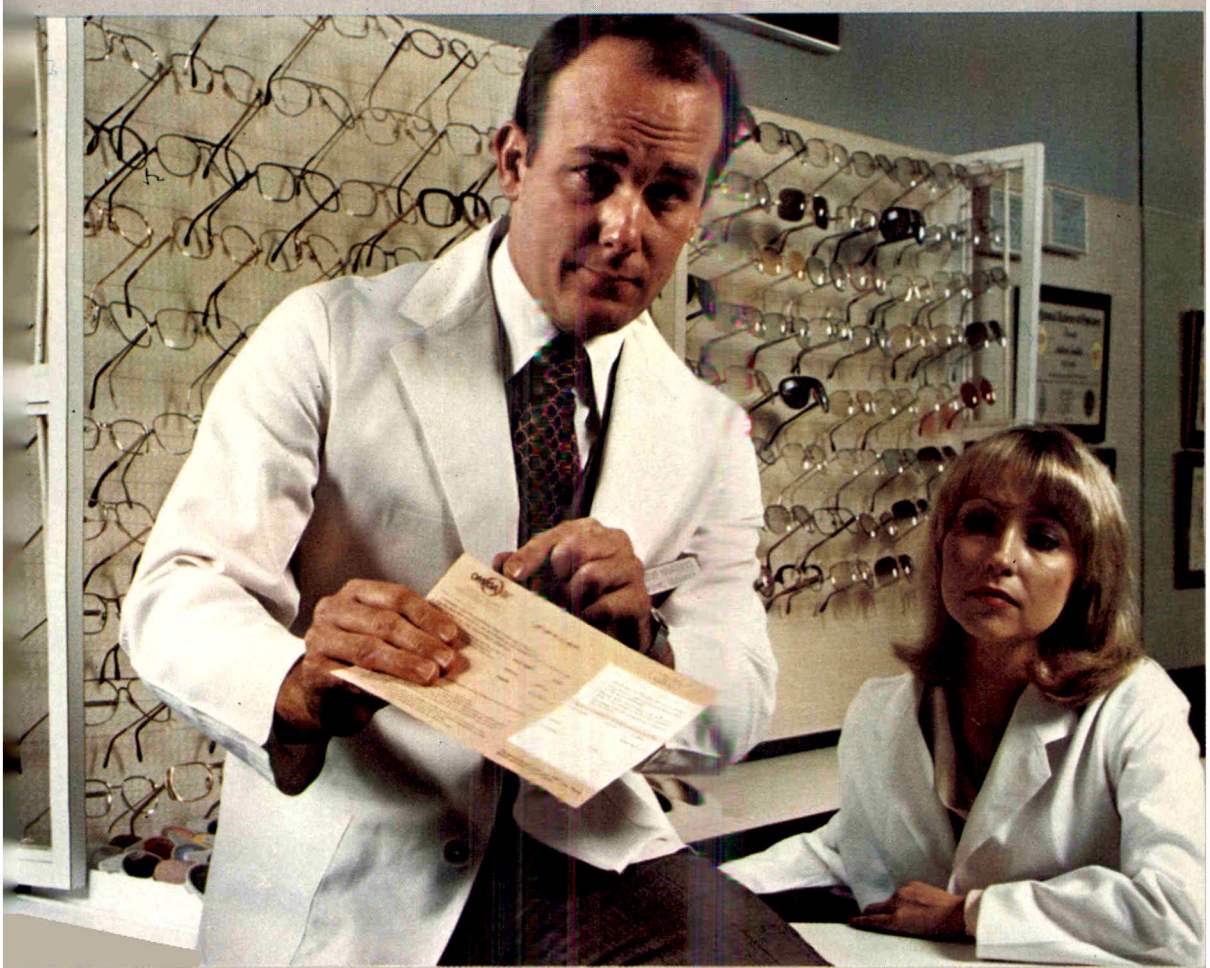
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2. Patient sends the application and premium payment directly to OCLICO. And OCLICO sends the policy directly to the patient.

That's all!

You have no records to file. No proof-of-loss forms, no complex forms of any kind to explain to your patients.

And taking care of a claim is a simple procedure, too. There's only one step!

You collect your own replacement fee. The "Total deductible" which covers your professional replacement fee and the insurance deductible is the only amount printed on the face of an OCLICO policy – and your patient pays the full amount to you. Later, your contact lens laboratory bills you only for the insurance deductible portion on their regular laboratory invoice.

All unnecessary paperwork is out. There's virtually no involvement on your part at all.

Plus, OCLICO covers all risks of loss or damage. For all kinds of contact lenses. Now, *that's* protection!

You can find out more about this beneficial program. Fill out the coupon and mail it to OCLICO.

In those states where Omega Contact Lens Insurance Company is not licensed, the plan is underwritten by Great American Insurance Company.

OMEGA CONTACT LENS INSURANCE COMPANY

P.O. Box 220006
Dallas, Texas 75222
(214) 241-7000

I'd like to keep things simple. Please send me more information on OCLICO. AJO 4/80

Name_____

Address _____

City _____ State_____ Zip_____

Telephone ( )_____

IT CAN MAKE YOUR

It's the new office computer from Acuity Systems.

And it's specifically designed to take a successful solo or group eye care practice like yours, and make it even more productive.

Because it speeds up paperwork in your front office, accelerates your cash flow, and gives you a more complete financial picture of your practice. It also lets you spot problems before they occur, so you can manage your practice better. And it shows you how to make your working day more efficient, so you can spend more time seeing patients.

What's more, it does all this without adding staff. And without adding space.

Think of your next patient as 15 pieces of paperwork.

You may not realize it, but every patient who enters your office creates as many as 15 separate pieces of paperwork.

And no matter how small your practice is, handling those ledger cards, bills, files, and insurance forms (to name only a few) takes thousands of staff hours a year, as well as your own time. Time you could be putting to better use.

But the Acuity Systems office computer drastically cuts the time spent on paperwork. Because it keeps complete patient accounts, types up insurance forms, and schedules appointments. It also produces itemized bills and daily bank deposit slips. And it performs a variety of other time-consuming daily record-keeping chores quickly and accurately.

It also functions as a word processor. In this mode, it can search your records for patients who need to be recalled, and then type labels for reminder postcards. Plus it can generate personalized form letters for insurance inquiries, referral replies, and other repetitive correspondence.

What's more, the Acuity Systems office computer stores all its files electronically on magnetic disks. This allows your staff instant access to records, speeding answers to patient inquiries, and improving patient relations.

You get a better cash flow, plus vital financial data.

Because Acuity Systems' office computer does everything with incredible speed, patients can receive bills on the spot, and insurance claims can be processed the same day.

And, it also keeps track of aging accounts for you. At the touch of a button, it can produce a list of past-due patients your staff can utilize for an on-going collection program.

As a result, collectibles are greatly reduced. So you'll have a steadier, more reliable cash flow that will allow you to manage your funds better.

In addition, the Acuity Systems office computer automatically prepares a variety of important financial reports, including trial balance, profit and loss statements, and balance sheets. These reduce your need for costly accounting services. And they give you a day-to-day understanding of your financial position you may never have had before.



The Acuity Systems' office computer, part of the Intelligent Office™ system of practice management. Shown are the display terminal with keyboard, high-speed printer, and disk drive. The disk drive can also be placed in a closet, allowing the terminal and printer to be placed on a desk top.

It puts your finger on the pulse of your practice.

In addition to performing all your daily bookkeeping and billing functions, Acuity Systems' office computer also compiles a variety of practice management reports, including:

- practice income by doctor, procedure, day, or any of a variety of other variables.
- revenue analysis, allowing you to monitor your sources of income.
- inventory control (for practitioners who dispense).

With these reports, you'll be able to monitor your own workload, or that of other members of your practice. You'll be able to see where your income comes from, and where you may be able to reduce your overhead. And you'll have the information you need to manage your practice effectively.

What's more, since the computer is completely contained in your office, only you have access to this confidential patient and practice information.

It speaks plain English, so it's easy to operate.

You might think that a computer system this sophisticated would

© 1980 Acuity Systems Incorporated.

PRACTICE PERFECT.

need its own programmer. But the Acuity Systems office computer doesn't, because it's preprogrammed. In fact, it's so easy to use that your secretary can be trained to run it in just a few hours. There's no special computer language to learn, because it actually prompts your secretary with simple, step-by-step instructions. Which means it's almost impossible to make a mistake.

What all this means is that, whether yours is a solo or a multi-doctor practice, the Acuity Systems office computer has the features and functions you need for eye care, programmed in the way that's most convenient to use them.

With Acuity Systems behind it, you can be sure it'll work.

And that's a feeling of security you won't get elsewhere.

Because most other "computer companies" sell you the computer and the program, and then kiss you goodbye. They don't help you through the transition period, when the computer is new. While some of them do train your employees, they make no provision for training new employees later on. None of them follow up to make sure the system's working right. And few of them actually service both the hardware and software.

But at Acuity Systems, we give you all the services the other companies don't. Like complete planning for the transition to computerization by our Practice Management Analyst. Complete training, plus follow-up and new employee training by our Customer Service representatives. And 24-hour service of any problem by our own nationwide service network.

Why do we do all this for you? Because in the past 10 years, we've acquired an excellent reputation from our other products, the Auto-Refractor® and the Auto-Lensmeter™. And making sure our new office computer works perfectly in every practice is the only way we're going to keep that reputation.

Perfect the way you practice.

Send for our free booklet, "The Doctor's Guide to Efficient Practice Management". Better still, call us toll-free: **800-336-0359.***



Designed for eye care professionals, by the people who know eye care best.

As an eye care professional, both the way you run your practice and the procedures you perform are very different from the way other practitioners operate.

Yet all too many of the new "doctor's computers" offer a single program which attempts to meet the needs of all practitioners, regardless of specialty.

The Acuity Systems program on the other hand, is the result of thousands of hours spent studying efficient eye care practices, as well as four years of research and development in the United States and Canada.

Tell me more.

- ☐ Please send me your free booklet "The Doctor's Guide to Efficient Practice Management."
- ☐ Please call to arrange a time for a systems analyst to visit my office to determine its feasibility for such a computer/word processor system.
- ☐ Please call to arrange a time for a representative to visit my office to demonstrate the system and fully explain all its features.

☐ M.D.
☐ O.D.

Name _____

Street _____

City _____ State _____ Zip _____

Your Practice Specialty _____

Telephone ( ) _____

We would like to tailor a package of information best suited to your interest, so please provide us with this information to help us best respond:

1. I ☐ have ☐ have not previously investigated a computer system for my office needs.
2. I ☐ have ☐ have not previously investigated a word processing system for my office.
3. My patient billing currently ☐ is ☐ is not computerized.
4. I have a front-office staff of _____ persons.
5. In a typical day I see approximately _____ patients.
6. About _____% of my patients require some type of 3rd-party form to be completed by me.
7. I currently am re-paid by _____ types of 3rd party insurance or government plans.
8. My interest in such a system is ☐ immediate ☐ 6 to 12 months ☐ long-term.

Mail to: Acuity Systems, Inc. AJOPH080
11413 Isaac Newton Sq., Reston, VA 22090

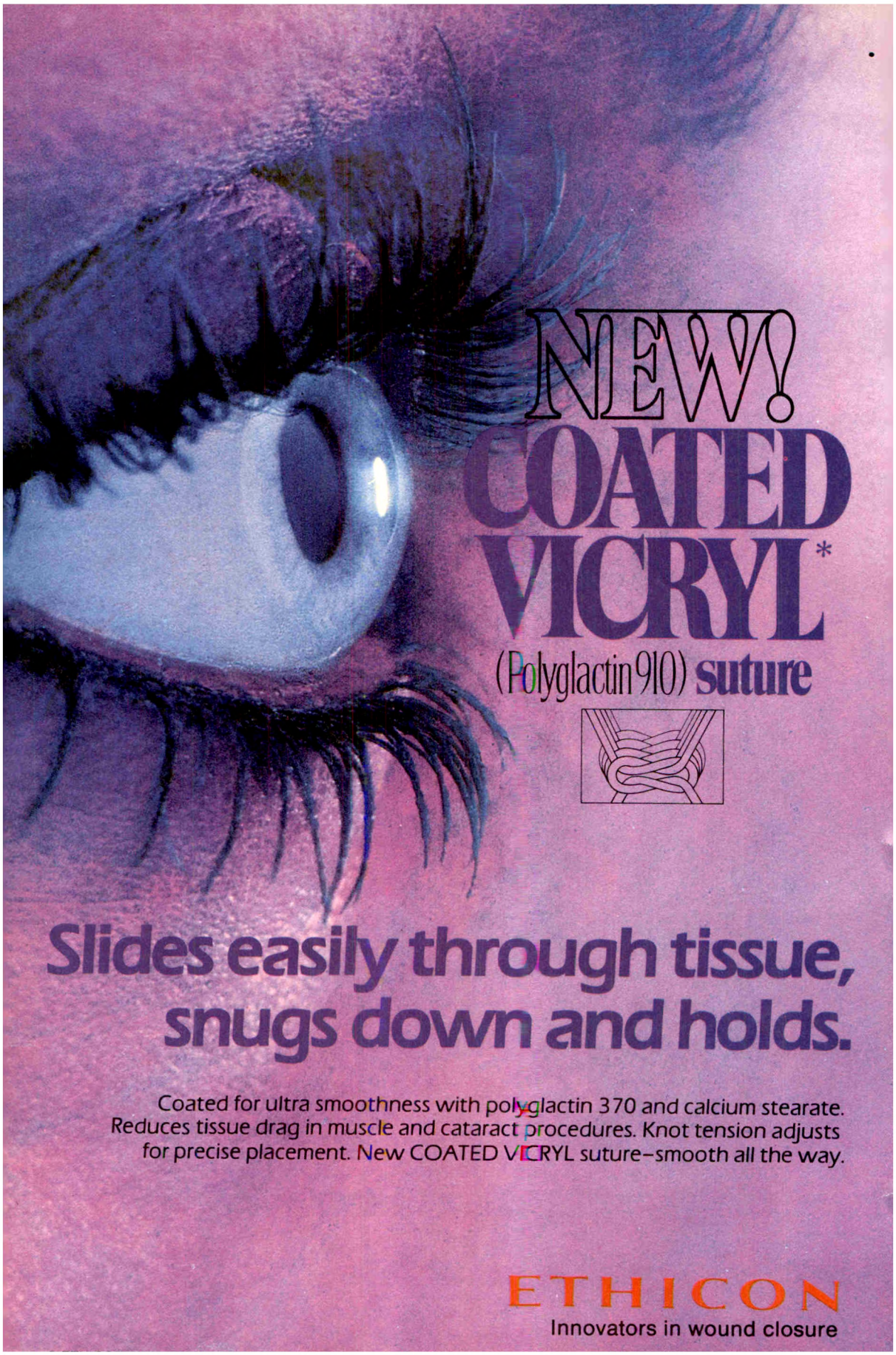
ACUITY SYSTEMS INC.

Advanced Instrumentation and Systems for Health Care

11413 Isaac Newton Sq. Reston, VA 22090

Medical Instrumentation Division, Simmonds Precision Products, Inc.

*In Virginia, call collect: (703) 471-4700.


NEW!
COATED
VICRYL*
(Polyglactin 910) suture
Slides easily through tissue, snugs down and holds.
Coated for ultra smoothness with polyglactin 370 and calcium stearate. Reduces tissue drag in muscle and cataract procedures. Knot tension adjusts for precise placement. New COATED VICRYL suture–smooth all the way.
ETHICON
Innovators in wound closure

Revised: 3/79

VICRYL*
(Polyglactin 910) Suture
SYNTHETIC ABSORBABLE SUTURE

Coated VICRYL*
(Polyglactin 910) Suture
Coated with Polyglactin 370 and Calcium Stearate
SYNTHETIC ABSORBABLE SUTURE

DESCRIPTION
VICRYL (polyglactin 910) synthetic absorbable suture is prepared from a copolymer of glycolide and lactide. These substances are derived respectively from glycolic and lactic acids. The empirical formula of the copolymer is $(C_2H_2O_2)_m(C_3H_4O_2)_n$.

Coated VICRYL synthetic absorbable suture is prepared by coating the VICRYL (polyglactin 910) suture material with a mixture composed of equal parts of a copolymer of glycolide and lactide (polyglactin 370) and calcium stearate.

These sutures are sterile, inert, nonantigenic, nonpyrogenic, and elicit only a mild tissue reaction during absorption. The braided and monofilament sutures are colored violet to enhance visibility in tissue. The braided suture is also available undyed (natural).

VICRYL and Coated VICRYL sutures differ from U.S.P. maximum diameter requirements by less than 0.05mm.

ACTIONS
Two important characteristics describe the *in vivo* behavior of absorbable sutures: first, tensile strength retention, and second, the absorption rate (loss of mass).

Subcutaneous tissue implantation studies of both VICRYL and Coated VICRYL suture in rats show at two weeks post-implantation approximately 55% of its original tensile strength remains, while at three weeks approximately 20% of its orginal strength is retained.

Intramuscular implantation studies in rats show that the absorption of these sutures is minimal until about the 40th post-implantation day. Absorption is essentially complete between the 60th and 90th days.

INDICATIONS
VICRYL and Coated VICRYL synthetic absorbable sutures are intended for use as absorbable sutures or ligatures.

CONTRAINDICATIONS
These sutures, being absorbable, should not be used where extended approximation of tissues under stress is required.

WARNINGS
The safety and effectiveness of VICRYL (polyglactin 910) and Coated VICRYL sutures in neural tissue and in cardiovascular tissue have not been established.

Under certain circumstances, notably orthopedic procedures, immobilization by external support may be employed at the discretion of the surgeon.

Do not resterilize.

PRECAUTIONS
The VICRYL suture knots must be properly placed to be secure. Place the first throw in precise position for the final knot, using a double loop; tie the second throw square, using horizontal tension; additional throws are advisable.

Coated VICRYL sutures, which are treated to enhance handling characteristics, require the standard surgical technique of flat and square ties with additional throws if indicated by surgical circumstance and the experience of the surgeon.

Skin and conjunctival sutures remaining in place longer than 7 days may cause localized irritation and should be removed as indicated.

Acceptable surgical practice must be followed with respect to drainage and closure of infected wounds.

ADVERSE REACTIONS
Reactions reported in clinical trials which may have been suture related have been minimal. These include skin redness and induration, rare instances of hemorrhage, anastomotic leakage, wound separation in the eye, and abscesses.

DOSAGE AND ADMINISTRATION
Use as required per surgical procedure.

HOW SUPPLIED
VICRYL sutures are available sterile as braided dyed (violet) strands in sizes 9-0 thru 3 (metric size 0.3—6) and undyed (natural) strands in sizes 8-0 thru 3 (metric size 0.4—6), in a variety of lengths, with or without needles, and on LIGAPAK® ligating reels. VICRYL sutures, monofilament, dyed (violet) are available in sizes 10-0 (metric size 0.2) and 9-0 (metric size 0.3), in a variety of lengths with needles.

Coated VICRYL sutures are available sterile, as braided dyed (violet) and undyed (natural) strands in sizes 8-0 thru 2 (metric size 0.4—5), in a variety of lengths, with or without needles, and on LIGAPAK ligating reels.

Both VICRYL and Coated VICRYL are also available in sizes 4-0 thru 1 (metric size 1.5—4) attached to ATRALEASE® CONTROL RELEASE® removable needles.

998414 Made in U.S.A.

*Trademark

ETHICON

MID WINTER NATIONAL MEETING

SAHARA HOTEL, LAS VEGAS, NEVADA
January 29 — February 1, 1981
(Meeting adjourns 12:00 noon Sunday)

CONTACT LENS ASSOCIATION OF OPHTHALMOLOGISTS, INC.

CONTACT LENS COURSES

INTRAOCULAR LENS COURSES

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SYMPOSIA ON CONTACT LENSES

SYMPOSIA ON INTRAOCULAR LENSES

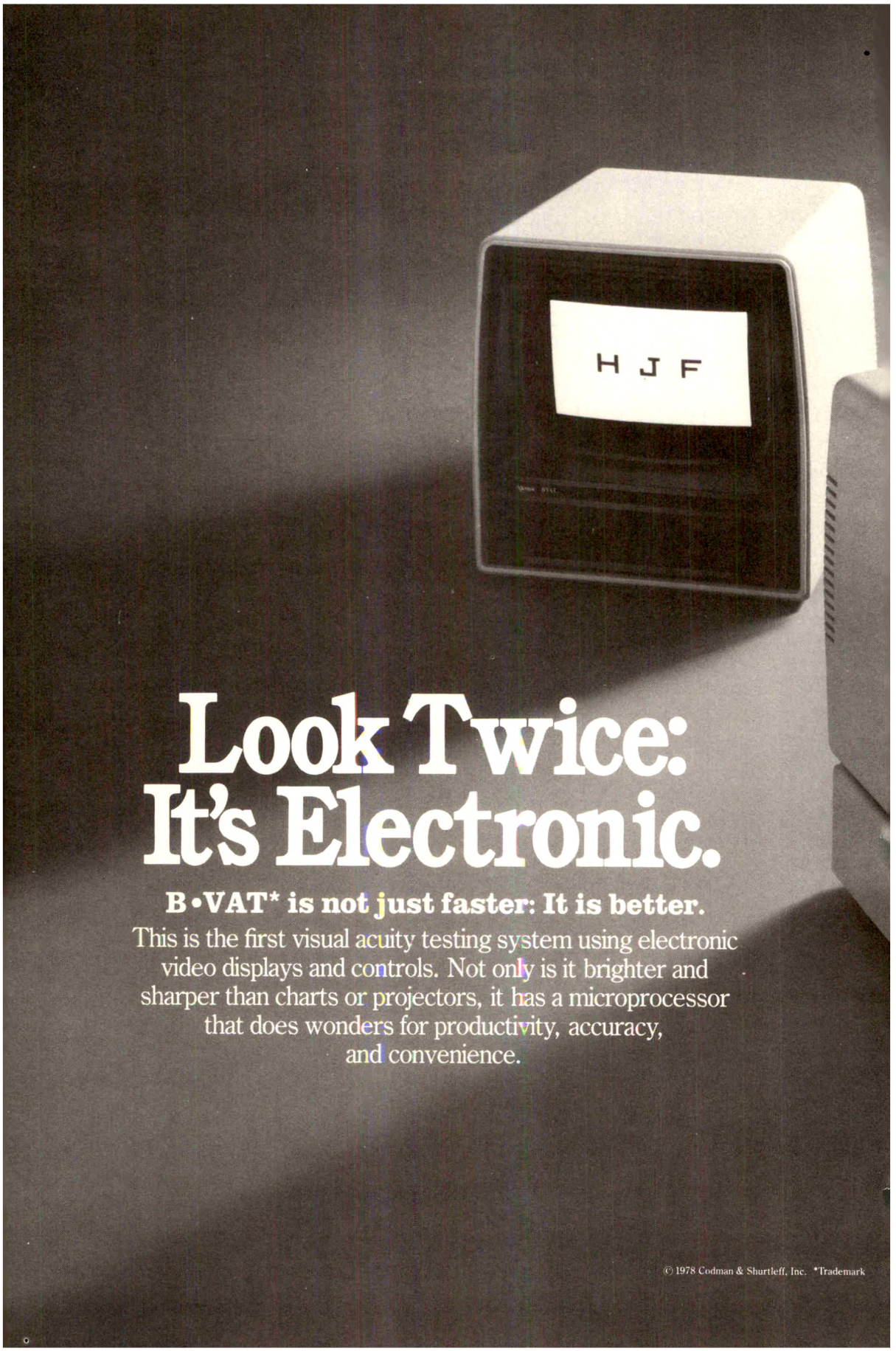
OPHTHALMIC ASSISTANTS COURSE

PRACTICE MANAGEMENT SEMINAR

Free Papers on Contact & Intraocular Lenses. Abstract Accepted No Later than July 1, 1980. An abstract of 200 words must accompany request.

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Ellis Gruber, M.D.
Contact Lens Association of Ophthalmologists, Inc.
2620 Jena Street
New Orleans, Louisiana 70115

For information regarding rental of exhibit space, contact:
Mrs. Marie S. Salassi
Executive Secretary
Contact Lens Association of Ophthalmologists, Inc.
2620 Jena Street
New Orleans, Louisiana 70115

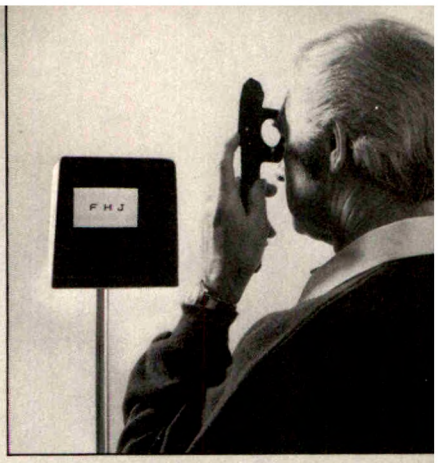


Look Twice: It's Electronic.

B•VAT* is not just faster: It is better.

This is the first visual acuity testing system using electronic video displays and controls. Not only is it brighter and sharper than charts or projectors, it has a microprocessor that does wonders for productivity, accuracy, and convenience.

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Your patient's display. B•VAT's character displays are exceptionally sharp, bright, and precisely proportioned. They can't be memorized, because random combinations may be generated each time a display goes up. Successive lines always have completely different characters. You can establish acuity, in Snelling increments or in increments of five, all the way from 20/10 to 20/400 (for the first time a low-vision patient's acuity can be closely determined). The display can be set up at any viewing distance between 10 and 20 feet, with the letter sizes adjusted accordingly. A duo-chrome test is built in.

Your control console. You have your own display showing exactly what the patient sees and a control console. That means you can face the patient normally and watch his actions while simultaneously selecting displays and monitoring them.

You select characters and lines instantly at the touch of a button. No waiting for slides to advance, no lens cleaning or bulb replacement.

Patient acuity threshold is established quickly using B•VAT's zoom control: A 20/15 letter grows in increments of five automatically until the patient recognizes it, at which point the size is displayed in lighted numbers on your control panel.

No need to stop and record test results. They can be stored in B•VAT's "memory" and instantly recalled for recording.



B•VAT is a better idea whose time has come. It does everything the standard test requires, only faster, more accurately, and with unprecedented convenience.

Two good looks will convince you. Mail the coupon or call us and we'll arrange a demonstration. 800-225-0462.



Mentor

☐ Please call me to arrange a demonstration of B•VAT.
☐ Please send me more information.

Name _____

Address _____

_____ Zip _____

Telephone _____

Mentor Division of Codman, Randolph, Mass. 02368

183 newbury st. / danvers, ma 01923 / USA

☐ Please send more information

☐ Please have your representative contact me

Name _____ Title _____

Hospital or Clinic _____

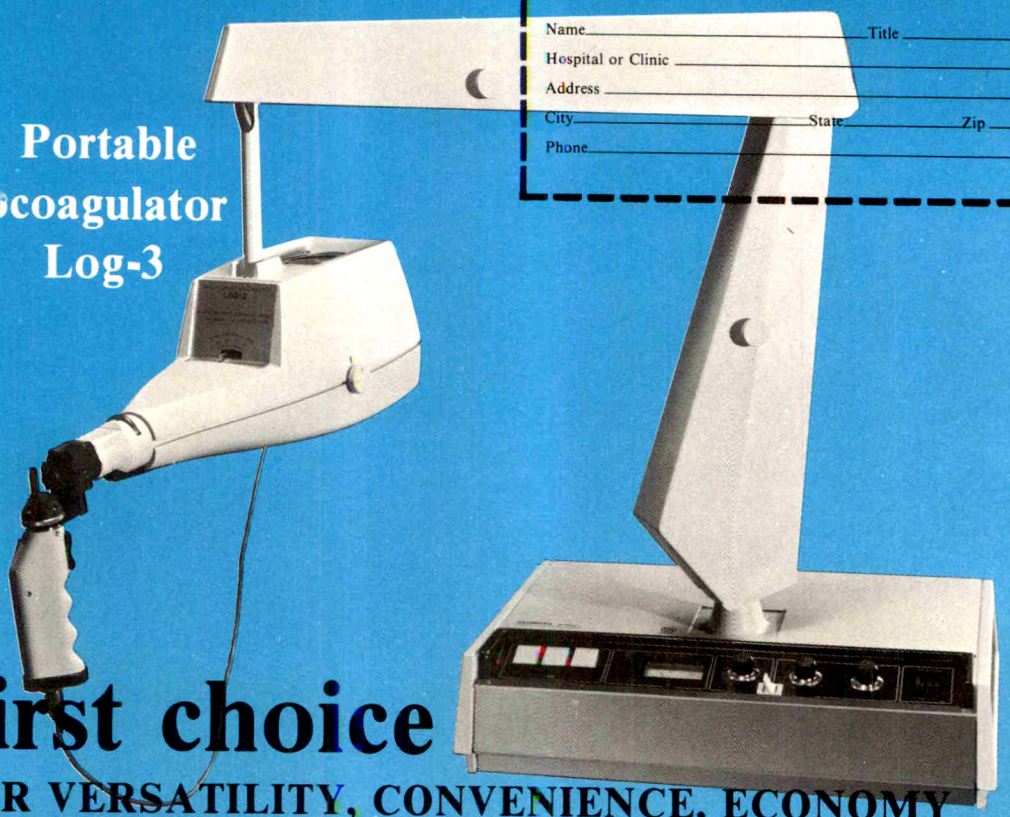
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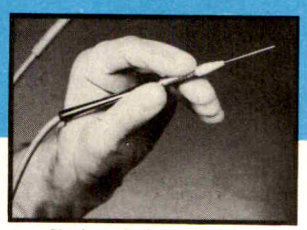
A

Portable Photocoagulator Log-3



First choice

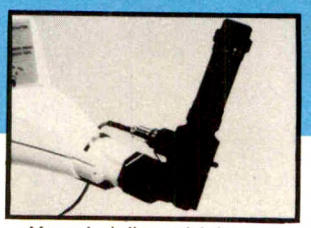
FOR VERSATILITY, CONVENIENCE, ECONOMY IN OFFICE, CLINIC, OR HOSPITAL



Charles endophotocoagulator



Iris coagulator attachment



Monocular indirect ophthalmoscope

Endo, Iris, and Exo Photocoagulation

The truly portable LOG-3 gives ophthalmologists the convenience of performing photocoagulation procedures whenever desired. This high quality, lightweight, compact instrument may be placed on a roll-around table and plugged to any outlet for mobility within office, clinic, or hospital. The unit can be hand carried from one facility to another. What's more, its attractive low price makes the LOG-3 an exceptionally cost-efficient ophthalmic instrument for private practice or institutional use.

Log-3 Features

- Direct ophthalmoscope delivery system standard. Monocular indirect erect image system with zoom and focus optional.
- A Charles Endophotocoagulator for Trans Pars Plana coagulation may be used.
- Powerful 300 watt Xenon lamp with built in parabolic reflector permits 1.5° aperture for close macula work and 6° for fast and efficient pan retinal application.
- Unique "optimatic" control combines power and time settings in a single dial.
- Articulated arm extends to make patient repositioning unnecessary.
- Operates on any standard line voltage.
- No installation costs.

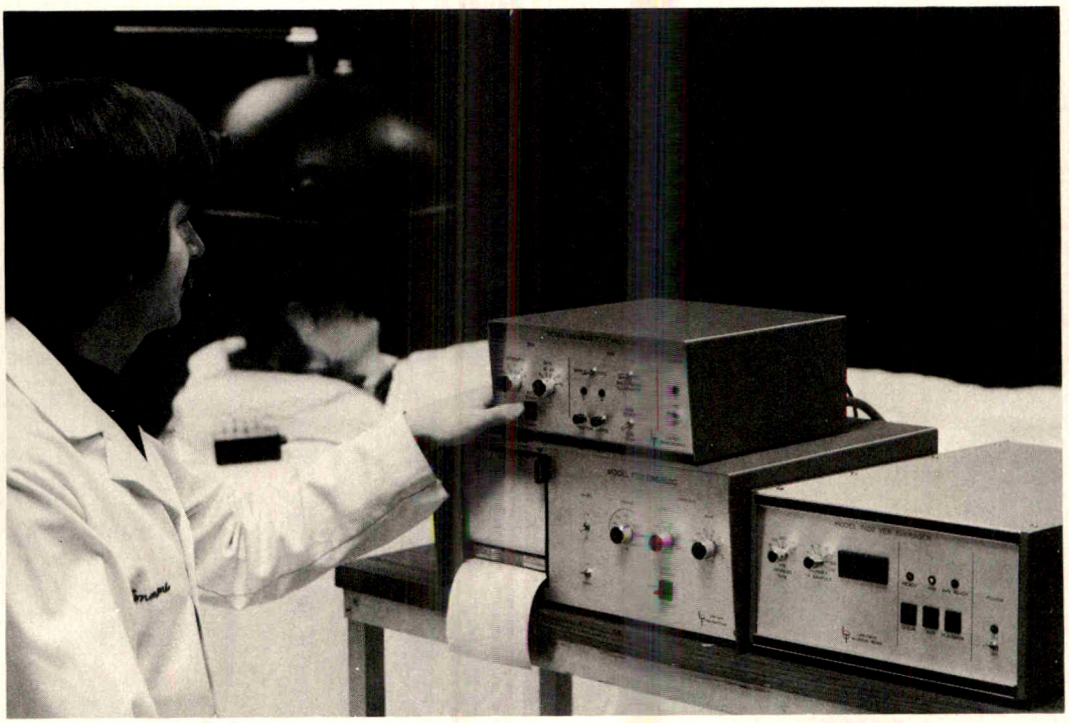
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VISUAL ELECTROPHYSIOLOGY TEST SYSTEM



VERSATILE: A single instrument records both the EOG and the ERG. In ERG mode, switch-selectable recording parameters optomize for conventional ERG, Flicker, Early Receptor Potential, or Oscillatory Potential. A plug-in digital averager expands system capability to include visual evoked responses (VERs) and low-level ERGs (eg., with eyelid rather than contact lens electrodes). Both ERG /EOG and averager are available in either single- or dual-channel versions. Also available is a complete selection of accessory instrumentation, including stimulators, electrodes, and monitor oscilloscope.

ACCURATE: ERG and VER responses are stored in digital memory to achieve the high frequency response necessary to record the ERG and VER with fidelity.

CLINICALLY PRACTICAL: Rugged, compact and lightweight. Simplified controls allow technical personnel to obtain high-quality records with minimal training. All responses are plotted at relatively slow paper speed to create compact records. Repeated activation of playback switch makes multiple copies of stored ERG or VER waveforms for teaching, reporting and multiple filing.

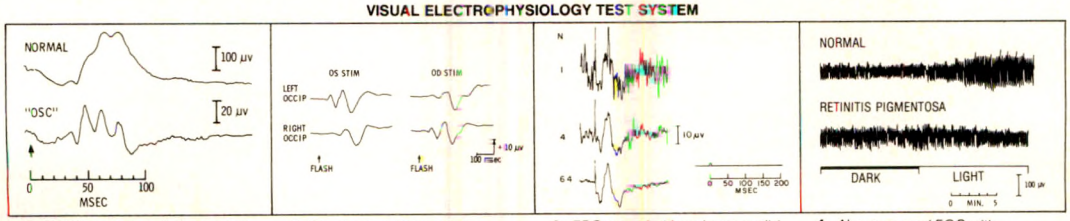
BACKED BY EXTENSIVE SERVICE FACILITIES: With purchase of a complete ERG system, trained Life-Tech medical instrumentation specialists will provide on-site installation and operator instruction.

All Life-Tech instruments are backed by a nationwide service center network supported by a comprehensive factory service department.

For additional information, call or write:

Life-Tech Instruments, Inc.
BOX 36221 - HOUSTON, TEXAS 77036 - (713) 783-6490

VISUAL ELECTROPHYSIOLOGY TEST SYSTEM



1. *ERG from normal dark-adapted eye. Above: "Normal" mode; Below: "OSC" mode, gain increased. In "OSC" mode the response is filtered to emphasize the oscillatory potential.*

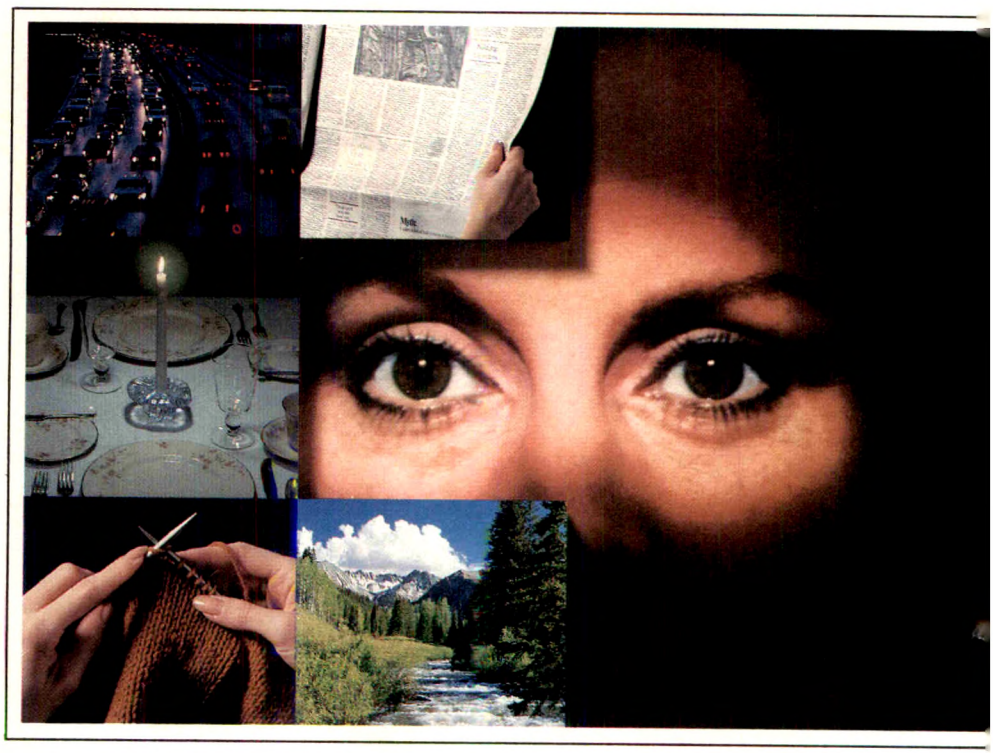
2. *Visual evoked responses recorded from right and left occiputs (dual channel system). The patient has an optic chiasm lesion.*

3. *ERG recorded from lower eyelid with averaging. "N"s at left of traces indicate the number of responses averaged.*

4. *Above: normal EOG with light-peak /dark-trough ratio of 2.4. Below: abnormal EOG in retinitis pigmentosa with light-peak / dark-trough ratio of 1.0. (EOG mode, 0.5 mm chart speed).*

In chronic open-angle glaucoma therapy

Two good reasons to switch patients to



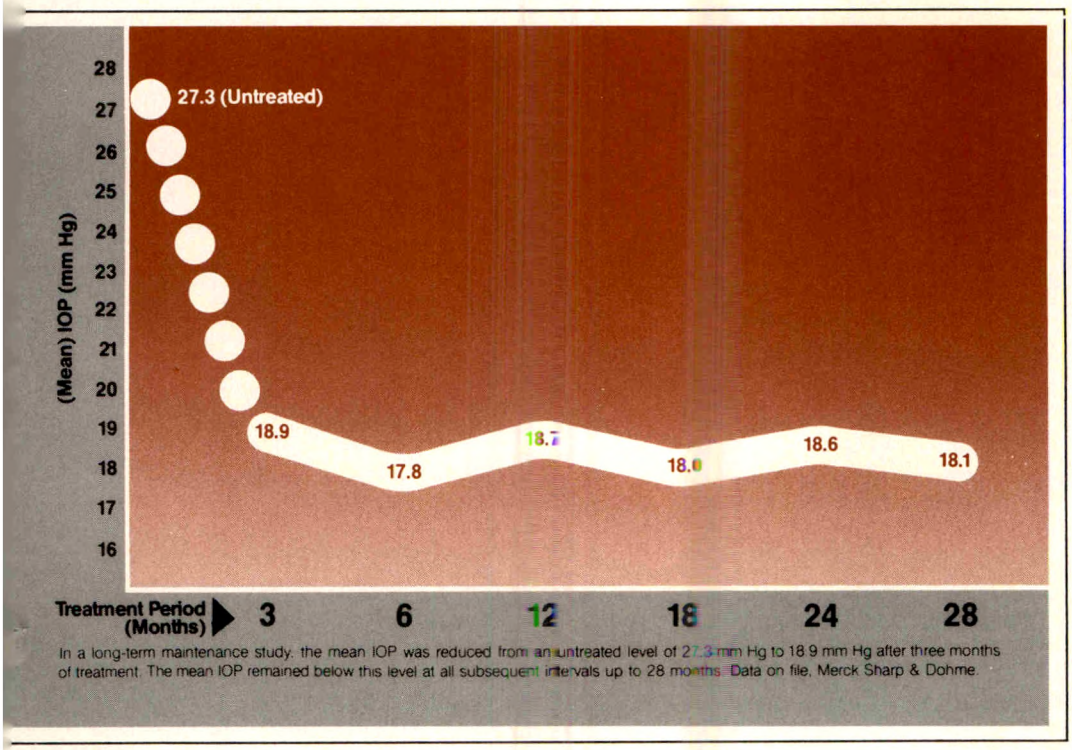
1. A better quality of life for many patients...

Patients on TIMOPTIC® (Timolol Maleate, MSD) generally do *not* experience the following therapy-limiting side effects:

change in pupil size/dimming of vision/night blindness*

TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product. Occasionally, mild ocular irritation has been reported.

Timoptic®
(TIMOLOL MALEATE | MSD)
OPHTHALMIC SOLUTION



In a long-term maintenance study, the mean IOP was reduced from an untreated level of 27.3 mm Hg to 18.9 mm Hg after three months of treatment. The mean IOP remained below this level at all subsequent intervals up to 28 months. Data on file, Merck Sharp & Dohme.

2. Efficacy maintained in long-term therapy...

- No tolerance was observed in 102 patients for 28 months
- TIMOPTIC was significantly more effective than pilocarpine or epinephrine in reducing elevated IOP*
- TIMOPTIC reduced IOP in 3 out of 4 patients*

*Based on controlled multiclinic studies, evaluating over 1000 patients.

MSD MERCK SHARP DOHME

For a brief summary of Prescribing Information, please see following page. Copyright © 1979 by Merck & Co., Inc.

NOW AVAILABLE
10-ml OCUMETER®



Timoptic®

(TIMOLOL MALEATE | MSD) OPHTHALMIC SOLUTION

a beta-adrenergic blocking agent in ophthalmic solution

a significant advance for the reduction of IOP in:

- chronic open-angle glaucoma
- aphakic glaucoma
- some patients with secondary glaucoma
- patients with elevated intraocular pressure at sufficient risk to require lowering of the ocular pressure
- some patients who respond inadequately to multiple antiglaucoma drug therapy

How to start patients on TIMOPTIC:
Usual starting dosage: one drop 0.25% TIMOPTIC in each eye twice a day.

How to transfer from current therapy to TIMOPTIC:
1. On the first day: continue with the agent(s) already being used and add one drop 0.25% TIMOPTIC in each eye b.i.d.

2. On the second day: discontinue the previously used agent(s) and continue with TIMOPTIC in each eye b.i.d.

If clinical response is not adequate:
Dosage may be increased (from the 0.25% solution) by changing to one drop 0.5% TIMOPTIC twice a day in each eye. Dosages above one drop of 0.5% TIMOPTIC twice a day generally have not been shown to produce further reduction of IOP. If the intraocular pressure is maintained at satisfactory levels, the dosage schedule may be changed to one drop once a day in each eye.

Because of diurnal variations in intraocular pressure, satisfactory response to the once-a-day dose is best determined by measuring the intraocular pressure at different times during the day.

In patients with a history of severe cardiac disease, pulse rates should be checked.

℞

Timoptic
(Timolol
Maleate | MSD)
0.25%
one drop
in each eye
b.i.d.

CONTRAINDICATIONS: TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product.
PRECAUTIONS: Should be used with caution in patients with known contraindications to systemic use of beta-adrenergic receptor blocking agents. These include bronchial asthma; sinus bradycardia and greater than first-degree block; cardiogenic shock; right ventricular failure secondary to pulmonary hypertension; congestive heart failure; concomitant use with adrenergic-augmenting psychotropic drugs. In patients with a history of severe cardiac disease, pulse rates should be checked. Patients who are already receiving a beta-adrenergic blocking agent orally and who are given TIMOPTIC should be observed for a potential additive effect either on the intraocular pressure or on the known systemic effects of beta blockade.

Sufficient clinical data have not been obtained to show safety or efficacy in narrow-angle or angle-closure glaucoma.
Pregnancy: TIMOPTIC has not been studied in human pregnancy. The use of TIMOPTIC in pregnant women requires that the anticipated benefit be weighed against possible hazards.
Use in Children: Since clinical studies in children have not been done, TIMOPTIC is not recommended for use in children at this time.
ADVERSE REACTIONS: In clinical trials of up to three years duration prior to release in 1978, signs and symptoms of mild ocular irritation were reported occasionally. Local hypersensitivity reactions, including localized and generalized rash, have occurred rarely.

Slight reduction of the resting heart rate (mean reduction 2.9 beats/minute, standard deviation 10.2) has been observed in some patients. Rarely, episodes of acute bronchospasm have been reported in patients with bronchospastic disease (see PRECAUTIONS).
Note: TIMOPTIC has been used in patients with glaucoma wearing conventional (PMMA) hard contact lenses, and has generally been well tolerated. TIMOPTIC has not been studied in patients wearing lenses made with materials other than PMMA.
HOW SUPPLIED: TIMOPTIC Ophthalmic Solution, 0.25% and TIMOPTIC Ophthalmic Solution, 0.5%. Both are available in 5-ml and 10-ml plastic OCUMETER® Ophthalmic Dispensers with a controlled drop tip.
580 (DC7046009)

MSD
MERCK SHARP & DOHME

For more detailed information, consult your MSD representative and the full Prescribing Information.
Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486



THE AMERICAN ACADEMY OF OPHTHALMOLOGY'S 1980 ANNUAL MEETING OFFERS:

- An opportunity to learn about the latest scientific advances in eye care from around the world.
- 22 half-day symposia and programs covering all aspects of ophthalmology.
- 52 new instruction courses and more than 250 courses repeated from last year by popular demand.
- 45 scientific exhibits and 74 posters.
- A daily film program on techniques not demonstrated easily by other means.
- About 200 technical exhibits displaying the newest technology, instruments, products, services and publications.
- Social events and other opportunities to meet informally and to exchange information with other ophthalmologists.
- A chance to visit the United States' second largest city during the week of the Presidential election.

The program will feature scientific sessions on:

- amblyopia
- cataracts
- computers in ophthalmology
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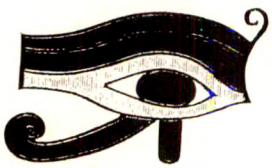
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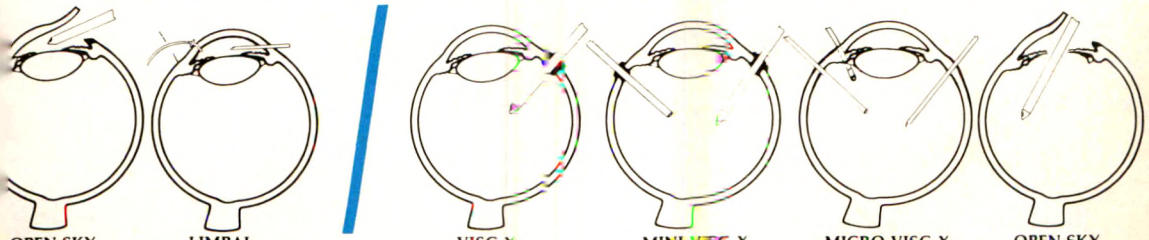
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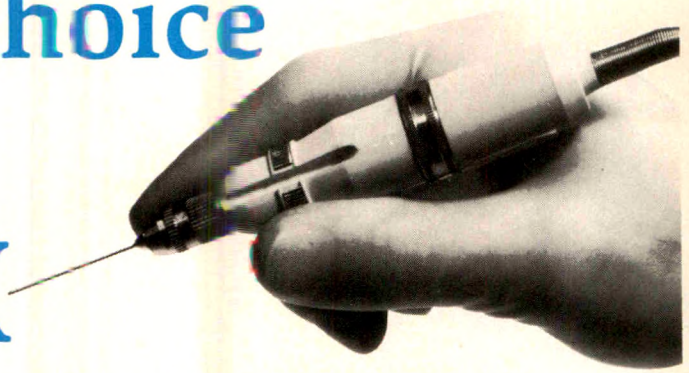
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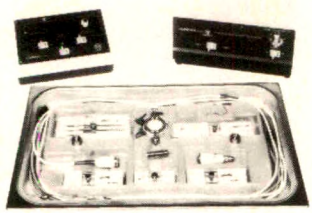
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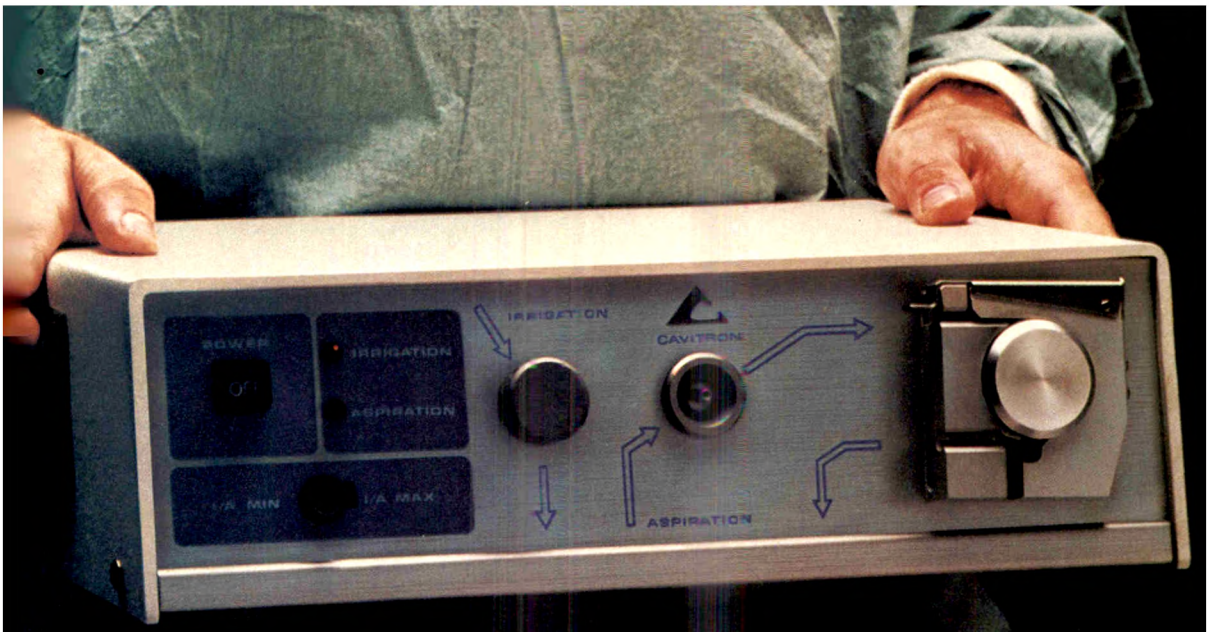
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ACTIONS The gram-positive bacteria against which gentamicin sulfate is active include coagulase-positive and coagulase-negative staphylococci, including certain strains that are resistant to penicillin; Group A beta-hemolytic and nonhemolytic streptococci; and *Diplococcus pneumoniae*. The gram-negative bacteria against which gentamicin sulfate is active include certain strains of *Pseudomonas aeruginosa*, indole-positive and indole-negative *Proteus* species, *Escherichia coli*, *Klebsiella pneumoniae* (Friedlander's bacillus), *Haemophilus influenzae* and *Haemophilus aegyptius* (Koch-Weeks bacillus), *Aerobacter aerogenes*, *Moraxella lacunata* (diplobacillus of Morax-Axenfeld), and *Neisseria* species, including *Neisseria gonorrhoeae*. Although significant resistant organisms have not been isolated from patients treated with gentamicin at the present time, this may occur in the future as resistance has been produced with difficulty *in vitro* by repeated exposures.

INDICATIONS GARAMYCIN **Ophthalmic Solution** and **Ointment** are indicated in the topical treatment of infections of the external eye and its adnexa caused by susceptible bacteria. Such infections embrace conjunctivitis, keratitis and keratoconjunctivitis, corneal ulcers, blepharitis and blepharoconjunctivitis, acute meibomianitis, and dacryocystitis.

CONTRAINDICATIONS GARAMYCIN **Ophthalmic Solution** and **Ointment** are contraindicated in patients with known hypersensitivity to any of the components.

WARNINGS GARAMYCIN **Ophthalmic Solution** is not for injection. It should never be injected subconjunctivally, nor should it be directly introduced into the anterior chamber of the eye.

PRECAUTIONS Prolonged use of topical antibiotics may give rise to overgrowth of nonsusceptible organisms, such as fungi. Should this occur, or if irritation or hypersensitivity to any component of the drug develops, discontinue use of the preparation and institute appropriate therapy.

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ADVERSE REACTIONS Transient irritation has been reported with the use of GARAMYCIN **Ophthalmic Solution.**

Occasional burning or stinging may occur with the use of GARAMYCIN **Ophthalmic Ointment.**

DOSAGE AND ADMINISTRATION GARAMYCIN **Ophthalmic Solution:** instill one or two drops into the affected eye every four hours. In severe infections, dosage may be increased to as much as two drops once every hour.

GARAMYCIN **Ophthalmic Ointment:** apply a small amount to the affected eye two to three times a day.

HOW SUPPLIED GARAMYCIN **Ophthalmic Solution—Sterile,** 5-ml. plastic dropper bottle, sterile, boxes of one and six. Store away from heat.

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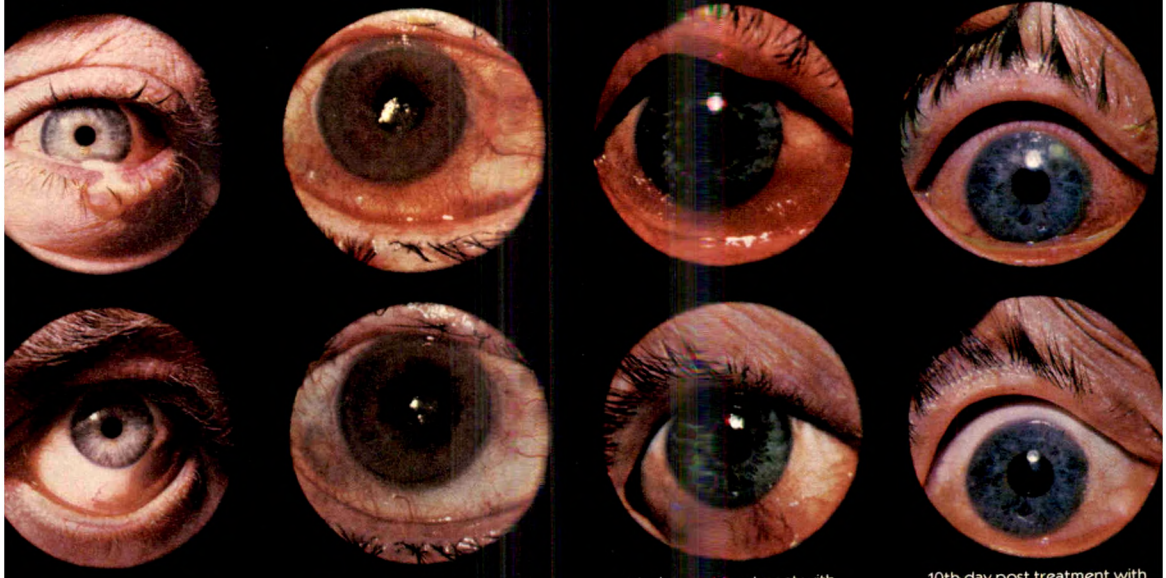
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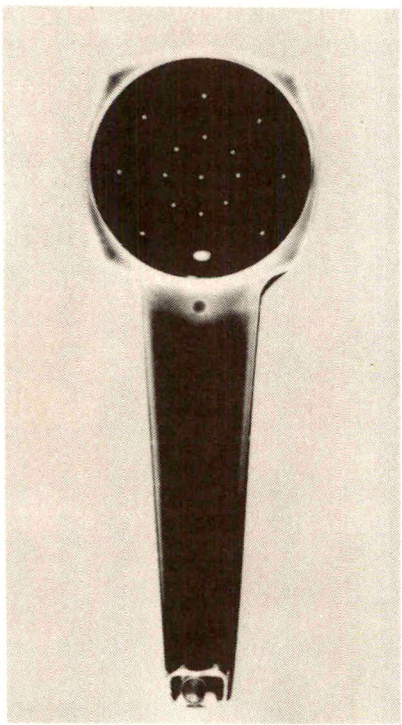
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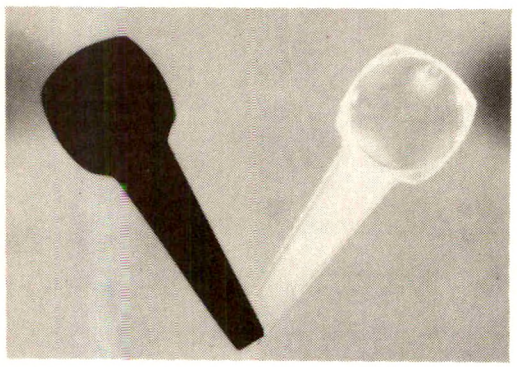
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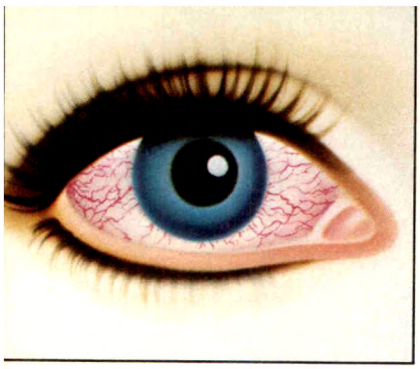
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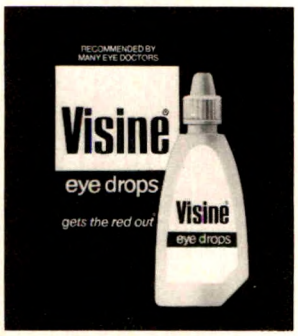
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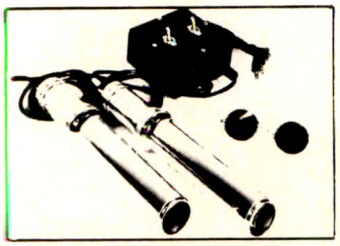
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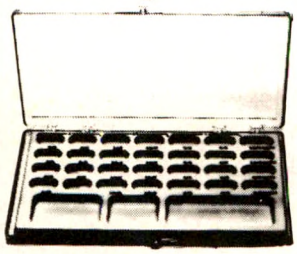
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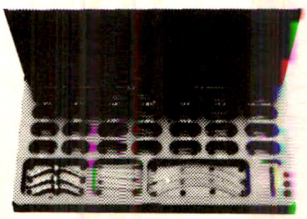
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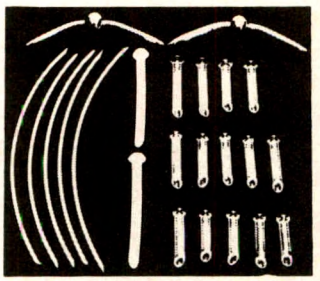
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J.C. Mustarde', L.T. Jones M.D. and A. Callahan M.D., Ophthalmic Plastic Surgery---Up-To-Date Aesculapius Publishing Company, 1970.
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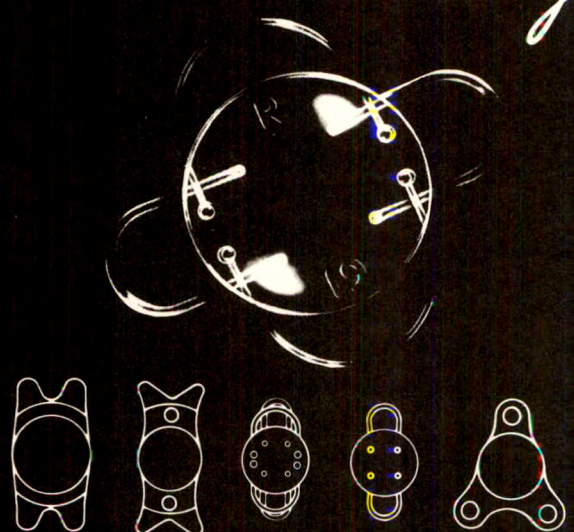
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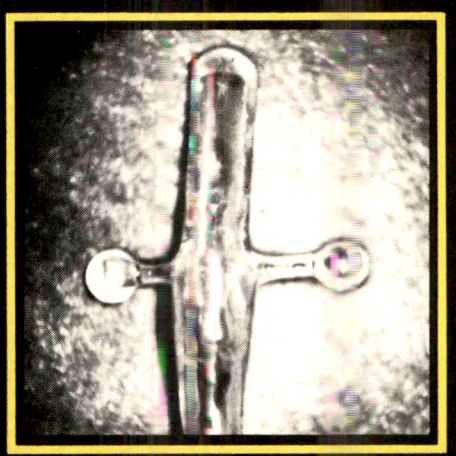
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Description. Vira-A (vidarabine, adenine arabinoside, or Ara-A) is an antiviral drug for topical ophthalmic administration. The chemical name is 9-β-D-arabinofuranosyladenine. Each gram of the ophthalmic ointment contains 30 mg of vidarabine monohydrate equivalent to 28.11 mg of vidarabine in a sterile, inert, petrolatum base.

Clinical Pharmacology. Vira-A is a purine nucleoside obtained from fermentation cultures of *Streptomyces antibioticus*. Vira-A possesses *in vitro* and *in vivo* antiviral activity against Herpes simplex types 1 and 2, Varicella-Zoster, and Vaccinia viruses. Except for Rhabdovirus and Oncornavirus, Vira-A does not display *in vitro* antiviral activity against other RNA or DNA viruses, including Adenovirus.

The antiviral mechanism of action has not been established. Vira-A appears to interfere with the early steps of viral DNA synthesis. Vira-A is rapidly deaminated to arabinosylhypoxanthine (Ara-Hx), the principal metabolite. Ara-Hx also possesses *in vitro* antiviral activity but this activity is less than that of Vira-A. Because of the low solubility of Vira-A, trace amounts of both Vira-A and Ara-Hx can be detected in the aqueous humor only if there is an epithelial defect in the cornea. If the cornea is normal, only trace amounts of Ara-Hx can be recovered from the aqueous humor.

Systemic absorption of Vira-A should not be expected to occur following ocular administration and swallowing lacrimal secretions. In laboratory animals, Vira-A is rapidly deaminated in the gastrointestinal tract to Ara-Hx.

In contrast to topical idoxuridine, Vira-A demonstrated less cellular toxicity in the regenerating corneal epithelium of the rabbit.

Indications and Usage. Vira-A Ophthalmic Ointment, 3%, is indicated for the treatment of acute keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus types 1 and 2. Vira-A is also effective in superficial keratitis caused by Herpes simplex virus which has not responded to topical idoxuridine or when toxic or hypersensitivity reactions to idoxuridine have occurred. The effectiveness of Vira-A Ophthalmic Ointment, 3%, against stromal keratitis and uveitis due to Herpes simplex virus has not been established.

The clinical diagnosis of keratitis caused by Herpes simplex virus is usually established by the presence of typical dendritic or geographic lesions on slit-lamp examination. In controlled and uncontrolled clinical trials, an average of seven and nine days of continuous Vira-A Ophthalmic Ointment, 3%, therapy was required to achieve corneal re-epithelialization. In the controlled trials, 70 of 81 subjects (86%) re-epithelialized at the end of three weeks of therapy. In the uncontrolled trials, 101 of 142 subjects (71%) re-epithelialized at the end of three weeks. Seventy-five percent of the subjects in these uncontrolled trials had either not healed previously or had developed hypersensitivity to topical idoxuridine therapy.

The following topical antibiotics: gentamicin, erythromycin, chloramphenicol; or topical steroids: prednisolone or dexamethasone, have been administered concurrently with Vira-A Ophthalmic Ointment, 3%, without an increase in adverse reactions.

Normally, corticosteroids alone are contraindicated in Herpes simplex virus infections of the eye. If Vira-A Ophthalmic Ointment, 3%, is administered concurrently with topical corticosteroid therapy, corticosteroid-induced ocular side effects must be considered. These include corticosteroid-induced glaucoma or cataract formation and progression of a bacterial or viral infection.

Vira-A is not effective against RNA virus or adenoviral ocular infections. Vira-A is also not effective against bacterial, fungal, or chlamydial infections of the cornea or nonviral trophic ulcers.

Contraindication. Vira-A Ophthalmic Ointment, 3%, is contraindicated in patients who develop hypersensitivity reactions to it.

Warnings. ***Use in Pregnancy:*** Vira-A (vidarabine) parenterally is teratogenic in rats and rabbits. Ten percent Vira-A ointment applied to 10% of the body surface during organogenesis induc fetal abnormalities in rabbits. When 10% Vira-A ointment was a plied to 2 to 3% of the body surface of rabbits, no fetal abn malities were found. This dose greatly exceeds the total reco mended ophthalmic dose in humans. The possibility of embryo or feta damage in pregnant women receiving Vira-A (vidarabi ophthalmic ointment), 3%, is remote. The topical ophthalmic do is smal, and the drug relatively insoluble. Its ocular penetration very low. However, a safe dose for a human embryo or fetus h not been established. Consequently, Vira-A should be used or when clearly indicated.

It is not known whether Vira-A is excreted in human milk. As general rule, because many drugs are excreted in human mi nursing should not be undertaken while a patient is under trea ment. However, excretion of vidarabine in breast milk is unlike because Vira-A is rapidly deaminated in the gastrointestinal tra

Mutagenic Potential: Results of *in vitro* experiments indica that vidarabine can be incorporated into mammalian DNA and ca induce mutation in mammalian cells (mouse L5178Y cell line Thus far, *in vivo* studies have not been as conclusive, but there some evidence (dominant lethal assay in mice) that vidarabir may be capable of producing mutagenic effects in male ger cells.

It has also been reported that vidarabine causes chromosom breaks and gaps when added to human leukocytes *in vitro*. Whi the significance of these effects in terms of mutagenicity is no fully understood, there is a well-known correlation between th ability of various agents to produce such effects and their abilit to produce heritable genetic damage.

Oncogenic Potential: Chronic parenteral (IM) studies c vidarabine have been conducted in mice and rats.

In the mouse study, there was a statistically significant ir crease in liver tumor incidence among the vidarabine-treate females. In the same study, some vidarabine-treated male mic developed kidney neoplasia. No renal tumors were found in th vehicle-treated control mice or the vidarabine-treated femal mice.

In the rat study, intestinal, testicular, and thyroid neoplasia oc curred with greater frequency among the vidarabine-treate animals than in the vehicle-treated controls. The increases i thyroid adenoma incidence in the high-dose (50 mg/kg) male and the low-dose (30 mg/kg) females were statistically signifi cant.

Hepatic megalocytosis, associated with vidarabine treatmen has been found in short- and long-term rodent (rat and mouse studies. It is not clear whether or not this represents a pre neoplastic change.

The recommended frequency and duration of administration should not be exceeded (see Dosage and Administration).

Precautions. The diagnosis of keratoconjunctivitis due to Herpes simplex virus should be established clinically prior to prescribing Vira-A Ophthalmic Ointment, 3%.

Patients should be forewarned that Vira-A Ophthalmic Ointment, 3%, like any ophthalmic ointment, may produce a temporary visual haze.

Although viral resistance to Vira-A has not been observed, this possibility may exist.

Adverse Reactions. Lacrimation, foreign-body sensation, conjunctival injection, burning, irritation, superficial punctate keratitis, pain, photophobia, punctal occlusion, and sensitivity have been reported with Vira-A Ophthalmic Ointment, 3%. The following have also been reported but appear disease-related: uveitis, stromal edema, secondary glaucoma, trophic defects, corneal vascularization, and hyphema.

Overdosage. Acute massive overdosage by oral ingestion of the ophthalmic ointment has not occurred. However, the rapid deamination to arabinosylhypoxanthine should preclude any difficulty. The oral LD_{50} for vidarabine is greater than 5,020 mg/kg in mice and rats. No untoward effects should result from ingestion of the entire contents of a tube.

Overdosage by ocular instillation is unlikely because any excess should be quickly expelled from the conjunctival sac. Too frequent administration should be avoided.

Dosage and Administration. Administer approximately one half inch of Vira-A Ophthalmic Ointment, 3%, into the lower conjunctival sac five times daily at three-hour intervals.

If there are no signs of improvement after 7 days, or complete re-epithelialization has not occurred by 21 days, other forms of therapy should be considered. Some severe cases may require longer treatment.

After re-epithelialization has occurred, treatment for an additional seven days at a reduced dosage (such as twice daily) is recommended in order to prevent recurrence.

How Supplied. N 0071-3677-07 (Stock 18-1677-139)

Vira-A Ophthalmic Ointment, 3%, is supplied sterile in ophthalmic ointment tubes of 3.5 g. The base is a 60:40 mixture of solid and liquid petrolatum. **UK**

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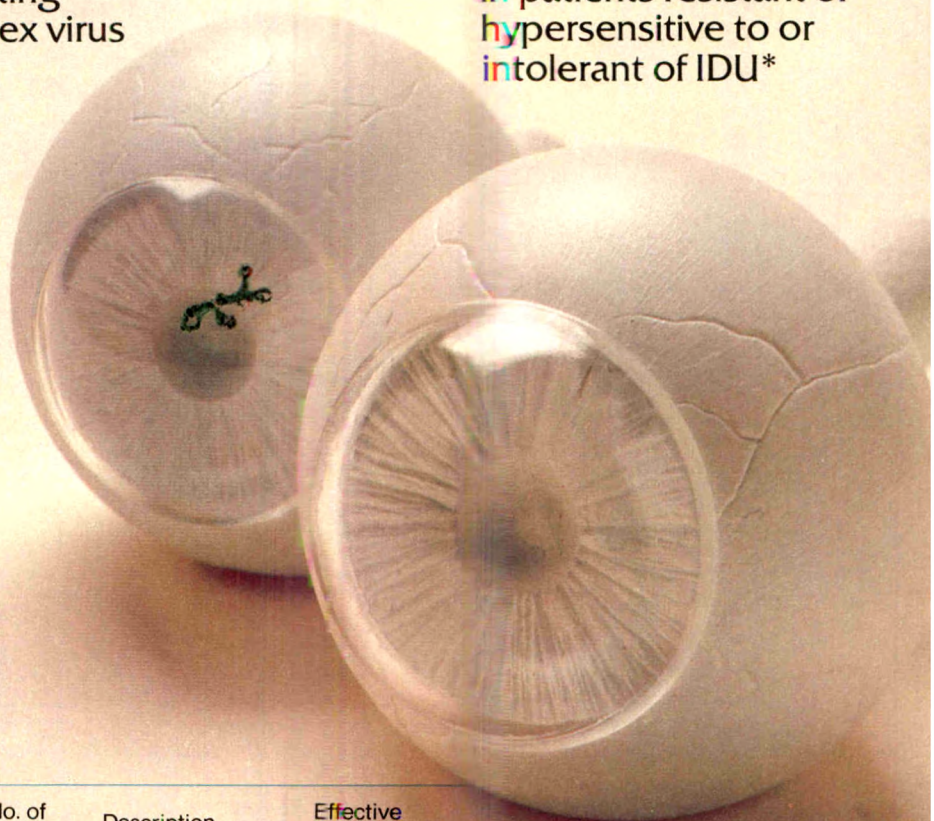
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Clinical Evidence	No. of Subjects	Description	Effective Response
Vira-A Controlled Trials	81	Previously untreated	70 subjects (86%)
Vira-A Uncontrolled Trials	142	75% had been resistant or hypersensitive to IDU	101 subjects (71%)

NOTE: In the controlled and uncontrolled trials, 70 and 101 subjects, respectively, re-epithelialized at the end of three weeks on Vira-A.

Data on file, Medical Department, Parke-Davis

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SURGICAL ADVANCES AND RESULTS OF KERATOCONUS

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In addition to obtaining clear corneal grafts, corneal surgeons strive to obtain emmetropic anastigmatic penetrating keratoplasty in the correction of keratoconus; however, no reliably reproducible technique for this has yet been found. In this study we attempted to improve our current surgical treatment of keratoconus and to reduce postoperative ametropia and astigmatism. The use of double continuous monofilament nylon suture with an 8-mm graft has a significant advantage over interrupted sutures in the reduction of postoperative myopia.[1] Residual astigmatism, however, was not substantially reduced. The corneal wedge resection[2] and the use of the surgical keratometer[3] also have been tried in the attempt to decrease resultant astigmatism. However, large astigmatic errors are still a primary problem in the final correction of keratoconus.[4-8]

Cutting the corneal button from the posterior surface (endothelial side up) gives a graft anterior diameter that is 0.2 mm smaller than that cut from the anterior side.[9] Therefore, the use of a larger donor button should compensate for this discrepancy. The use of through-and-through corneal sutures[10,11] effects better wound closure and prevents posterior wound edge gaping and thus provides a more securely approximated wound.

We compared the results of penetrating keratoplasty for keratoconus using disparate-sized and same-sized donor button and recipient bed. Keratoconus was selected because it is a common disorder requiring keratoplasty and because the peripheral cornea, though often thin and irregular, is not vascularized or otherwise significantly pathologic. Separately, we are analyzing grafts done for other pathologic conditions during the same time span and using similar surgical techniques.[12]

MATERIAL AND METHODS

For this study, we reviewed 94 consecutive cases of penetrating keratoplasty. Of these, 82 were suitable for analysis. Any patient previously operated on was not included in this study. In each case, the patient was not able to wear contact

From the State University of New York, Downstate Medical Center, Department of Surgery, Division of Ophthalmology, Brooklyn, New York (Dr. Troutman); and the Manhattan Eye, Ear and Throat Hospital, New York, New York (Dr. Gaster). Dr. Gaster was a Heed Foundation Fellow (1978-1979).

Reprint requests to Richard C. Troutman, M.D., State University of New York, Division of Ophthalmology, 450 Clarkson Ave., Brooklyn, NY 11203.

lenses or could not achieve useful vision before keratoplasty.

The operative technique included the following: A Pierse scleral expander ring was sutured in place. The two trephines to be used were carefully examined under the microscope for any nicks or flaws which might damage the tissue. We used the Troutman corneal punch to cut the donor button, endothelial side up. The patient's cornea was then incised with the trephine and the anterior chamber was entered. We used right and left cutting corneal scissors to complete the incision. Any remaining shelf of tissue was meticulously excised to ensure vertical apposition of the wound. A midperipheral iridectomy was performed in all cases. We placed the donor button in the recipient bed and, using the surgical keratometer, adjusted it to the position with the least amount of distortion possible. Six temporary 10-0 nylon fixation sutures were inserted and the anterior chamber was reformed. A double continuous 10-0 nylon suture with through-and-through bites was placed and the knots were buried. After removal of the fixation sutures and corneal ring, suture tension was adjusted and monitored with the surgical keratometer to an essentially anastigmatic state as shown by a circular pattern of the keratometer projection. Corticosteroids were applied topically three times a day and antibiotic drops were used four times a day postoperatively. Mydriatic or cycloplegic drops were not used routinely. The patient was discharged from the hospital on the third or fourth postoperative day. Keratometry and refraction were performed on each postoperative visit. The sutures were removed at about the sixth postoperative month.

Results

Eighty-two eyes of 53 patients qualified for analysis in this study. Twenty-

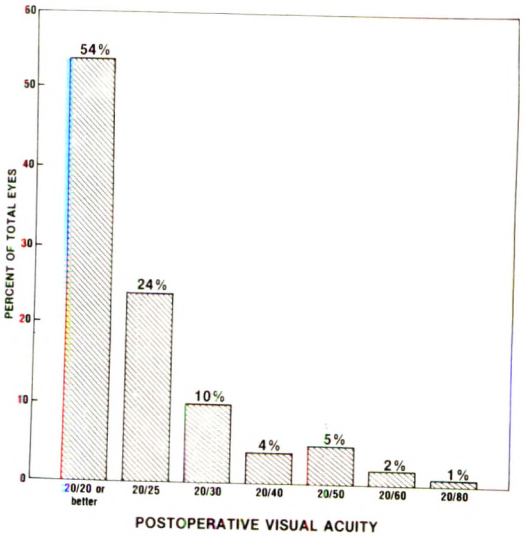


Fig. 1. (Troutman and Gaster). Postoperative visual acuity after keratoplasty for keratoconus (82 eyes).

nine patients had bilateral grafts. Of the 53 patients, 28 were male and 25 were female. Age at the time of surgery was, on the average, 41 years, ranging from 14 to 70 years. Postoperative visual acuity with follow-up of more than six months was 6/12 (20/40) or better in 75 eyes (92%) (Fig. 1). Of the seven eyes with visual acuity less than 6/12 (20/40), four were amblyopic and two had graft reaction that was being treated. Of the 82 eyes, 80 (98%) had clear grafts at the time of study.

The following complications were noted: Graft reaction occurred in six eyes (7.3%). Four were successfully treated and two required regrafting. One eye was regrafted twice. Of the six patients with graft reaction, only one had had previous keratoplasty in the fellow eye. The complications listed in Table 1 are classified according to the graft size.

Three different sizes of grafts were used. The first group (30 eyes) had the same-sized graft and recipient opening, an 8.0-mm diameter graft in an 8.0-mm diameter recipient opening. The second

TABLE 1

COMPLICATIONS AFTER KERATOPLASTY FOR KERATOCONUS

Complication	Eyes		Graft Size in 8.0-mm Recipient Opening			
	No.	%	8.0 mm	8.1 mm	8.2 mm	Other
Graft reaction	6	7.3	2	1	2	1*
Adhesion	2	2.4				
Iris to suture 1			1			
Iris to cornea 1				1		
Fixed pupil	2	2.4			1	1
Wound dehiscence	1	1.2	1			
Suture untied	1	1.2	1			
Iris prolapse	1	1.2				1
Transient elevation of intraocular pressure	1	1.2	1			
Corneal ulcer	1	1.2			1	

*8.0-mm graft in 7.5-mm recipient opening.

group (11 eyes) had an 8.1-mm graft in an 8.0-mm bed, and the third group (27 eyes) had an 8.2-mm graft in an 8.0-mm opening (Table 2). In 14 eyes the graft and the recipient opening were of sizes different from those just indicated. Because these other groups were too small to analyze, either separately or collectively, they were excluded from the pre- and postoperative K readings as averaged below. The average postoperative keratometer readings of these three groups were 42.3 diopters for the 8.0-mm grafts, 43.4 diopters for the 8.1-mm grafts, and 42.7 diopters for the 8.2-mm grafts. The average astigmatic errors for these three groups as measured by the keratometer were 3.8 diopters for the 8.0-mm grafts, 4.0 diopters for the 8.1-mm grafts, and 4.1 diopters for the 8.2-mm grafts (Fig. 2). The average spherical equivalent of all eyes approximated emmetropia, which is a marked improvement over the moderate spherical equivalent myopia, averaging 3.0 to 5.0 diopters, reported in previous studies.[1] To ascertain how the keratometer readings change with time, after keratoplasty, the K readings were averaged before and after suture removal for each of the three groups. These averages

TABLE 2

POSTOPERATIVE AVERAGE K READINGS AND AVERAGE ASTIGMATISM BY REFRACTION ACCORDING TO GRAFT-RECIPIENT OPENING RELATIONSHIP

Graft Size (mm)	Recipient Opening (mm)	No. of Eyes	Average K Reading (Diopters)	Average Astigmatism by Refraction (Diopters)
8.0	8.0	30	42.3	3.8
8.1	8.0	11	43.5	4.0
8.2	8.0	27	42.7	4.1
Other	Other	14	—	—

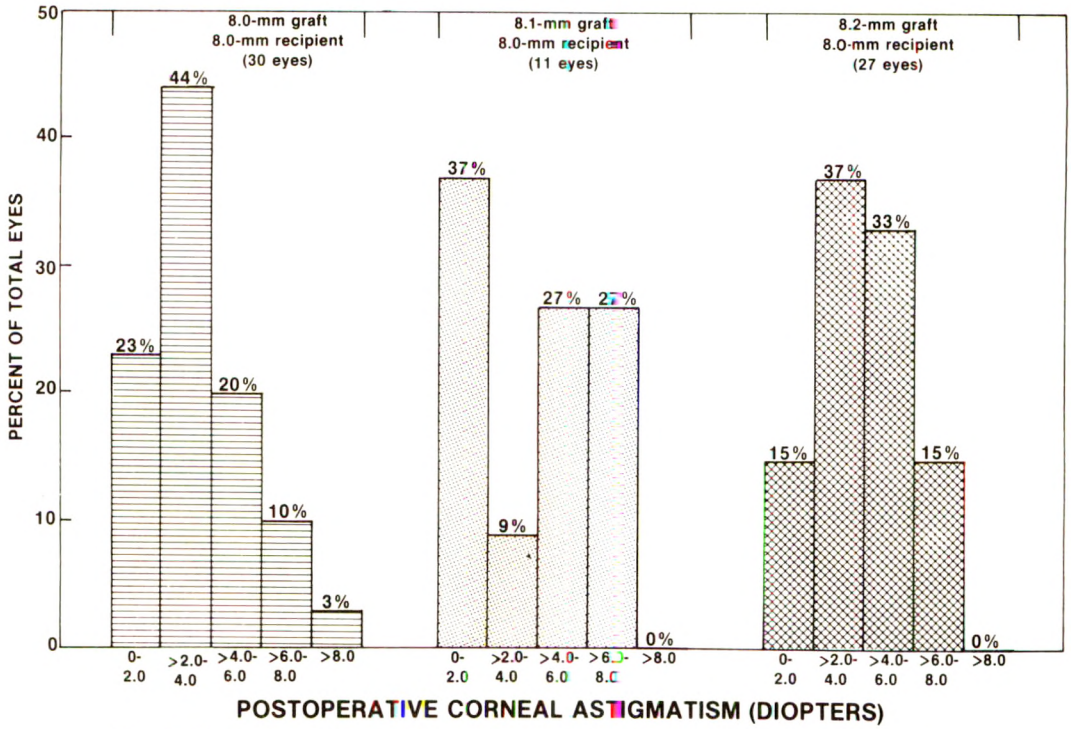


Fig. 2. (Troutman and Gaster). Corneal astigmatism after keratoplasty for keratoconus.

were based on only those eyes for which both readings were available. Keratometry done before and after suture removal showed no significant difference in the amount of astigmatism or any trend in the axis of the astigmatism. The average keratometer readings shown in Table 3 indicate the mean, the standard deviation, the standard error of the mean, and the range for each group.

Discussion

In the keratoconic cornea, the cone is often oval in shape and distortion may extend to the corneal periphery.[13] The use of through-and-through sutures has the advantages of complete closure of the wound from the anterior to the posterior surface and of reduction in postoperative myopia, and it also has the potential disadvantage that the graft is closely approximated in depth to the recipient abnormal, keratoconus-distorted peripheral cornea and thus may take on this abnormal astigmatic shape postoperatively. This circumstance may explain the continued persistence of the postoperative 4.0 diopters of astigmatism that we found in this study, despite changing from 8.0 to 8.1 or 8.2-mm diameter grafts. This difference in graft size did not significantly change the refractive power of the eye toward hyperopia and perhaps the use of an even larger graft in keratoconus, such as an 8.5-mm graft, in combination with a through-and-through closure, might result in less distortion by the peripheral recipient cornea.

An even more difficult task has been the surgical alteration of the peripheral cornea. Two patients not included in this

TABLE 3

AVERAGE KERATOMETER READINGS BEFORE AND AFTER SUTURE REMOVAL

Graft Size	Before Suture Removal	After Suture Removal
8.0-mm graft in 8.0-mm recipient		
No. of eyes	22	22
Mean	40.70	42.53
Range	33.25–48.50	37.87–46.75
S.D.	3.90	2.18
Standard error of the mean	0.83	0.46
8.1-mm graft in 8.0-mm recipient		
No. of eyes	10	10
Mean	41.50	43.55
Range	35.50–45.25	40.25–47.87
S.D.	3.22	2.72
Standard error of the mean	1.02	0.86
8.2-mm graft in 8.0-mm recipient		
No. of eyes	21	21
Mean	41.76	42.96
Range	36.62–48.00	40.00–48.12
S.D.	3.48	2.10
Standard error of the mean	0.76	0.46

study had penetrating keratoplasty larger than 9 mm and both suffered graft reaction which required repeat grafting. Surgical alteration of the peripheral cornea might be effected by using an oval, rather than a circular, graft oriented in such a way as to correct the meridional defect. This has been attempted but it has been difficult to predict exactly the amount of correction.[14]

Keratometer readings were taken before and after suture removal. There was no significant decrease in astigmatism caused by any maneuver performed with the sutures, that is, early vs. late removal or selective removal of one of the double continuous sutures. Moreover, despite meticulous technique and the use of the surgical keratometer to fixate the graft as spherically as possible at the end of the keratoplasty, we still found up to 11.0 diopters of keratometer-measured astigmatism after suture removal. It is important to differentiate between keratometer-measured astigmatism and refraction-measured astigmatism, especially in patients who require a high plus correction, because refraction-measured astigmatism is less than keratometer-measured astigmatism as the result of the difference in the plane of the measurement — the former being in the spectacle plane, the latter being at the cornea. These findings emphasize the lack of control we have on astigmatism after removing sutures in the cornea after keratoplasty for keratoconus.

The finding of 98% clear grafts with excellent visual recovery confirms the results of other studies.[4-8,15,16] Additionally, there was no postoperative glaucoma and only two eyes had fixed unresponsive pupils, both traumatic in origin. The low incidence of graft reaction and the few other complications associated with the procedure are further indications of the advances thus far achieved. Despite a recent article[17] that indicated an increased incidence of graft rejection after keratoplasty on the second eye, we found that only one patient of 29 with bilateral grafts had a graft reaction in the first eye after the second graft was performed.

SUMMARY

We analyzed 82 eyes of 53 patients who underwent penetrating keratoplasty for keratoconus. Despite meticulous attention to microsurgical technique, and the use of the corneal punch, 10-0 monofilament nylon double continuous suture, and the surgical keratometer, and sizing the donor graft to recipient bed, we found a postoperative astigmatism of about 4.0 diopters. The percentage of clear grafts is now high and the complication rate low. However, we still have not developed a technique that ensures an

anastigmatic cornea after keratoplasty for keratoconus.

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THERMAL WEDGE WITH PENETRATING KERATOPLASTY TO REDUCE HIGH CORNEAL CYLINDER

JOSEPH L. HATCH, M.D.
Salt Lake City, Utah

An ideal time to take measures to reduce excessive amounts of corneal astigmatism is during keratoplasty. This can be done by a new technique that involves the use of a thermal wedge resection.

Topical cautery is used to shrink corneal collagen and draw a thermal wedge of corneal tissue into a previously marked area. The mark is made by gently pressing the trephine, which is to be used to cut the recipient button, on the patient's cornea. The thermal wedge procedure affords the same net effect as a tissue wedge resection combined with keratoplasty. This new technique has resulted in reduction of the astigmatic error by as much as 8.5 diopters.

Often the preoperative condition is associated with wound healing difficulties from previous keratoplasty. This technique can also be used in other cases with high cylindrical errors. The patient's cornea may or may not have scarring or edema.

MATERIAL AND METHODS

A keratometer is used to determine the flattest and steepest meridians of the cornea. Figure 1 shows the appearance of the mires of the Bausch and Lomb keratometer in a case where the flat meridian of the cornea is at 45 degrees (1:30 to 7:30 o'clock on the corneal dial). The mires are pear-shaped. The more pointed end of the mire represents the flattest area on the flat meridian. This flat zone is located on the corneal dial at 7:30 o'clock in this case, because the mires seen in the keratometer are the reverse of the corneal condition.

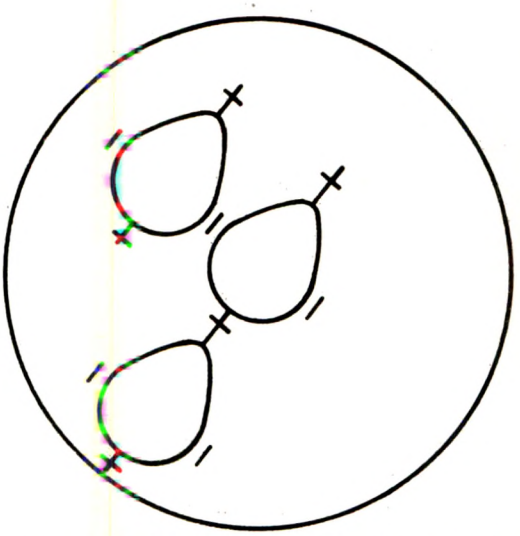


Fig. 1 (Hatch). Bausch and Lomb keratometer mires indicate flat area on cornea at the 7:30 o'clock position.

The proper-sized trephine is gently impressed on the cornea in order to outline the area to be treated. A Hildreth cautery is used to apply the surface thermal applications over a three clock-hour quadrant just inside the outlined imprint of the recipient button. This is done in the flattest zone of the flattest meridian (6 to 9 o'clock in this case) in order to steepen it (Fig. 2).

If the cylinder is 6 to 8 diopters or greater, two thermal wedges are done (6 to 9 o'clock and 12 to 3 o'clock in this example), 180 degrees opposite each other on the flat meridian (Fig. 3).

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TABLE

RESULTS OF THERMAL WEDGE WITH PENETRATING KERATOPLASTY TO REDUCE HIGH CORNEAL CYLINDER

Case No., Age (yrs)	Diagnosis	Diameter, Eye, Date of Surgery	Best Visual Acuity Before Surgery	Midpoint of Quadrant(s) Treated	Keratometer Before Surgery
1, 74	Bullous keratopathy	7.5 mm, RE, 10/15/75	6/30 (20/100)	7:30 o'clock	41.00 × 50 degrees 45.00 × 140 degrees
2, 50	Scarred graft, aphakia	7.5 mm, RE, 10/22/74	6/18 (20/60)	6 o'clock	42.00 × 85 degrees 55.00 × 175 degrees
51	Scarred graft, early cataract	7.5 mm, LE, 11/19/75	6/30 (20/100)	3:30 o'clock	38.50 × 162 degrees 53.00 × 72 degrees
3, 28	Anterior membrane dystrophy	7 mm, LE, 3/1/76	6/18 (20/60)	3 and 9 o'clock	45.50 × 180 degrees 49.50 × 90 degrees
29	Anterior membrane dystrophy	7 mm, RE, 12/14/77	6/60 (20/200)	3 and 9 o'clock	45.00 × 180 degrees 49.50 × 90 degrees
4, 56	Clear graft with high cylinder, cataract	7.5 mm, LE, and lens extraction, 6/24/76	Light perception and projection	1 and 7 o'clock	39.00 × 55 degrees 49.50 × 145 degrees
5, 47	Clear graft with wound edge slip	7.5 mm, LE, 9/22/76	6/12 (20/40) with −15.50 +6.00 × 10 degrees	5:45 o'clock	51.50 × 115 degrees 55.00 × 25 degrees
6, 61	Old keratitis	7 mm, RE, and lens extraction, 9/23/76	6/120 (20/400)	3 and 9 o'clock	43.00 × 180 degrees 48.00 × 90 degrees
7, 29	Bullous keratopathy, aphakia, nystagmus	6 mm, LE, 7/1/77	Counting fingers at 4 feet	4 and 10 o'clock	45.00 × 170 degrees 53.00 × 80 degrees

After the application of the cautery, a penetrating keratoplasty is performed.

RESULTS

The Table contains a summary of the results of all patients operated with this new procedure over a three-year period. The flat axis changed 30 degrees or less in eight of the nine eyes operated.

Case 5 represents an overcorrection with the flat axis shifting 60 degrees (115 to 55 degrees), and an actual cylinder

Keratometer After Surgery	Diopters Cylinder Increase or Decrease	Postoperative Correction (Contact Lens or Spectacles)	Visual Acuity After Surgery
35.00 × 35 degrees 42.50 × 125 degrees	+3.50	40.00 + 7.00	6/7.5 (20/25)
42.75 ×105 degrees 47.75 × 15 degrees	−8.00	44.00 +12.50 or +7.00 +5.25 × 10 degrees	6/9 (20/30)
38.00 × 160 degrees 44.00 × 70 degrees	−8.50	39.00 +4.00	6/9 (20/30)
44.00 × 150 degrees 45.50 × 60 degrees	−2.50	+1.50 +.75 × 170 degrees	6/6 (20/20)
45.75 × 150 degrees 48.25 × 60 degrees	−2.00	+2.00 +3.50 × 150 degrees	6/6 (20/20)
45.50 × 70 degrees 49.00 × 160 degrees	−7.00	+2.00 +3.50 × 150 degrees	6/12 (20/40)
45.25 × 55 degrees 48.50 × 145 degrees	−.25 (actually near −6.00) −see text	−4.00 +1.50 × 145 degrees	6/7.5 (20/25)
37.00 × 165 degrees 40.50 × 75 degrees	−1.50	39.00 +18.00	6/9 (20/30)
49.50 × 180 degrees 51.50 × 90 degrees	−6.00	−3.50 +2.00 × 90 degrees	6/120 (20/400)

change of about 6.0 diopters. The keratometry readings in the 50s were reduced to the more normal mid-40s postoperatively. The patient was satisfied with the cylinder portion of the spectacle correction of only 1.5 diopters.

In Case 1 the cylinder increased from 4.0 diopters preoperatively to 7.5 diopters postoperatively. The graft is clear and vision improved greatly, despite an increase rather than the decrease of cylinder.

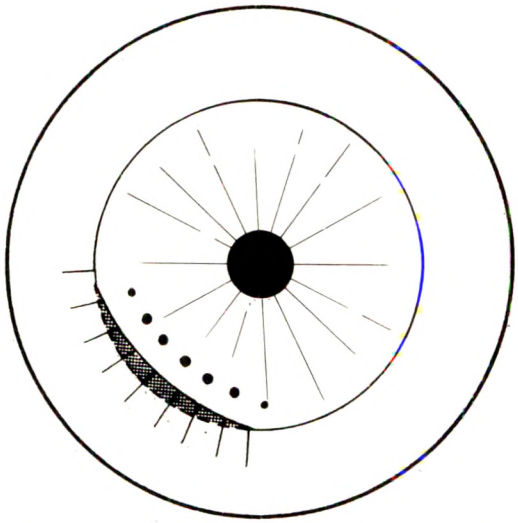


Fig. 2 (Hatch). Thermal wedge centered at the 7:30 o'clock position. The dots indicate thermal cautery applications. The shaded area represents the crescent wedge of corneal tissue drawn into the diseased button to be excised.

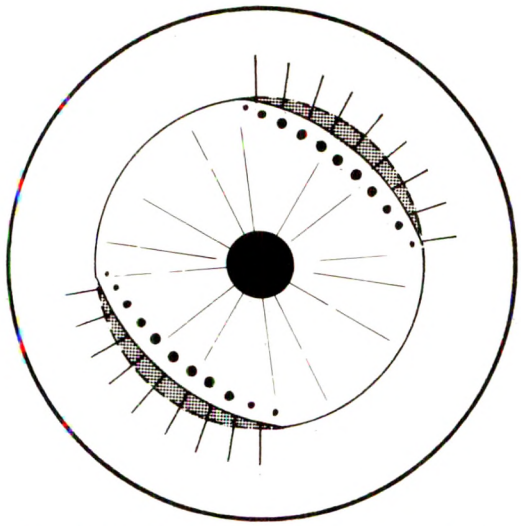


Fig. 3 (Hatch). Double thermal wedge. The dots indicate thermal cautery applications. The shaded areas represent crescentic wedges of corneal tissue drawn into the diseased button to be excised.

Final visual acuity was 6/12 (20/40) or better in all cases except for Case 7. In this case the patient had congenital cataract, nystagmus, and amblyopia. However, there was an improvement in visual acuity from counting fingers at 4 feet to 6/120 (20/400). Corneal cylinders were reduced as much as 8.5 diopters. All patients had postoperatively improved visual acuities with spectacles or contact lenses.

Discussion

I have treated several cases of high cylinder error after corneal transplant using surface thermal cautery only. This was done at the healed wound at the appropriate quadrant. In these instances I easily obtained the expected results of correction or some overcorrection without permanent injury to the graft or recipient, but the keratometry always reverted to pretreatment findings in a few weeks or months. The simplicity of the new thermal procedure is that a crescentic wedge of recipient tissue is drawn into the recipient button and permanently excised with a round trephine at surgery (Figs. 2 and 3). Donor tissue was obtained with the same trephine.

The ultimate effect of the single thermal wedge is like putting a round graft into a site that is pear-shaped anteriorly and round posteriorly. A double thermal wedge procedure is like putting a round graft into a site with an oval anterior opening and a round posterior opening.

All patients with high corneal cylinders before this operation showed marked reduction in cylinder without overcorrection after thermal wedge, plus graft. This contrasts with an actual surgical tissue (not thermal) wedge resection combined with penetrating keratoplasty, which often has unpredictable results and occasional marked overcorrection. This degree of predictability is a most pleasant development in the corneal surgical field.

Barner[1] reviewed the surgical treatment of corneal astigmatism, but did not mention a thermal wedge procedure.

Summary

I devised a new technique using a thermal wedge resection at the time of keratoplasty to reduce excessive amounts of corneal astigmatism. Surface cautery was used to draw this thermal wedge of corneal tissue into the area to be trephined and removed at surgery from the patient's cornea. The final result was the same as combining a surgical tissue wedge resection with keratoplasty. This new technique has resulted in reduction of the astigmatic error by as much as 8.5 diopters.

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Fifty years ago this month in THE JOURNAL:

When Edward Nettleship had retired from practice, his broad interest in heredity led him to study the Coppock family, affected by a peculiar form of cataract which has been called by that family name, or is known as the Doyne cataract for the name of the observer who first drew attention to it. Nettleship went on studying other forms of familial cataract and glaucoma. Then hereditary night-blindness as observed in a French family was traced back more than two hundred years, with the help of a French priest and the parish registers. By Nettleship's work, ophthalmology was placed in the lead of other branches of medicine in the study of heredity; and he was honored by a special volume of the publications of the Galton Eugenics Laboratory. Marcus Gunn's observation and exhibition of the changes in the retinal vessels associated with arteriosclerosis, as his observations became appreciated, confirmed, and supplemented by others, have added to the value and importance of every ophthalmoscopic examination of a patient past middle life.

Edward Jackson, William Henry Carmalt

Am. J. Ophthalmol. 13:705, 1930

CORNEAL EPITHELIUM IN PENETRATING KERATOPLASTY

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Intact corneal epithelium is essential to postoperative wound healing and graft transparency after penetrating keratoplasty.[1,2] Techniques in keratoplasty vary regarding the maintenance of donor epithelium at the time of the surgical procedure. Some surgeons believe that the donor epithelium should be removed at the time of surgery, whereas others believe the donor epithelium should be kept intact, especially when the host corneal epithelium is unhealthy.[3] Even if donor epithelium remains on the cornea during surgery, studies have been done that indicate the epithelium is frequently nonviable and may disintegrate during the early postoperative days.[4]

A factor that may influence donor epithelial integrity and viability is the method of corneal storage. When human eyes were studied after moist chamber storage for six hours, the donor corneal epithelium was found to be tattered and denuded in areas of mild trauma.[5] When tissue was transplanted immediately after enucleation in rabbits, donor corneal epithelium was found to be retained for many months after keratoplasty.[6] The study suggested that such fresh tissue was necessary to prevent loss of the epithelial layer.

Studies of corneal epithelial preservation using rabbit eyes in the laboratory have indicated that corneal epithelium could be maintained in a normal state if the donor eye was cooled promptly after death.[7] Metabolic requirements were thereby sharply reduced, prolonging the life of the stored epithelium. Additionally, when the rabbit cornea was removed from the depleted aqueous humor of the donor eye and placed in a nutrient-rich medium (McCarey-Kaufman corneal bathing medium) at 4°C, donor epithelium remained viable for as long as 12 days.[8]

There has been a scarcity of published clinical data regarding maintenance of donor corneal epithelium during times of storage and keratoplasty in humans. In one clinical study, donor corneas were stored in a moist chamber before keratoplasty. The graft epithelium was absent after surgery. Subsequent healing was accomplished by the fifth or sixth postoperative day.[9] Upon review of our records of patients undergoing keratoplasty, we found that the graft epithelium in most of our patients was healed by the second postoperative day. An obvious difference in techniques was that we used McCarey-Kaufman corneal bathing medium for donor corneal storage and used no moist chamber storage.

We report the results of a prospective clinical study in which we evaluated donor corneal epithelium at the time of McCarey-Kaufman storage, at transplantation, and postoperatively to determine if the donor epithelium could remain intact throughout the storage and surgical procedures. We also studied the number of days required for the epithelium to heal postoperatively when donor epithe-

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lium was missing at the time of keratoplasty. For uniformity of patient population, we studied patients with bullous keratopathy, the most frequent diagnosis for which penetrating keratoplasty is performed here. Although these patients have extensive preoperative epithelial edema, postkeratoplasty epithelial healing has not been a major clinical problem. Thus, a homogeneous series of bullous keratopathy patients treated with penetrating keratoplasty using McCarey-Kaufman stored donor corneas was selected for this study.

Subjects and Methods

This was a prospective clinical study of 66 consecutive patients undergoing penetrating keratoplasty here between 1977 and 1979 for corneal edema resulting from cataract extraction or Fuchs' dystrophy with cataract. All surgery was performed by the same surgeon (R.F.M.) and followed postoperatively by the same observer (R.F.M.).

Donor tissue was stored in McCarey-Kaufman medium at 4°C as soon as possible after donor death, enucleation, and transportation to the Michigan Eye Bank. During surgery the donor graft was prepared by punching from the endothelial surface against a Teflon block with a disposable 8.0- or 8.5-mm trephine. The host bed was prepared by making a deep corneal groove with an 8.0-mm disposable trephine. The anterior chamber was entered with a razor-blade knife and the incision was completed with corneal scissors to produce minimal inward beveling of the incision. Anterior vitrectomy using a vitrectomy instrument was performed on most patients. The donor corneal button was sutured into the recipient bed with either continuous or interrupted 10-0 monofilament nylon suture carefully so as not to disturb the donor corneal epithelium. Donor epithelium was examined with the slit-lamp microscope at the time of storage and at the end of surgery with fluorescein and a cobalt blue filter under the operating microscope to determine if the epithelium was intact at these times. The operated eye was firmly patched at the end of the operative procedure.

At each daily postoperative dressing change the epithelium was examined with the slit lamp under fluorescein and a cobalt blue filtered light source. Topically administered prednisolone acetate, antibiotic, and cycloplegic drops were instilled and a pressure patch was reapplied until total graft healing was achieved. The day of epithelial healing of the graft was defined as that day when epithelium was found intact at the time of dressing change. If healing failed to proceed or regressed at any time postoperatively, despite pressure patching, a therapeutic soft contact lens was applied. The therapeutic lens was discontinued after healing was achieved, as determined by fluorescein examination after periodic removal of the therapeutic lens. After discharge from the hospital, the patient was seen at one week, three weeks, and then monthly until after suture removal. At each post-operative visit, the epithelium was examined as previously described. Follow-up time ranged from three to 26 months, with a median of 14 months.

Results

Examination of the donor corneas at the time of storage showed that 43 eyes had intact epithelium; 23 had 5 to 100% (median, 50%) epithelium missing. Examination of the donor corneas at the end of surgery showed that 16 of the grafts had epithelium intact, while 50 had 5 to 100% (median, 20%) epithelium missing. Thus, 27 of the donor corneas that had epithelium intact at the time of storage lost 5 to

TABLE 1

DONOR DATA OF EYES WITH CORNEAL EPITHELIUM INTACT AT STORAGE SHOWING NO SIGNIFICANT CORRELATION BETWEEN STORAGE TIMES AND LOSS OF EPITHELIUM AT SURGERY

Storage Time	Epithelium Intact at End of Surgery (16 Eyes)		Epithelium Not Intact at End of Surgery (27 Eyes)		
	Range (hrs)	Median (hrs)	Range (hrs)	Median (hrs)	P Value*
Death to enucleation	0.5–7.0	2.0	0.5–11.5	2.0	.86
Enucleation to storage	1.0–15.0	6.0	1.0–10.0	5.0	.25
Storage to use	7.0–63.0	41.0	7.5–62.5	27.0	.20
Total	18.5–79.0	48.0	15.0–77.5	36.0	.18

*Mann-Whitney U-Two Sample Comparison Test.

100% (median, 10%) epithelium by the end of the surgical procedure. Evaluation of the donor corneal storage data (Table 1) in donor corneas that had epithelium intact at the time of storage showed no statistically significant differences in the various time intervals between donors that still had epithelium intact and those that had lost epithelium by the end of surgery (Mann-Whitney U-Two Sample Comparison Test). Donor corneas could be stored as long as 79 hours, with 63 hours in McCarey-Kaufman medium, and still have epithelium intact at the end of the surgical procedure.

The number of days for grafts to heal postoperatively ranged from one to 12 days, with a median of two days in 63 of the 66 patients. The remaining three patients required therapeutic soft contact lenses for epithelial healing. Twenty-one of the grafts were healed on the first postoperative day, 40 were healed by the second day, 55 were healed by the third day, and 60 had epithelium intact by the fourth day after surgery. One graft required six days, another eight days, and another required 12 days to heal totally without contact lens therapy.

Of the 16 grafts with donor corneal epithelium intact at the end of the surgical procedure, nine still had graft epithelium intact on the first postoperative day and seven had 5 to 10% of peripheral graft epithelium missing in suture areas. The seven grafts with postoperative epithelial defects required two to four days (median, two days) to restore epithelial integrity.

Evaluation of postoperative epithelial healing time in relation to the presence or absence of intact donor epithelium at the time of surgery (Table 2) showed that within the group of 16 donor corneas with epithelium intact at the end of surgery, 14 healed on or before the second postop-

TABLE 2

POSTKERATOPLASTY EPITHELIAL HEALING TIME

Donor Epithelium Intact at Surgery	Days to Heal	
	Two Days or Less	More Than Two Days
Yes (16 eyes)	14 eyes	2 eyes
No (50 eyes)	26 eyes	24 eyes
Total (66 eyes)		

erative day, and two healed after the second day. In contrast, within the group of 50 donor corneas that had 5 to 100% (median, 20%) epithelium missing at the end of surgery, 26 healed on or before the second postoperative day, and 24 required more than two days to heal. The difference in healing times between the two groups was statistically significant (Mann-Whitney U-Two Sample Comparison Test, P=.03). Additionally, the amount of donor epithelium found to be intact at the end of surgery was statistically significant in relation to the number of days to heal postoperatively (Goodman-Kruskal Gamma Two-Way Cross-Tabulation Test). As the amount of epithelium intact at the end of surgery decreased, the number of days to heal postoperatively increased.

Other factors that were evaluated and found to have no apparent relationship to epithelial integrity included the donor and recipient patients' ages and sex, extent of host corneal edema, suture technique, disparate donor graft size, Schirmer tests, corneal sensation, diabetes, and glaucoma.

Discussion

Corneal epithelial healing is accomplished by the processes of cell migration and proliferation.[10,11] In the initial phase of healing, epithelial cells slide in an orderly fashion over the defect until it is closed without evidence of mitotic activity in the participating cells. Later, mitoses become abundant. The energy source for the healing activity is provided mainly from stored glycogen in the epithelial cells.[12] Epithelial glycogen levels decrease rapidly after one hour of storage in situ or after 48 hours' storage in a moist chamber; however, rejuvenation of glycogen stores occurs when the cornea is placed in a nutrient-rich medium.[8] As corneal epithelium becomes depleted of its metabolic stores, epithelial edema develops with eventual loss of cell viability.[7]

Transplantation of donor epithelium is of no value if the cells are not viable because they are promptly lost during, or soon after, surgery. We found that even though the donor epithelium appeared intact at the end of surgery, it was frequently absent in patches at the early dressing changes. Therefore, the viability of donor corneal epithelium could not be evaluated simply by assessment of its presence or absence at the end of the surgical procedure. However, such assessment was adequate to establish a significant relationship between the amount of epithelium on the donor button at the time of surgery and the length of time required for postoperative healing.

Other studies have shown that when donor corneas were stored in moist chambers, epithelium was easily removed by light rubbing of the surface with a gauze or cellulose sponge.[13] In the present study, donor corneal epithelium tended to be firmly adherent and resistant to removal by mild trauma. Although many donor corneas had lost epithelium by the end of the keratoplasty procedure, the amount lost was small. Postoperative epithelial loss was generally in the peripheral graft in the vicinity of sutures where graft surfaces were elevated and subject to increased trauma. It was not evident from the results of the present study why epithelium remained intact on some donor corneas throughout McCarey-Kaufman storage, surgery, and the postoperative period, and in others the donor epithelium did not remain intact. A factor that was not evaluated, but should be considered, was the method of punch technique with the epithelium lying on a Teflon block during preparation of the donor tissue at the time of keratoplasty. This may have induced epithelial loss because of punching trauma.

Abnormal tear film distribution, related to irregular graft surfaces in areas around sutures, was a factor common to a group of patients who had unusual postoperative epithelial healing problems. Included in this group were three patients who healed between the sixth and 12th postoperative days and three patients who required therapeutic soft contact lenses for healing. The graft irregularities took the form of surface depressions or elevations around suture sites, resulting in an unstable tear film and subsequent epithelial integrity problems.

The results of this study indicate that in corneas with chronic edema the length of time required for postoperative epithelial healing was directly related to the amount of donor corneal epithelium that remained intact throughout the storage and surgical periods. The length of time for grafts to heal postoperatively was consistently longer when donor epithelium was absent at the end of surgery. As the amount of epithelium intact at the end of surgery decreased, the number of days to heal postoperatively increased. The risk of postoperative complications, such as graft ulceration and opacification, tends to increase as the length of time of persistent epithelial defects increases.[14] The results of the present study may be even more significant when considering severely damaged host corneas, such as those suffering severe chemical burns, where absence of donor corneal epithelium may result in delayed healing with subsequent ulceration and opacification of the graft.

Summary

We studied corneal epithelium in 66 patients with bullous keratopathy treated with penetrating keratoplasty using McCarey-Kaufman stored donor corneas. Epithelium was evaluated at times of storage, surgery, and postoperative dressing changes.

Epithelium was intact in 43 of the donor corneas at storage, and 23 had 5 to 100% (median, 50%) epithelium missing. At the end of the keratoplasty procedure, 16 grafts had epithelium intact, and 50 had 5 to 100% (median, 20%) epithelium missing. Postoperative epithelial healing time ranged from one to 12 days, with a median of two days. Postoperative healing was significantly prolonged when donor corneal epithelium was missing at keratoplasty. As the amount of epithelium intact at the end of surgery decreased, the number of days to heal postoperatively increased. We found that donor corneas could be stored as long as 79 hours, with 63 hours in McCarey-Kaufman medium, and still have epithelium intact at the end of the keratoplasty procedure.

Acknowledgment

George W. Williams, Ph.D., provided statistical analysis of the data.

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Ophthalmic Miniature

Binocular Ophthalmoscope.—We owe to Dr. Giraud-Teulon, and following him, Mr. J. Z. Lawrence, our thanks for the invention of the binocular ophthalmoscope, by means of which we are enabled to save a great deal of time and uncertainty in determining the position of extravasations, exudations, deposits, etc., in the vitreous humor, retina and choroid. It is true, as Dr. Schweigger admits, that with the monocular ophthalmoscope it is possible to ascertain nearly all that the binocular instrument can reveal to us; but a moment's glance with both eyes is worth an hour's gaze with one. Probably in a short time ophthalmologists will use it to the exclusion of the monocular ophthalmoscope. A comparison of the two would be better than any description we can here give. Time will undoubtedly simplify as well as cheapen this instrument. It is, in the highest sense of the term, an advance upon our previous means of observation. Simply as a scientific or philosophical instrument, it will be of great value, as probably enabling us to settle many points in reference to binocular vision and so-called stereoscopic effect. It is a new means of experimenting in this field of physiological optics. Its general use will be looked forward to with interest by both the philosophical and pathological ophthalmologist.

Trans. Am. Ophthalmol Soc.
3:5, 1866

HISTOCOMPATIBILITY ANTIGEN FREQUENCIES IN DIABETIC RETINOPATHY

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AND

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The association of multiple histocompatibility specificities with insulin-dependent diabetes strengthens the genetic theory of the pathogenesis of this disorder, and strongly suggests extensive heterogeneity.[1-4] A possible genetic contribution to diabetic microangiopathy has rarely been studied, although there is some evidence that genetic factors play a role in its pathogenesis.[5] Few studies have analyzed histocompatibility antigen frequencies in relation to diabetic eye disease.[6-8] None of these studies has compared large, matched groups, with and without proliferative retinopathy. If an indicator of proliferative retinopathy could be identified early in the course of the disease, improvement of the patient's metabolic control could result in better prognosis.

In the present study we compared the histocompatibility antigen frequencies in patients with insulin-dependent diabetes that differed only in the presence or absence of proliferative retinopathy.

SUBJECTS AND METHODS

Nonproliferative diabetic retinopathy group—One hundred insulin-dependent diabetic patients were studied. All of them had diagnosis of disease before age 40 years. The patients were recruited from diabetes clinics at the University of Minnesota and from area physicians on the basis of minimal retinopathy after long duration of insulin-dependent diabetes. Each patient underwent a complete ophthalmic examination by us. The vitreoretinal examination and grading of retinopathy was done by a member of the Retina Service. All patients in this group had background or nonproliferative retinopathy (microaneurysms, retinal hemorrhages, exudates) and no evidence of neovascularization. These patients were chosen in such a way that they matched for duration of disease with the patients with proliferative retinopathy.

Proliferative diabetic retinopathy group—Two hundred insulin-dependent diabetic patients were studied. All of them had diagnosis of disease before age 40 years. All patients were recruited from the Retina Clinic, University of Minnesota. Proliferative retinopathy was documented in each case by a dilated ophthalmoscopic examination by a member of the Retina Service; 105 patients in this group participated in the Diabetic Reti-

From the Departments of Medicine (Division of Endocrinology and Metabolism) (Drs. Barbosa and King and Ms. Noreen) and Ophthalmology (Drs. Ramsay, Knobloch, and Cantrill), University of Minnesota; and the Department of Pathology, Harvard University School of Medicine (Dr. Yunis), Boston, Massachusetts. This study was supported by Grants 1 R01 EY02445 (Dr. Ramsay), 1 R01 AM20729 (Dr. Barbosa), General Clinical Research Centers Program (RR-400), and National Institutes of Health Contract N01-HD-8-2846 (Dr. Barbosa).

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nopathy Study at the University of Minnesota.

Control group—A total of 847 healthy, local blood donors from Minnesota underwent histocompatibility typing and served as a control group.

Population characteristics—All patients and blood donors were white and were from the five-state upper midwest region of the United States (Minnesota, Wisconsin, Iowa, North Dakota, and South Dakota). Data recorded for each patient included age, sex, age at diagnosis of diabetes, history of diabetes mellitus in a first-degree relative, history of episodes of ketosis, and history of neuropathy and nephropathy.

HLA typing—Fifty-one histocompatibility antigens of the A and B series were determined with a standard two-stage dye exclusion microcytotoxicity test,[9] using a panel of 90 well-characterized antisera capable of detecting all defined histocompatibility and most "w" specificities. Differences in histocompatibility antigen frequencies between the groups were studied by Chi-square analysis. When appropriate the P values obtained were corrected by multiplying them by the number of comparisons performed. The relative risks were calculated as equal to (patients positive/patients negative) × (controls negative × controls positive).

Results

The mean age was 43.1 years for the nonproliferative retinopathy group and 35.8 years for the patients with proliferative retinopathy. The nonproliferative retinopathy group contained 54 males and 46 females and the proliferative retinopathy group contained 114 males and 86 females. The mean age at onset of disease was 17.3 years in the nonproliferative retinopathy group and 13.7 years in the proliferative retinopathy group. The mean known duration of diabetes at the time of tissue typing was 25.8 years in the nonproliferative retinopathy group and 22.1 years in the proliferative retinopathy group. A history of diabetes mellitus in a first-degree relative was present in 34% (34 of 100) of the patients with nonproliferative retinopathy and 37.5% (75 of 200) of the patients with proliferative retinopathy. Neuropathy was present in 32% (32 of 100) of the patients with nonproliferative retinopathy, and in 30% (60 of 200) of the patients with proliferative retinopathy. Nephropathy was present in 24% (24 of 100) of the patients in the nonproliferative retinopathy group, and 32% (64 of 200) of the patients in the proliferative retinopathy group. Sixty-six percent (132 of 200) of the proliferative retinopathy group and 72% (72 of 100) of the nonproliferative retinopathy group reported ketosis. No significant differences were found between the two groups for these data.

Table 1 shows histocompatibility antigen frequencies obtained in the control population and in the two diabetic groups. The antigens are those that have previously been shown to have abnormal frequencies in insulin-dependent diabetic patients. Other A and B loci antigens studied were present in normal frequencies and are not shown. We did not find an increased frequency of the phenotype X/B8 (X being an antigen other than A_1) in patients with proliferative retinopathy, as reported by others.[10] When compared to the healthy controls, both diabetic groups showed a significantly increased frequency of HLA-B8, HLA-B15, HLA-B18, and the phenotype HLA-B8/HLA-B15, and a decreased frequency of HLA-B7 and HLA-B12. The comparison between the two diabetic groups shows a significant difference for HLA-B7 ($\chi^2=10.0$; $P_c<.03$). Thus a diabetic who is HLA-B7 positive is about three times less

TABLE 1

HLA FREQUENCIES IN DIABETICS WITH PROLIFERATIVE RETINOPATHY AND WITHOUT PROLIFERATIVE RETINOPATHY, MATCHED FOR DURATION OF DISEASE*

Antigens	Control, % (N=847)	NPR, % (N=100)	PR, % (N=200)	NPR vs Control		PR vs Control		NPR vs PR	
				χ^2†	RR	χ^2	RR	χ^2	RR
HLA-B7	31.5	20	7.0	5.1‡	0.54	48.3§	0.16	10.0‖	0.30
HLA-B8	21.3	43	45.0	22.3§	2.81	46.5§	3.04	NS	
HLA-B12	30.0	9	11.0	18.6§	0.23	29.1§	0.29	NS	
HLA-B15	18.8	27	29.5	3.8	1.60	11.3§	1.81	NS	
HLA-B18	5.5	13	16.0	7.2¶	2.54	23.9§	3.24	NS	
HLA-B8/HLA-15	1.5	12	9.5	34.2§	8.75	32.0§	6.73	NS	

* NPR designates nonproliferative retinopathy; PR, proliferative retinopathy; RR, relative risk; and NS, not significant.
† Chi-square statistics.
‡ P<.025.
§ P<.001.
¶ P<.01.
‖ P<.02 (corrected).

likely to develop proliferative retinopathy than a patient who is HLA-B7 negative.

To further characterize histocompatibility antigen differences that may exist between the diabetic groups, the patients were separated by age at diagnosis (Table 2). For diagnosis of disease from age 0 to 14 years there were 45 patients with nonproliferative retinopathy and 129 patients with proliferative retinopathy. For diagnosis between age 15 and 40 years there were 55 patients with nonproliferative retinopathy, and 71 patients with proliferative retinopathy. The duration of disease remained similar for the two groups. As in the total matched group, HLA-B7 is significantly less frequent in the proliferative retinopathy group with age at diagnosis between 15 and 40 years (χ^2=8.0; P_c<.03). Additionally, HLA-B15 is significantly more frequent (χ^2=7.89; P_c<.03) for the proliferative retinopathy group in the same subset

TABLE 2

HLA FREQUENCIES IN DIABETICS WITH PROLIFERATIVE RETINOPATHY AND WITHOUT PROLIFERATIVE RETINOPATHY AND DIAGNOSIS OF DISEASE BEFORE AGE 15, AND BETWEEN AGES 15 AND 40 YEARS*

Antigens	Age at Diagnosis 0-14 years					Age at Diagnosis 15-40 years					
	NPR (N=45)		PR (N=129)			NPR (N=55)		PR (N=71)			
	No.	%	No.	%	χ^2†	No.	%	No.	%	χ^2	RR
HLA-B7	7	15.6	10	7.8	NS	13	23.6	4	5.6	8.0‡	0.19
HLA-B8	22	48.9	57	44.2	NS	21	38.2	33	46.5	NS	
HLA-B12	2	4.4	12	9.3	NS	7	12.7	10	14.1	NS	
HLA-B15	18	40.0	31	24.0	NS	9	16.4	28	39.4	7.89‡	3.32
HLA-B18	4	8.9	23	17.8	NS	9	16.4	9	12.7	NS	
HLA-B8 / HLA-B15	9	20.0	8	6.2	NS	3	5.5	11	15.5	NS	

* NPR designates nonproliferative retinopathy; PR, proliferative retinopathy; RR, relative risk, and NS, not significant.
† Chi-square statistics.
‡ P <.03 (corrected).

of patients. When comparing these two groups of patients the relative risk for HLA-B7 is 0.19 and for HLA-B15 is 3.32. Thus, for the subset of patients with disease onset between age 15 and 40 years, the diabetics who are HLA-B7 positive are five times less likely, and the diabetics who are HLA-B15 positive are three times more likely to develop proliferative retinopathy than diabetics with other antigens.

Discussion

Patients who develop insulin-dependent diabetes before the age of 40 years have a greater than 50% chance of developing proliferative retinopathy after a duration of disease of 30 years or more.[11] Conversely, a significant percentage of patients do not develop proliferative changes even after a duration of disease of 30 to 40 years. While hyperglycemia is generally recognized as an important factor in the development of the duration-related microvascular complications of diabetes, attempts to correlate diabetic control with microangiopathy in long-term survivors have been inconclusive.[12] We have studied histocompatibility antigens in patients with proliferative retinopathy, because an association with the histocompatibility complex would indicate a genetic contribution to microangiopathy. The patients in the two groups were matched for several clinical features, especially duration of disease, which is the best established risk factor for diabetic retinopathy. Although all patients with nonproliferative retinopathy were found to be free of proliferative retinopathy, possibly some may later develop neovascularization. We found the expected increments in the frequency of HLA-B8, HLA-B15, and HLA-B8/HLA-B15 and decrements of HLA-B7 and HLA-B12 when comparing insulin-dependent diabetic patients with controls. Additionally, the patients in the proliferative retinopathy group showed a further deficit of HLA-B7, which resulted in a significant difference between patients with nonproliferative retinopathy and patients with proliferative retinopathy for this antigen. Because an upward trend of HLA-B15 in the proliferative group was present (Table 1), we stratified both groups for age at diagnosis. Only in those with diagnosis of disease between 15 and 40 years of age is HLA-B15 associated with proliferative retinopathy. Thus, the patients with proliferative retinopathy have an excess of HLA-B15 independently of age at diagnosis, whereas those without show an excess only when diabetes is diagnosed in the first 15 years. These deviations of histocompatibility antigen frequencies do not, of course, indicate an absolute risk (HLA-B15) or protection (HLA-B7) but only a statistical probability indicated by the relative risks given previously. For the patients with onset of disease between age 15 and 40 years, for instance, those with HLA-B7 have a risk for proliferative retinopathy only one fifth as large as those patients without HLA-B7. The value of these antigens as markers for proliferative retinopathy depends, however, on their frequency in the background population. In populations in which HLA-B7 is rare, such as American Indians (2%) or Arabs (6%), its value would be limited.

Several other groups have studied histocompatibility in patients with retinopathy.[6-8] Becker and associates[6] reported an increase of HLA-B8 and HLA-A in diabetics with onset of disease after age 30 years only when retinopathy is absent. The stage of retinopathy, however, was not defined, making comparison with our study difficult. Others[7] compared 12 patients with proliferative retinopathy and 31 patients with severe nonproliferative retinopathy and showed no significant relationship, although a trend for HLA-B15

compatible with our results was present. A similar trend can be seen in a recent report,[8] although the number of patients was too small to achieve statistical significance.

Our findings indicate the existence of histocompatibility-associated genetic factors that play a role in proliferative retinopathy. The mechanism by which these genetic factors influence retinopathy is not known. Preliminary studies have suggested associations of HLA-B15 with high insulin antibody titers,[13] and immune complexes with retinopathy.[14] Whether these mechanisms are related to the association with the histocompatibility complex is not clear.

The finding of a histocompatibility-associated risk factor for proliferative retinopathy may have an important influence on the management of patients with insulin-dependent diabetes. Improved methods of diabetic control and medical treatment could be used in this high-risk group in an attempt to delay or prevent proliferative retinopathy. Additionally, the identification of this group would indicate the necessity of repeated ophthalmic examinations and the use of photocoagulation therapy before the development of the complications of proliferative retinopathy. Future controlled clinical trial testing treatments of proliferative diabetic retinopathy should take these results into consideration.

Summary

We studied the histocompatibility antigens A and B in 300 insulin-dependent diabetics: 200 had proliferative retinopathy and 100 did not. The two groups were matched for known duration of diabetes and other clinical features. In both groups the frequencies of HLA-B8, HLA-B18, and HLA-B8/HLA-B15 were significantly higher, and those of HLA-B7 and HLA-B12 were significantly lower than in healthy controls. The patients with proliferative retinopathy were significantly less often positive for HLA-B7 ($\chi^2 = 10.0$; $P_c < .03$) than patients with nonproliferative retinopathy. When both groups were stratified for age at diagnosis, there were additional differences. HLA-B15 was significantly more frequent in the proliferative retinopathy group with age at diagnosis between 15 and 40 years (nonproliferative retinopathy = 16.4%; proliferative retinopathy = 39.4%; $\chi^2 = 7.89$, $P_c < .03$; relative risk = 3.32) and HLA-B7 significantly less frequent (nonproliferative retinopathy = 23.6%; proliferative retinopathy = 5.6%; $\chi^2 = 8.0$, $P_c < .03$; relative risk = 0.19). These differences in histocompatibility frequencies between patients with and without proliferative retinopathy indicate a genetic contribution to diabetic retinopathy.

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Ophthalmic Miniature

There was no flour on Ruth's hands when she received them in the triangular parlor, but there were pleasant smiles upon her face, and a crowd of welcomes shining out of every smile, and gleaming in her bright eyes. By-the-bye, how bright they were! Looking into them for but a moment, when you took her hand, you saw, in each, such a capital miniature of yourself, representing you as such a restless, flashing, eager, brilliant little fellow—

Ah! if you could only have kept them for your own miniature! But, wicked, roving, restless, too impartial eyes, it was enough for any one to stand before them, and straightway, there he danced and sparkled quite as merrily as you!

F. G. de Fontaine
The Fireside Dickens
New York, Dillingham, 1888

CONGENITAL BILATERAL ARTERIAL ANASTOMOSIS BETWEEN THE CHOROID AND PERIPHERAL RETINA

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Nijmegen, The Netherlands

Vascular anastomoses between the choroid and retina may be acquired or congenital. Acquired anastomoses have been described in conditions associated with damage of the pigment epithelium and Bruch's membrane, such as toxoplasmic retinitis,[1-3] senile disciform degeneration of the macula,[4] photocoagulation,[5] and trauma.[6,7]

Congenital anastomoses occur as cilioretinal arteries, and opticociliary arteries and veins at the disk, but these have also been reported in a few cases in atypical colobomas[8,9] and at the ora serrata.[10]

We discovered a bilateral anastomosis between a choroidal artery and dilated retinal vessels in the inferior periphery of the fundus of a woman who had a macular pucker and small exudative retinal detachments. To the best of our knowledge, a similar condition has not been described previously.

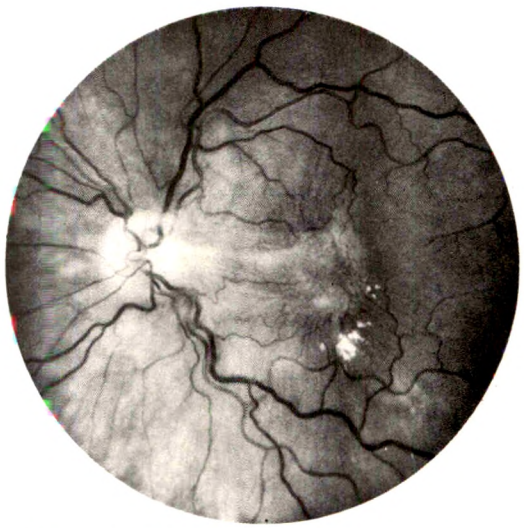


Fig. 1 (van Nouhuys and Deutman). Left eye. Preretinal fibrosis is present at the macula with retinal traction lines radiating from it. There are small exudates.

CASE REPORT

A 26-year-old woman was referred to us two weeks after she noticed blurred vision in the left eye. Her general medical history was unremarkable. At the initial examination in March 1979 her visual acuity was R.E.: 6/4.5 (20/15) and L.E.: 6/48 (20/160). Cornea, anterior chamber, and iris of both eyes were normal. The lenses were clear. With a fully dilated pupil the inferior edges of both lenses were visible from the 4- to 8-o'clock positions, where they were somewhat irregular. No gap in the zonules could be observed. A few punctate opacities could be seen in the vitreous of both eyes. In the posterior pole of the left eye gray preretinal fibrosis and a few small exudates were visible (Fig. 1). The posterior pole of the right eye was normal. In both eyes a local and not prominent exudative detachment of the neuroretina was seen in the inferior periphery, extending centrally toward the equator. The retinal vessels in the region of the detachments in both eyes were deformed and dilated and accompanied by a few small exudates and hemorrhages (Figs. 2 and 3). Both fundi of this woman did not have much pigmentation and the choroidal vessels could easily be observed. The electroretinogram and electro-oculogram were normal and color vision was within normal limits in both eyes.

Fluorescein angiography of the left posterior pole clearly showed the distortion of the paramacular vessels. In the late phases of angiography an intensive leakage was visible at the site of the peripheral detachments obscuring details of the retinal vessels (Fig. 4).

Our patient was hospitalized for clinical observation, because we could not make a definite diagnosis at that time. All general and laboratory examinations gave normal results and included the following: blood sedimentation rate, complete blood cell count, serum electrolytes, glucose, blood urea nitrogen, alkaline phosphatase, Rose and latex test, agglutination tests for toxoplasmosis, VDRL, serum protein electrophoresis, hematologic examinations for abnormal hemo-

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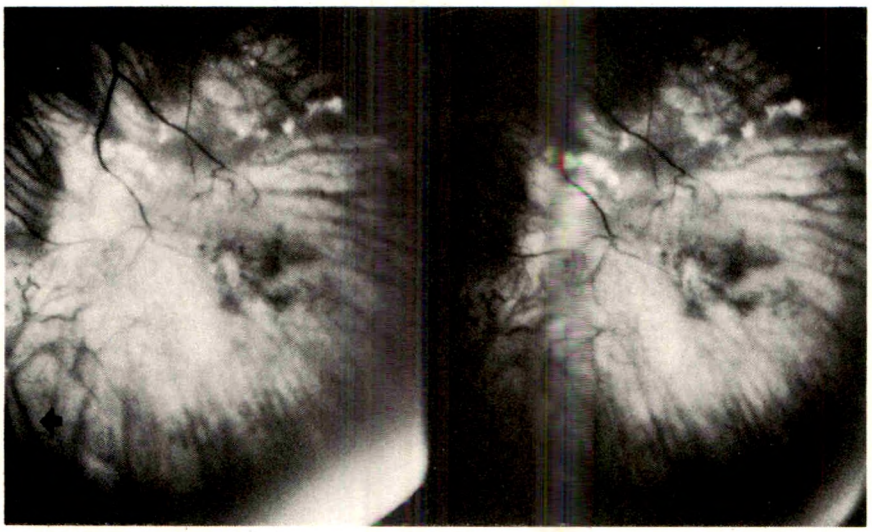


Fig. 2 (van Nouhuys and Deutman) Stereoscopic photographs of inferior equatorial area of left fundus. The retina peripheral to the equator shows an exudative detachment, small hemorrhages, and deep exudates. The vessels in the detached retina are abnormal and show irregular dilatations and hairpin-like loops. Note the choroidal artery (arrow). Use +6 diopter lenses for stereoscopic observation.)

globins, and X-ray film examinations of chest and paranasal sinuses. Four sisters and one brother of our patient were examined and they did not show any fundus abnormality. Her mother had diabetic retinopathy. Because an inflammatory cause was considered as a possibility, 60 mg of prednisone was given orally daily. No improvement of visual acuity or retinal lesions was observed and the daily dose of corticosteroids was quickly diminished and discontinued after six weeks. Ultrasound examination was performed in both eyes, but the detachments were too small to be shown by A- or B-scan. Otherwise results of this examination were normal.

Fluorescein angiography was repeated and we tried to make a rapid series of exposures of the early phase of the inferior retinal periphery of the right

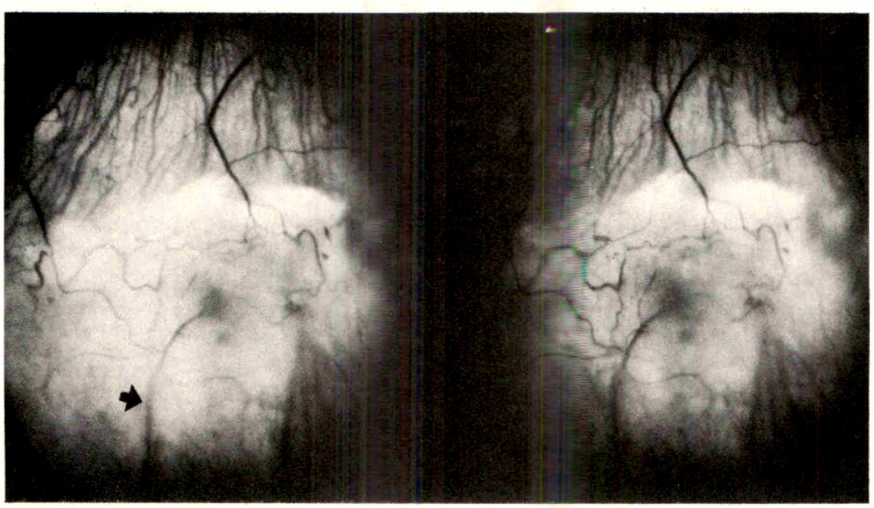


Fig. 3 (van Nouhuys and Deutman). Stereoscopic photographs of inferior equatorial area of right eye. The peripheral retina also shows some serous detachment, exudates, hemorrhages, and aberrant vessels. Note choroidal artery (arrow).

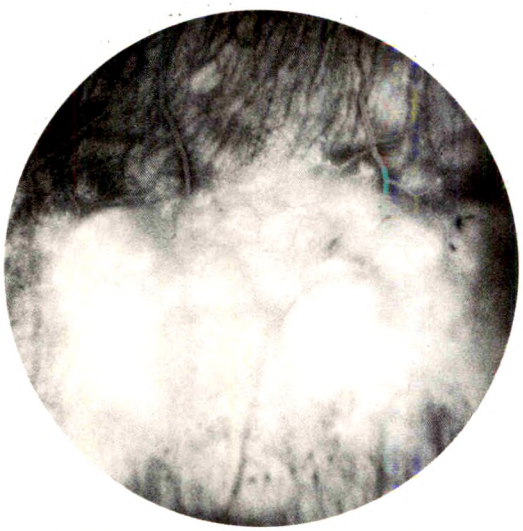


Fig. 4 (van Nouhuys and Deutman). Right eye. Fluorescein angiogram. Staining of subretinal fluid by fluorescein in late phase.

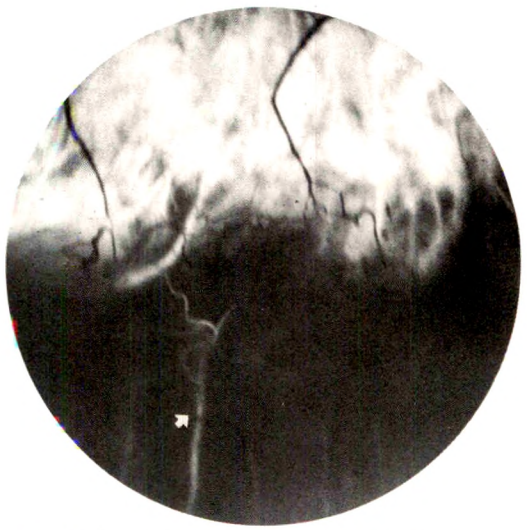


Fig. 5 (van Nouhuys and Deutman). Right eye. Fluorescein angiogram of periphery at 6-o'clock position. Arterial phase demonstrates absence of choriocapillaris under peripheral retina. The choroid and retina central to the equator are normal. A choroidal artery is filled with dye in a centripetal direction (arrow). Compare with Figure 3.

eye. On this angiogram a definite absence of the choriocapillaris could be seen under the detached retina. One choroidal artery appeared to be filled by fluorescein in a centripetal direction (Fig. 5). Near the equator this artery gave off several branches, which were connected directly with dilated and abnormal retinal vessels (Fig. 6). From these dilated vessels a profuse leakage stained the retina and subretinal fluid rapidly. After comparing the angiogram with the stereoscopic photographs (Fig. 3) of the same region the vascular anastomosis between choroid and retina could be observed better. The arterial phase of the fluorescein angiogram of the left fundus also showed absence of the peripheral choriocapillaris at the 6-o'clock position (Fig. 7). A large choroidal artery was filled with dye from the periphery. Several branches of this artery were connected with aberrant vessels in the equatorial retina. The arteriovenous phase (Fig. 8) showed connections of these vessels with the normal-appearing retinal arterial and venous branches.

During the ten months since her first visit to our outpatient clinic the patient's visual acuity and fundus lesions have not changed and no other therapy has been given.

Discussion

The choroidal arteries forming the anastomosis in both eyes of our patient are clearly recurrent ciliary arteries. The blood flow came from the anterior choroid in a central direction, as could be

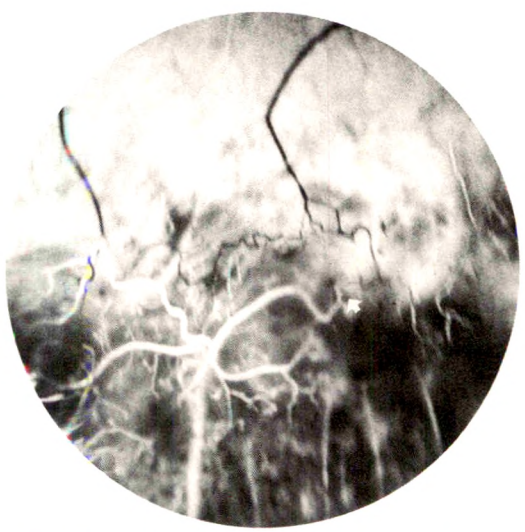


Fig. 6 (van Nouhuys and Deutman). Right eye. Fluorescein angiogram of the late arterial phase. Several branches of choroidal artery are filled with fluorescein. Some of them show communications through small aberrant vessels with retinal veins (arrow). Note also small retinal vein twisted around branch of choroidal artery (left). Compare with Figure 3.



Fig. 7 (van Nouhuys and Deutman). Left eye. Fluorescein angiogram of inferior periphery. Arterial phase shows absence of choriocapillaris peripheral to the equator and large choroidal artery filled with dye in centripetal direction. Several branches of this artery are already visible. Compare with Figure 2.

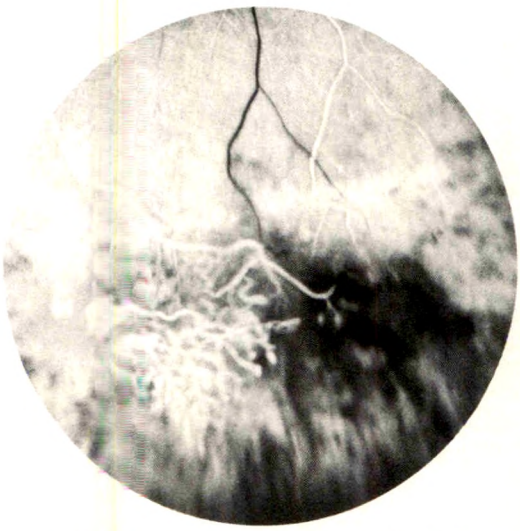


Fig. 8 (van Nouhuys and Deutman). Left eye. Arteriovenous phase of fluorescein angiogram of inferior periphery. Network of aberrant vessels in retina shows connections with retinal artery and vein. Note disappearence of dye from choroidal artery.

seen on the angiogram. Generally it is not possible to discover these peripheral vessels clearly by fluorescein angiography, but in our case the observation was possible, because of the localized absence of the choriocapillaris and retinal pigment epithelium. The bilateral inferior position and the absence of the choriocapillaris suggest that the primary congenital anomaly is a partial typical coloboma of the anterior choroid and retinal pigment epithelium. Partial colobomas of the choroid always show a defect in the choriocapillaris, Bruch's membrane, and retinal pigment epithelium,[11] but the layer of the larger choroidal vessels may be intact, as in our case.

We have not observed a gap in the ciliary body when ophthalmoscopy with indentation was performed, nor did we see any pigmentations bordering the defect of the choriocapillaris. After complete dilatation of the pupils the inferior edge of both lenses could be observed (Fig. 9). This phenomenon may be a symptom of a minimal coloboma of the lens. However, we could not observe a gap in the zonules, something usually associated with such colobomas.

The defect in Bruch's membrane and pigment epithelium creates a continuity between the choroid and retina, through which developing vessels may grow. Vascular anastomoses associated with a coloboma have been reported incidentally in cases of atypical colobomas of the posterior pole.[8,9]

In our case a recurrent ciliary artery appears to be involved. This is the last vessel in the embryologic development of the choroid. The formation of these arteries, originating from the primary arterial circle of the iris and supplying the anterior part of the choroid, takes place from the sixth to the ninth month of intrauterine life.[12] Finally, the recurrent ciliary arteries growing in a posterior direction form anastomoses with the posterior cili-

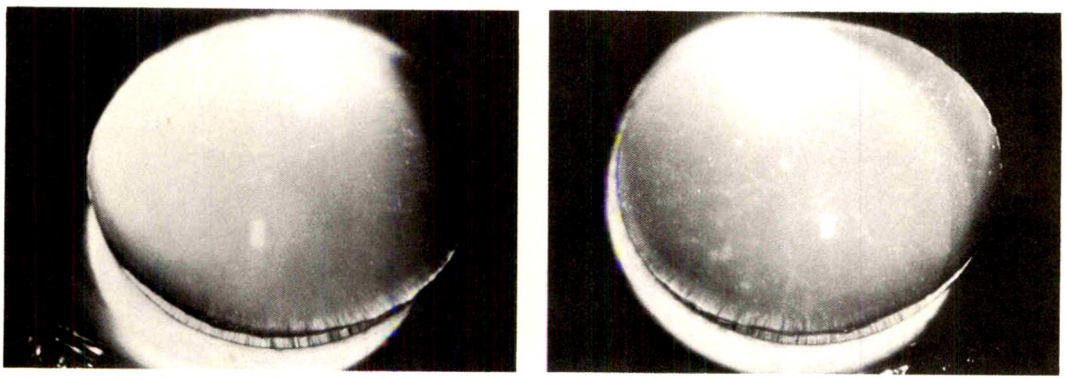


Fig. 9 (van Nouhuys and Deutman). Slit-lamp photograph with retroillumination of right eye (left) and left eye (right). With a fully dilatated pupil the inferior edges of both lenses are visible from the 4- to 8-o'clock position. There is no gap in the zonules.

ary vessels at the equator. There has been much speculation regarding the nature of this anastomosis. François, Neetens, and Collette[13] and Heimann[12] could not show arterial anastomoses at the equator, but Wybar,[14] Ring and Fujino,[15] and Weiter and Ernest[16] showed arterial anastomoses between the recurrent ciliary arteries and the posterior choroidal arteries in neoprene casts of human eyes. We assume that in our patient, in the last months of intrauterine life, a developing recurrent ciliary artery could not make the usual connection with the choriocapillaris, because of its absence at the site of the partial coloboma. It then formed an anastomosis with retinal vessels because of the absence of Bruch's membrane and retinal pigment epithelium, and consequently did not anastomose with the posterior choroidal arteries.The anastomosis with the recurrent ciliary artery induced an increased perfusion of the retinal capillaries, causing dilatation and leakage. The retinal exudates and small hemorrhages as well as the exudative detachments are in all probability the result of the leakage caused by decompensation of the capillary endothelium.

Fogle, Welch, and Green[17] described a case of bilateral retinal exudative vasculopathy associated with retinitis pigmentosa. In both eyes the abnormal vessels were derived from choroidal vessels, and in one eye this observation could be confirmed histopathologically. However, they could not show a connection between these abnormal vessels and the retinal vessels.

The pucker in the posterior pole of the left eye has to be considered a reaction to the long-standing exudative detachment, and there is some risk that the same will happen in the right eye. Theoretically, it is possible to close the anastomosis by photocoagulation and to stop the process of leakage that maintains the subretinal fluid. However, photocoagulation may cause a secondary macular pucker.

The differential diagnosis of this condition is not difficult. The edema of the peripheral retina, the position in the inferior retinal periphery, and the presence of white punctate opacities in the vitreous might remotely suggest a pars planitis. However, the absence of fluffy exudates on the pars plana and the anomalous retinal vessels indicate a noninflammatory nature. The marked dilatation and irregularities of smaller retinal vessels and the exudative detachments, in addition to the small exudates and hemorrhages, may indicate Coats' disease, but

the typical features of Coats' disease such as miliary aneurysms and waxy yellow subretinal exudates were not seen in our patient.

Summary

A healthy 26-year-old woman, who had a decrease of vision in her left eye caused by a macular pucker, showed deformed retinal vessels, small exudative detachments of the neuroretina, and retinal exudates and hemorrhages in both fundi peripheral to the equator at the 6-o'clock position.

Fluorescein angiography and stereoscopic photographs clearly showed an anastomosis of a recurrent ciliary artery with peripheral retinal vessels and a localized absence of the choriocapillaris under the detached retina in both eyes.

The anastomosis probably developed in the last month of fetal life as the result of a localized absence of Bruch's membrane caused by an incomplete coloboma of the choroid that originated during the second month of gestation.

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CLINICAL CHARACTERISTICS OF VITAMIN A RESPONSIVE AND NONRESPONSIVE BITOT'S SPOTS

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Bitot's "hemerolopic spot"[1] has long been associated with vitamin A deficiency[2] and is probably the commonest clinical sign used in xerophthalmia prevalence surveys.[3-5] Because of reports of Bitot's spots occurring in the absence of vitamin A deficiency or night blindness, or failure to respond to vitamin A therapy,[6-11] however, the cause and significance of this lesion have been questioned.[6,9-14]

A new classification of xerophthalmia (Table 1) was recently proposed in an attempt to standardize definitions and increase the reliability of Bitot's spot as a sign of vitamin A deficiency.[15] Conjunctival xerosis (X1A) was considered the primary conjunctival manifestation of xerophthalmia, but insufficiently standardizable to serve as a useful screening criterion. Bitot's spots, defined as foamy patches at the temporal or, less often, nasal corneoscleral limbus, were divided into two groups: (1) those with concomitant conjunctival xerosis (X1B), assumed to represent active vitamin A deficiency; and (2) those without xerosis, assumed to be unrelated to vitamin A deficiency.

TABLE 1
XEROPHTHALMIA CLASSIFICATION

Classification	Description
X1A	Conjunctival xerosis
X1B*	Conjunctival xerosis with Bitot's spot
X2*	Corneal xerosis
X3A*	Corneal xerosis with ulceration
X3B*	Keratomalacia
XN†	Night blindness
XF†	Xerophthalmia fundus
XS*†	Corneal scar

* Acceptable criterion for xerophthalmia prevalence surveys.
† Secondary sign.

Unfortunately, this widely accepted classification was made without supportive data. To rectify this situation and test the validity of this classification, we examined, treated, and followed up patients with conjunctival xerosis and Bitot's spots seen here. Our results indicate Bitot's spots, conjunctival xerosis (X1A), and conjunctival xerosis with Bitot's lesions (X1B) are all basically the same lesion; nothing in their clinical appearance distinguishes vitamin A responsive from nonresponsive types; and more urgent and valuable considerations are the location and extent of conjunctival involvement, the patient's age, and presence or absence of concomitant night blindness and punctate keratopathy.

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Subjects and methods

Between June 1977 and July 1978 all patients with clear corneas and conjunctival lesions indicative of xerophthalmia were included in the study. Following standard practice,[16] conjunctival lesions were categorized by their appearance on hand-light examination: bare, dry, granular nonwettable patches, best viewed on oblique illumination, were classified as conjunctival xerosis (X1A); white patches were classified as Bitot's spots; and the presence of the two together was classified as Bitot's spots with conjunctival xerosis (X1B).

After obtaining informed-consent and a detailed history from the accompanying parent or guardian, the ophthalmologist examined the patient's eye with a Haag-Streit 900 slit lamp (with and without fluorescein) before and after removal of any superficial foam with a cotton-tipped applicator. The patient was then placed in a room darkened to the point that a normal observer could read headlines but not ordinary newsprint after ten minutes of adaptation. After 30 minutes' adaptation the patient was asked to locate his parent or guardian eight to ten feet away. This was followed by dilated, indirect ophthalmoscopy and a complete pediatric examination. Height, weight, and dietary history were obtained by a nutritionist.

Three milliliters of venous blood was collected, spun immediately, and the clear supernatant separated and stored in a refrigerator. Vitamin A determinations were carried out by the micromethod of Neeld and Pearson,[17] usually within one week and never more than two weeks after collection. Because of equipment failure serum vitamin A levels were unavailable for patients examined between July 15, 1977, and Jan. 15, 1978.

During the initial phase of this study patients were randomly treated with either low-dose (700 IU) or high-dose (200,000 IU) vitamin A in oil orally to assess better the significance of a response to therapy. Because some low-dose recipients clinically worsened, this trial was soon discontinued. Response is calculated from administration of the first high-dose capsule. Patients failing to respond to high-dose therapy received at least one and often two additional doses.

Patients were requested to return on three successive days, then weekly for the first month and monthly thereafter. Patients were considered responsive if their conjunctival lesions regressed or disappeared within two months of therapy and unresponsive if their lesions remained unchanged for at least three months.

The same ophthalmologist drew and photographed all lesions at initial examination and one or more follow-up visits. The normal deviate (z), chi square, and Student's *t*-test were used in the statistical analysis.

Results

Eighty-three consecutive cases were studied (Table 2). Among the 50 responsive patients, the lesions disappeared completely in 36 (cured) and showed marked regression (improved) in 14. In almost all instances improvement was apparent within one week of vitamin A therapy (Table 3). All nine unresponsive

TABLE 2

Outcome of cases

Outcome	No.	Total
Responded within two months		50
Cured	36	
Improved	14	
Unresponsive within three months		9
Indeterminate response		24
Inadequate early follow-up	18	
Miscellaneous	6	
Total examined		83

TABLE 3

SPEED OF IMPROVEMENT IN RESPONSIVE PATIENTS

Duration Since Therapy (days)	Patients Examined*	Patients Improved	
		No.	%
1-4	20	11	55
5-7	28	28	100
8-14	4	4	100
15-21	3	3	100
22-31	3	2	67
32-62	2	2	100

*Examined and not already improved at previous visit.

patients failed to improve within three months of receiving at least two massive doses of vitamin A.

Twenty-four cases were excluded from analysis: 18 patients had inadequate early follow-up; four low-dose recipients eventually improved spontaneously (presumably on an improved diet); and in two patients the initial lesions were extremely mild and their diagnosis and response to therapy uncertain.

Serum vitamin A levels were available in 22 cured, nine improved, and four unresponsive patients (Table 4). The mean level among improved patients was slightly lower than among cured patients, but not to a clinically or statistically significant degree. The mean levels of both, however, were significantly below that of the unresponsive group ($P<.001$).

At the initial visit, dry, granular, nonwettable patches of conjunctival xerosis, well described as appearing "like sandbanks at receding tide,"[18] were present on hand-light examination of every patient, responsive or not. In some, the xerotic surface appeared bare and free of overlying material (conjunctival xerosis, X1A) (Fig. 1); in others, it was partially or completely covered by an easily removed layer of white material (conjunctival xerosis with Bitot's spot, X1B) of a frothy, foamy character (Figs. 2 and 3) or denser, cheesy appearance (Fig. 3). In no instance did we encounter a Bitot's spot that lacked a xerotic base.

TABLE 4

SERUM VITAMIN A LEVELS*

	Cured	Improved	Unresponsive
No. of eyes	22	9	4
Mean	13.5	10.1	29.5
S.E.	1.47	2.10	4.17

*Levels given in μg/dl. Combined mean of cured and improved groups was significantly below that of the unresponsive group ($P < .001$).

Slit-lamp examination revealed little difference between these various lesions. Localized patches of conjunctival xerosis (X1A) consisted of fine, tiny, evenly distributed bubbles with clear, glistening conjunctiva between. Bitot's spots simply had larger, denser congregations of bubbles, which, in cheesy-appearing lesions became confluent, obliterating any view of the underlying conjunctiva.

In every eye with a xerotic lesion, responsive or not, the area adjacent to the temporal corneoscleral limbus was involved. In contrast, nasal involvement was far less common, and never appeared in the absence of temporal involvement. Nasal lesions were particularly rare among nonresponsive patients (Table 5, $P<.001$).

Nasal lesions were not only less common, but frequently less severe than their temporal counterpart (Table 6). In 13 eyes conjunctival xerosis with foamy Bitot's spots (X1B) was present temporally, but there was only mild conjunctival xerosis (X1A) nasally.

In three patients, all with responsive lesions and extremely low serum vitamin A levels, the xerosis was more extensive. In two, the areas of temporal and nasal involvement met inferiorly; in the third, superiorly as well. In this latter instance the conjunctiva was markedly thickened and opaque, and had a skin-like appearance (Fig. 4). Initially assigned to the

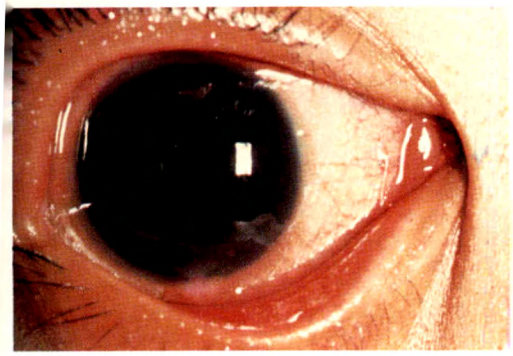


Fig. 1 (Sommer, Emran, and Tjakrasudjatma). Dry, granular conjunctival xerosis nasally in a 6-year-old girl. The lesion disappeared within two weeks of vitamin A therapy.

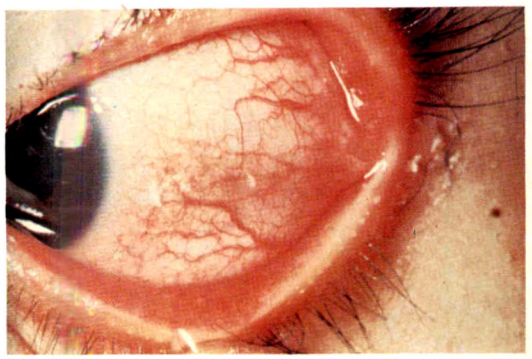


Fig. 2 (Sommer, Emran and Tjakrasudjatma). Small "foamy" Bitot's spot that healed within one month of vitamin A therapy in a 4-year-old boy.

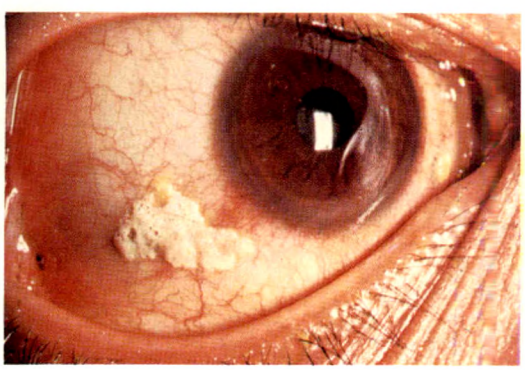


Fig. 3 (Sommer, Emran and Tjakrasudjatma). Nonresponsive Bitot's spot in a 20-year-old man. The superior margin has a foamy appearance, the rest has a cheesy appearance.

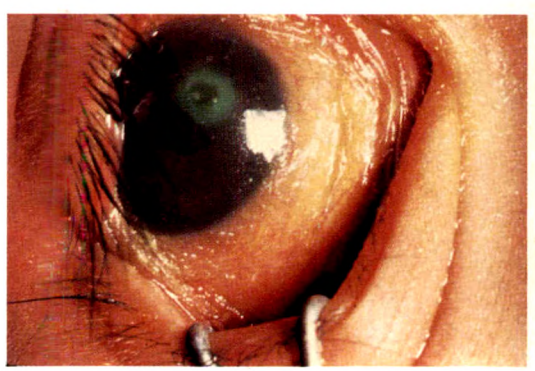


Fig. 4 (Sommer, Emran and Tjakrasudjatma). Widespread dryness, thickening, and skinlike alterations in the conjunctiva of a 1-year-old boy who developed corneal xerosis and a shallow ulcer (X3A) two weeks after low-dose therapy.

TABLE 5

PROPORTION OF ABNORMAL EYES WITH NASAL INVOLVEMENT*

	Cured	Improved	Unresponsive
Total No. of eyes	70	26	16
Eyes with nasal lesions	46 (66%)	15 (58%)	1 (6%)

*Combined rate among cured and improved groups was significantly below that of the unresponsive group ($P < .001$).

low-dose group, this patient returned at 14 days with frank corneal xerosis (X2) bilaterally and a small paraxial ulcer in the right eye (X3A). These lesions healed promptly on massive vitamin A therapy, and the child regained visual acuity of 6/6 (20/20) in both eyes. This was the only instance in which conjunctival wrinkling was obvious in primary gaze. Conjunctival wrinkling on lateral gaze[15] was no more apparent among the majority of children with conjunctival xerosis and Bitot's spots than among 76 normal age- and sex-matched controls.

As they healed, the lesions lost their overlying foam and shrank in size, becoming small xerotic patches or tiny isolated epithelial blisters located 1 to 2 mm from the corneoscleral limbus. Nasal lesions responded more rapidly than temporal ones, often dramatically so. Among six of 23 cured patients in whom both sites were involved, the two cleared at different times. In each instance the nasal lesions disappeared first. Among seven improved patients followed up for more than two months in whom both sites were involved, the nasal lesions cleared completely, usually within one to two weeks. Although showing early dramatic regression, none of the temporal lesions disappeared within the two-month period.

Responsive patients were considerably younger than nonresponsive patients (Table 7, $P<.001$). As a result, the proportion of nonresponsive patients increased sharply with age: one of 37 (3%) of patients under 6 years of age, two of 12 (17%) of patients aged 6 to 9 years, and six of ten (60%) of patients 10 years of age or older failed to respond to vitamin A. Boys accounted for 58% (29 of 50) of the responsive patients and 67% (six of nine)

TABLE 6

CLASSIFICATION OF TEMPORAL AND NASAL LESIONS IN EYES OF RESPONSIVE PATIENTS*

	Temporal Lesion			
Nasal Lesion	None	Conjunctival Xerosis (X1A)	Conjunctival Xerosis with Bitot's Spots (X1B) (F)†	Conjunctival Xerosis with Bitot's Spots (X1B) (C)†
None	4	7	28	1
Conjunctival Xerosis (X1A)		4	13	
Conjunctival xerosis with Bitot's spot (X1B) (F)†			40	
Conjunctival xerosis with Bitot's spot (X1B) (C)†				3

*(F) designates foamy Bitot's spots; and (C), cheesy Bitot's spots.
†Proportion of all lesions that were X1B, temporal vs nasal: $P < .05$.

TABLE 7

AGE DISTRIBUTION*

Age in Years Completed	Cured		Improved		Unresponsive	
	No.	Cumulative %	No.	Cumulative %	No.	Cumulative %
1	3	8				
2	11	39	3	21		
3	4	50	4	50		
4	5	64	1	57	1	11
5	3	72	2	71		11
6	6	89	1	79	1	22
7	2	95		79		22
8	1	97		79		22
9		97		79	1	33
≧10	1	100	3	100	6	100
Total	36		14		9	

*Percents rounded to nearest integer.

of the nonresponsive patients, a difference that is not statistically significant.

Night blindness was significantly less common among nonresponsive than in either group of responsive patients, both by history and clinical examination (Table 8). In three of 30 cured patients clinical testing was inconclusive: the children simply sat down and cried. All three had a history of night blindness and were among the youngest children examined, which suggests that their lack of cooperation may have resulted from impaired dark adaptation.

The presence of typical, vitamin A responsive punctate keratopathy was scored by the same system used in our original report.[19] It was significantly more common among both groups of responsive patients than among nonresponsive patients (Table 9). The average density and extent of superficial punctate keratopathy among positive cured and improved eyes was similar. In the single positive unresponsive patient, the number of lesions was small and limited to the inferonasal quadrant.

The general nutritional status of most of the responsive patients was good for Indonesia: only four of 50 had a weight for height below 80% of Western standards.[20] One of these four underweight children

TABLE 8

PRESENCE OF NIGHT BLINDNESS*

Test	Cured	Improved	Unresponsive
History available (No.)	33	12	9
History contributory (%)	91	83	33
Examination completed (No.)	27	12	9
Results positive (%)	78	67	0

*Statistical significance:

	History	Examination
Overall distribution,	$P < .001$	$P < .001$
Cured vs improved	$P < .48$	$P < .46$
Improved vs unresponsive	$P < .05$	$P < .002$
Cured vs unresponsive	$P < .001$	$P < .001$

TABLE 9

SUPERFICIAL PUNCTATE KERATOPATHY

	Cured	Improved	Unresponsive
No. tested*	27	12	8
No. positive†	26 (96%)	10 (83%)	1 (13%)
Density‡			
Mean	1.8	1.9	1.0
S.E.	0.16	0.13	—
Area‡			
Mean	2.2	2.1	1.0
S.E.	0.25	0.41	—

*Number of cases.

†Overall distribution $P < .001$; cured vs improved $P < .16$; improved vs unresponsive $P < .01$; and cured vs unresponsive $P < .001$.

‡Density and area is the average of both eyes for each positive case. Density was graded 1-3 (mild, moderate, severe) and area 1-4 (inferonasal quadrant, inferior half, inferior three fourths, and entire corneal surface).

and an additional, heavier child had kwashiorkor. Serum albumin was at least 3.5 g/100 ml in 70%; in only two (4%) was it below 3.0 g/100 ml. Angular stomatitis was rare.

Because of their older age, the nutritional status of the nonresponsive patients was better: only the single preschool child had a weight for height below 90% of standard (it was 80%); none had kwashiorkor, angular stomatitis, or a serum albumin below 3.5 g/100 ml.

DISCUSSION

The cause and significance of Bitot's spots have been repeatedly questioned. Some investigators[1,2,21] have reported Bitot's spots associated with night blindness or other evidence of vitamin A deficiency and their response to its administration. Others[6,8-10] have reported exactly the opposite: Bitot's spots in the absence of vitamin A deficiency and refractory to vitamin A therapy. Our observations confirm the general assumption that both types occur.[15] Fifty patients responded dramatically to vitamin A therapy, almost all within two to seven days. Nine patients failed to respond within three months of receiving at least twice as much vitamin A. Not surprisingly the mean serum vitamin A level of the responsive group was significantly below that of the nonresponsive group. It was also significantly below that of a randomly sampled group of Javanese children of similar age (20.0 μg/dl).[22]

Conjunctival changes traditionally associated with xerophthalmia were recently reclassified in a manner thought to better distinguish patients with active vitamin A deficiency (Table 1).[15] Bitot's spot was considered a nonspecific lesion and omitted entirely from the classification, while conjunctival xerosis (X1A), with its multiplicity of criteria and frequent overdiagnosis, was excluded from the list of acceptable screening criteria. Only Bitot's spot accompanied by conjunctival xerosis (X1B) was considered sufficiently reproducible and diagnostic of active vitamin A deficiency for use in survey work. Unfortunately, our results suggest these distinctions are unwarranted.

Conjunctival xerosis, dry nonwettable lesions well described as "emerging from their surroundings like sandbanks at receding tide,"[18] were easily recognized with oblique illumination from a hand-

light. Tandon and associates[4] reported that once seen they were not easily mistaken. On slit-lamp examination patches of conjunctival xerosis (X1A) and Bitot's spots were similar, differing only in their concentration of fine, superficial bubbles. Rather than distinct entities, conjunctival xerosis (X1A) and conjunctival xerosis with Bitot's spots (X1B) appear to be manifestations of the same xerotic process, as originally suggested by early writers.[2,23-26]

Despite claims to the contrary,[15,18,27] pigmentation and "wrinkling on lateral gaze" were of no help in distinguishing conjunctival xerosis (X1A). Except for unusual cases these changes were mild or absent and standardization was difficult and reproducibility poor. Classically, xerophthalmic wrinkling referred to grossly evident furrowing concentric with the corneoscleral limbus in the unmanipulated eye. This appears to be a late manifestation of advanced xerophthalmia; the entire bulbar conjunctiva often was thickened and skin-like,[24,26] as in the case reported here and in many with severe corneal involvement we have studied (unpublished data).

Nothing in their appearance distinguished vitamin A responsive from nonresponsive lesions. Every Bitot's spot in our study, foamy or cheesy, vitamin A responsive or nonresponsive, had a xerotic base, in agreement with classic descriptions of lesions associated[2,14,23] and unassociated[12] with vitamin A deficiency. Other characteristics were more helpful, notably the distribution of the lesions, the age of the patient, and the presence or absence of night blindness and superficial punctate keratopathy.

Nasal lesions (always accompanied by temporal lesions) were far more common in xerophthalmic eyes of responsive than nonresponsive patients (61 of 96 [64%] and one of 16 [6%] respectively). Although we appear to be the first to call attention to the significance of concomitant temporal and nasal involvement, our observations are consistent with earlier reports: nasal lesions have commonly been noted in cases of active vitamin A deficiency,[2,21,24] but rarely in cases with normal vitamin A nutriture.[9,12] Extension of temporal and nasal involvement below, to meet in the inferior quadrant (or occasionally above as well, to meet superiorly) is even more specific for active vitamin A deficiency, usually severe and advanced.

Patients with active vitamin A deficiency were also younger. Admittedly ours was a clinic population, but the difference in the age distribution of the two groups was striking: only 3% of patients under age 6 years but 60% over age 9 years failed to respond to vitamin A. Even in parts of India where nonresponsive lesions are fairly prevalent among preschool children[11,28] they rarely arise de novo in populations given vitamin A supplements.[28]

Responsive lesions were usually associated with other evidence of vitamin A related functional impairment. A history of night blindness is the easiest to collect and was present in almost 90% (40 of 45) of responsive patients. In an actual house-to-house survey of rural preschool children in Java a history of night blindness was present in over 60% (80 of 132) of those with Bitot's spots.[22] Others have reported similar rates.[23,26,29,30] As Stephenson[2] pointed out over 70 years ago, night blindness is more likely to be recognized among clinic patients because it is the reason most individuals seek help in the first place. Among those not attending a clinic the night blindness is likely to be milder or otherwise overlooked. One third (three of nine) of nonresponsive patients also claimed to be night blind. In none of the three could the diagnosis be confirmed clinically. Although a contributory history can exist with subtle impairment undetectable by our crude test,[22] the serum vitamin A level in the single

case in which it was available was normal (22 μg/dl). In contrast, clincally documented night blindness was present in at least 75% of responsive patients tested.

Although more practical for clinic than field testing, fluorescein-positive superficial punctate keratopathy was more likely to be present in responsive than in nonresponsive eyes (92% [88 of 96] vs 3% [two of 16]) and also to be denser and cover more of the surface.

None of the above associations proved infallible in distinguishing between cases of Bitot's spots with and those without active vitamin A deficiency. However, the presence of lesions on both sides of the cornea or a history of night blindness, especially in preschool children, stongly suggests active vitamin A deficiency. The diagnosis is almost assured if the xerosis involves the inferior 180 degrees, or more, of the conjunctiva or if the night blindness is confirmed clinically. Many cases of active vitamin A deficiency will not satisfy these more stringent criteria. Vital staining of the conjunctiva with lissamine green or rose Bengal, reported to be highly accurate in distinguishing between the two groups,[31] has not been as effective as reported earlier.[32]

The cause of the nonresponsive lesions remains uncertain. In none of the cases studied was there any evidence of local trauma, trachoma, pemphigus, keratoconjunctivitis sicca, collagen vascular diseases, or cirrhosis. It has been suggested that Bitot's spots in individuals without vitamin A deficiency in developing countries are caused by protein or nonspecific forms of malnutrition.[6,8,12,14,24] This seems unlikely. Malnutrition is commonest among preschool children, whereas nonresponsive patients are usually older, and all but one in our series (the youngest was a 4-year-old) were well nourished. It appears more likely that at least some of these nonresponsive lesions represent a persistent metaplastic change induced during a prior xerophthalmic episode. As already noted, nonresponsive lesions characteristically occur temporally,[9,12] the site of 16 of the 17 lesions in nonresponsive eyes in our series. The temporal quadrant also appears to be the area most susceptible to the effects of active vitamin A deficiency: among responsive cases temporal lesions were present in every affected eye, nasal lesions in only two thirds; temporal lesions were often more prominent than their nasal counterparts, never less prominent; and temporal lesions were often the last to heal, never the first. These observations are consistent with descriptions found in previous case reports.[14,21,30] In five of our improved patients the nasal lesions disappeared entirely. Although their temporal counterparts regressed soon after therapy, they persisted, in milder form, for at least three to five months. Examined during this latter period they would have appeared to be classic examples of nonresponsive cases without vitamin A deficiency.

In one patient, followed up for six months, the temporal lesions improved dramatically with therapy and, though never regaining their original size and prominence, waxed and waned throughout the period of observation, disappearing entirely only to reappear later, while serum vitamin A levels remained at their posttreatment high (>25 μg/dl). This case helps explain the observations of Sinha and Bang.[28] One half of the preschool children in their study village received massive doses of supplemental vitamin A and one half did not. Although new Bitot's spots continued to appear among vitamin A recipients, almost invariably these were children who had had Bitot's spots in the past. In contrast, new spots were found in previously normal placebo recipients. It seems that vitamin A deficiency is required for the production of nonresponsive spots, but once the metaplastic change has taken place other

factors can affect their prominence and size.

The particular susceptibility of the temporal area remains to be explained, though synergism between vitamin A deficiency and exposure probably plays an important role. We have followed up a child who had severe vitamin A deficiency with extensive conjunctival xerosis of both eyes and a xerotic, heavily keratinized unilateral staphyloma (from earlier keratomalacia) that protruded well beyond the eyelids. Although the slowest area to respond, the exposed surface of the staphyloma eventually lost most of its keratinization on massive doses of systemic vitamin A (unpublished data).

Summary

We examined and followed up 59 patients with conjunctival xerosis with and without classic Bitot's spots. Of these, 50 were responsive and nine were unresponsive to vitamin A.

Despite a recent World Health Organization classification suggesting otherwise, all cases appeared to represent the same process. In preschool children, the presence of the Bitot's spots on both sides of the corneoscleral limbus or in association with night blindness or punctate keratopathy suggests active vitamin A deficiency. Pigmentation and wrinkling were not useful diagnostic criteria and lesions isolated to the nasal corneoscleral limbus suggested a different diagnosis. At least some cases of nonresponsive conjunctival xerosis and Bitot's spots were probably sequelae of past vitamin A deficiency.

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COMPARISON OF ENDOTHELIAL CELL DENSITY IN DIABETICS AND A CONTROL POPULATION

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In diabetics, corneal decompensation is a frequent complication of closed vitrectomy.[1-3] It has been suggested that in diabetic corneas the primary abnormality resulting in higher complication rates resides in the epithelium,[4] possibly implicating the sorbitol pathway in the pathogenesis of epithelial complications. However, stromal edema, and many of the other complications, may also result from decreased endothelial cell function or relative decreased endothelial cell density in the diabetic population. This study was performed to determine if there is a relative decrease in endothelial cell density in diabetics with proliferative retinopathy as compared to the endothelial cell density of age-matched controls.

SUBJECTS AND METHODS

We studied 96 corneas from 52 diabetic patients who had had proliferative retinopathy for at least 14 years. The patients ranged in age from 18 to 77 years. All were taking insulin, and had indications for panretinal photocoagulation as outlined by the Diabetic Retinopathy Study Group.[5] Sixty-one normal corneas from 32 subjects ranging in age from 26 to 77 years were used as controls. Patients with previous intraocular surgery, glaucoma, iritis, or Fuchs' endothelial dystrophy were excluded from the study. The controls were all recruited from the hospital staff.

Central corneal thickness was measured by a Haag-Streit pachometer and intraocular pressure was measured by Goldmann applanation tonometry. A specular microscope was used to obtain central endothelial cell photographs. Three photographs of the central endothelial cells were obtained for each cornea. The photographs were projected on a Realistic Microform Reader using the fixed frame method previously described to determine the endothelial cell density.[6] The endothelial cell density from the three photographs was used to determine the mean value for each cornea.

The error inherent in the counting technique was established by calculating the error resulting from the three separate endothelial cell counts for the same photograph. The mean error between three separate photographs of the same area was similarly determined.

RESULTS

The error inherent in the fixed frame method of determining endothelial cell density was ± 5.67%. A best-fit linear regression curve was calculated for the diabetic and control population comparing endothelial cell density to age (Figure). While both curves intersected the ordinate at roughly the same value, the curve for diabetics was steeper. However, no statistical difference was found between the two curves ($P>.05$).

The mean endothelial cell density for each group was calculated by decade (Table). When a linear regression curve was then fitted to these data, the diabet-

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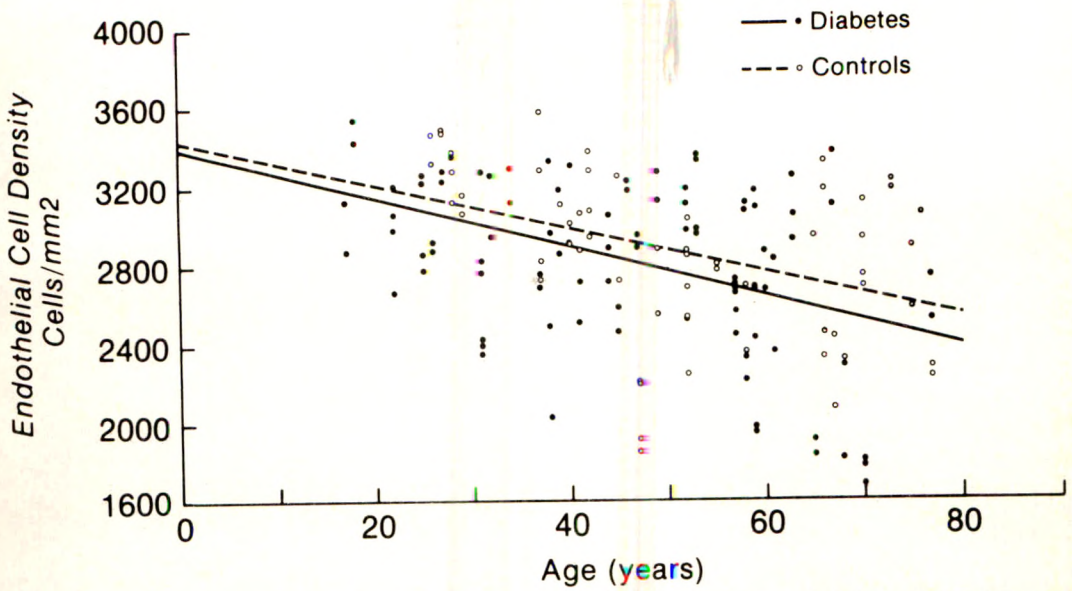


Figure (Pardos and Krachmer). Best-fit regression curves for data comparing endothelial cell density to age for diabetic and control populations. Regression curves describe equation $Y=-12.7X+3{,}398$ and $Y=-10.9X+3{,}419$, for diabetics and control group, respectively.

ics had a statistically significant lower endothelial cell density ($P<.008$). However, when Student's *t*-test was used to compare the mean endothelial cell density between diabetics and controls for each decade, the only significant difference ($P=.0075$) was found in the last decade (age 70 to 79 years). When this decade was excluded from the best-fit linear regression curve analysis, no statistical difference was found between the diabetic and control populations.

Discussion

Measurements of endothelial cell density in the normal population have shown a reciprocal relationship to age.[6-9] A similar relationship and values have been obtained from this study.

Although the linear regression curve

TABLE

MEAN ENDOTHELIAL CELL DENSITY BY DECADE FOR THE DIABETIC AND CONTROL POPULATION*

Decade	Controls		Diabetics	
	Eyes	ECD	Eyes	ECD
20-29	9	3,301	13	3,053
30-39	6	3,104	18	2,865
40-49	16	2,764	15	2,920
50-59	10	2,688	26	2,756
60-69	8	2,634	15	2,618
70-79	12	2,835	5	2,103

*ECD designates endothelial cell density.

for the diabetic population was found to be steeper, suggesting an accelerated rate of endothelial cell decrease compared to the control population, this difference was not statistically significant. The mean endothelial cell density was also compared by decade to evaluate the possibility of a nonlinear relation between endothelial cell density and age. Only in the last decade analyzed was a statistically significant difference found. However, this result, although statistically significant, is highly suspect, because in this decade only five corneas from three diabetics were examined. An attempt was made to recruit more diabetics, but there was a scarcity of candidates as defined by the protocol. The relative lack of diabetics with proliferative disease in the last decade examined may be reflected in their decreased life expectancy.[10]

We found no significant difference in endothelial cell density between a diabetic population with proliferative diabetic retinopathy and an age-matched control population. However, the functional capacity of these cells was not examined. Functionally, the diabetic endothelial cells may be less active than those of an age-matched control group. The implied susceptibility of diabetic corneas to injury is not reflected in their endothelial cell density.

Summary

We compared the endothelial cell density of 96 corneas from 52 diabetics with proliferative diabetic retinopathy wth 61 corneas from 32 normal subjects by specular microscopy. Although the regression curve of the diabetic group was steeper, we found no statistically significant difference in endothelial cell density between the two groups.

Acknowledgment

The Retina Service of the University of Iowa's Department of Ophthalmology referred these patients to us.

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RANDOMIZED CLINICAL TRIAL OF TOPICALLY ADMINISTERED CROMOLYN SODIUM FOR VERNAL KERATOCONJUNCTIVITIS

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Vernal keratoconjunctivitis is a recurrent, bilateral external ocular inflammatory disease of unknown cause. It occurs more frequently in children than in adults and commonly shows severe exacerbations in the spring of each year (hence the name vernal). The disease is characterized predominantly by intense ocular itching, tearing, photophobia, foreign body or burning sensation, or both, thick mucous discharge from the eyes, and blepharoptosis. The classic clinical feature is the cobblestone papillary reaction of the upper tarsal conjunctiva. Keratitis is common, and corneal ulceration and vascularization may occur.[1-5] Although the precise cause is virtually never identifiable, much evidence suggests that it is an allergic disorder in which IgE-mediated (Coombs and Gell type 1 hypersensitivity reaction) mechanisms play an important role.

Until recently, the conventional treatment of vernal keratoconjunctivitis included topically administered corticosteroids as the main form of therapy. Systemically and topically administered antihistamines, vasoconstrictors, cold compresses, surgical excision of giant papillae, radiation therapy, cryotherapy, and mucolytic agents, as well as various other therapeutic modalities, have also been prescribed. The most consistently effective therapy, in the experience of most clinicians who regularly treat patients with this condition, was topically administered corticosteroids.

Easty, Rice, and Jones[6] emphasized the need for alternative, more specific therapies for vernal keratoconjunctivitis. These authors stressed that corticosteroids did not achieve complete control in all cases and that the long-term complications of their use (glaucoma, cataract, herpes simplex keratitis) in a chronic disease may be substantial. These investigators reported results of a randomized, placebo-controlled, double-masked study on the effects of topically administered cromolyn sodium (disodium cromoglycate) on vernal keratoconjunctivitis. They found that the drug was effective in treating vernal catarrh, that it was well tolerated over long-term evaluations (without apparent significant side effects), and that it reduced or eliminated the need for topically administered corticosteroids. Hyams, Bialik, and Newmann[7] subsequently reported that cromolyn had no beneficial effect on their patients; Kasdan and associates[8] and Ostler, Martin, and Dawson[9] later reported the impressive therapeutic effect of cromolyn on their patients with vernal keratoconjunctivitis. Tabarra and Arafat[10] and Sayegh, Samerra'e, and Khateeb[11] reported corroborative evidence on the effectiveness of cromolyn in the treatment of vernal catarrh.

We report herein the results of a

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double-masked, double-crossover, placebo-controlled study of the effectiveness of topically administered cromolyn sodium (4%) in treating 11 patients with classic vernal keratoconjunctivitis associated with hyperimmunoglobulinemia E, eosinophilia, and atopy.

Subjects and methods

We studied 11 patients (including three siblings), ranging in age from 4 to 29 years, who had classic severe bilateral vernal keratoconjunctivitis (that is, vernal catarrh with cobblestones plus corneal involvement with superficial punctate keratitis or frank epithelial defect). Three were female, eight were male; six were white, five were black. No cases of limbal vernal keratoconjunctivitis were included in the study. All 11 patients also had a history of atopy (Table 1). All had required frequent and extensive use of corticosteroid drops to control the signs and symptoms of vernal catarrh.

Cromolyn sodium solution (4%) was prepared from commercially available capsules (Intal) by dissolving the powder (including the excipients) in physiologic saline with 0.01% benzalkonium chloride and 0.01% disodium edetate. Placebo drops were composed of physiologic saline with 0.01% benzalkonium chloride and 0.01% disodium edetate.

TABLE 1

Patient characteristics

Patient No., Sex, Age (yrs)	Atopic History	Serum IgE* (ng/ml)
1, M, 4	Eczema	520
2, M, 5	Eczema	640
3, F, 7	Eczema	967
4, M, 8	Asthma	1,210
5, M, 9	Hay fever	460
6, F, 12	Asthma	2,640
7, M, 12	Eczema	1,420
8, F, 15	Hay fever	485
9, M, 19	Eczema and hay fever	2,460
10, M, 22	Hay fever	625
11, M, 29	Asthma	1,840

*Normal range is 20-400 ng/ml, as determined by the radioallergosorbent test method.

Each patient was assigned two coded bottles of eyedrops: placebo and cromolyn. The study was of randomized, controlled, double-masked, and double-crossover design. Neither the patient nor the investigator knew which eye of each patient was receiving placebo and which was receiving cromolyn. The randomization schedule determined which eye received which drop preparation initially. All medications being taken at the beginning of the study were continued. Both eyes of each of the 11 patients were receiving one drop of 0.125% prednisolone acetate twice daily at the time of entry into the study; this therapy was continued for the duration of the study. During the study, each patient received one drop of one of the study drugs (either placebo or chromolyn) in the right eye four times daily and one drop of the other drug in the left eye four times daily. At the end of two weeks, the drug assignment for each eye was switched, and after an additional two weeks, the assignment was reversed once more.

Patients were seen weekly during the six-week study period and were examined as previously described.[7] A score of 0 to 3 was assigned for each eye, based on each of the following: upper and lower tarsal conjunctival papillae, blepharoptosis, Horner-Trantas dots, limbal nodules, bulbar conjunctival injection, superficial punctate corneal epithelial defects, and corneal erosion. The results were documented photographically at each visit. The daily subjective symptoms were scored from 0 to 4 by the patient or parents for itching, irritation, photophobia, and secretion.

In the double-crossover design, there were three periods of two weeks each,

totalling six weeks. In period 1, one eye of each patient received solution A, the other received solution B; in period 2, the solutions were switched; then in period 3, the solutions were switched back. The four factors of primary interest were bulbar injection, superficial punctate keratitis, itching, and secretion. Thus, for each factor there were two sequences: ABA, BAB, or CPC, PCP (C indicates cromolyn; P, placebo). Responses were recorded each week, the means and standard deviations for 11 patients were recorded, and weekly mean responses were plotted.

Using this double-crossover design, we assumed that the two treatment sequences were independent for any given patient; that is, there were no interaction effects between the two eyes. Next, in order to ensure independence between trials within a given sequence, we sampled only the second week in each period, that is, weeks 2, 4, and 6. This gave, for each patient, two independent three-block sequences; $C_2P_4C_6$, $P_2C_4P_6$. For example, C_2 refers to the measurement of a given condition for cromolyn in the second week of treatment; P_6 refers to the measurement for placebo in the sixth week.

Then we calculated the following:

$$D_1 = \frac{C_2 + C_6}{2} - P_4$$

$$D_2 = \frac{P_2 + P_6}{2} - C_4$$

These are modified versions of algorithms that were suggested for the switchback design by Cochran and Cox[12] and Ciminera and Wolfe.[13]

D_1 is the difference in sequence CPC between weeks 2 and 6 (cromolyn) and week 4 (placebo). D_2 is the difference in sequence PCP between weeks 2 and 6 (placebo) and week 4 (cromolyn). These differences, as computed, adjust for any trend effects within sequences. If the two test solutions have similar effects, then D_1 and D_2 should have means of approximately zero. Departures from this value suggest dissimilarity.

We computed D_1 and D_2 for each patient in each condition observed. This gave an $11 \times 4 \times 2$ matrix: 11 patients, by four conditions, by two sequence-difference measures. Finally, we performed an analysis of variance[14] to determine whether there were significant differences between the two sequences (F statistic).

Results

Conjunctival injection—The mean scores and standard deviations of the major factors of interest were recorded for each week (week 1 to week 6) (Table 2). The mean scores of these factors are also plotted in Figures 1 to 4. The effect of cromolyn therapy was readily apparent. Conjunctival injection began to decrease within one week after cromolyn therapy was begun, and continued to improve as therapy was continued into the second week (Fig. 1). However, when placebo was substituted for cromolyn, conjunctival injection steadily became more pronounced and had nearly reached initial levels after two weeks.

Table 3 shows the means and standard deviations of sequence differences D_1 and D_2 for each study factor. For conjunctival injection, the means for D_1 and D_2 were not zero, which suggests that the two solutions caused different effects. The F statistic (Table 4) for the analysis of variance shows that the variance associated with the differences in the two sequences, CPC and PCP, is statistically significant ($P<.001$).

Superficial punctate keratitis—The effects of cromolyn therapy on superficial punctate epithelial defects of the cornea were less dramatic than the effects observed on the other study factors. An apparent trend of less superficial punc-

TABLE 2

WEEKLY SUMMARIES*

Condition	Sequence	No.	Statistic	Week 1	2	3	4	5	6
Bulbar injection	CPC	11	Mean	1.3	0.5	1.9	2.5	1.0	0.4
			S.D.	0.5	0.5	0.5	0.7	0.5	0.5
	PCP	11	Mean	2.8	2.8	1.2	0.6	1.8	2.5
			S.D.	0.4	0.4	0.4	0.7	0.4	0.7
SPK	CPC	11	Mean	1.1	0.4	0.5	1.0	0.4	0.2
			S.D.	0.7	0.5	0.7	1.0	0.5	0.4
	PCP	11	Mean	1.7	1.7	1.4	0.5	0.7	1.2
			S.D.	0.6	0.6	0.7	0.7	0.7	0.8
Itching	CPC	11	Mean	0.8	0.4	2.3	3.2	0.6	0.1
			S.D.	0.6	0.5	0.8	0.6	0.5	0.3
	PCP	11	Mean	3.6	3.7	1.1	0.4	2.2	2.9
			S.D.	0.7	0.6	0.7	0.5	0.9	0.9
Secretion	CPC	11	Mean	1.3	0.8	2.0	2.5	0.9	0.3
			S.D.	0.5	0.8	0.7	0.5	0.3	0.5
	PCP	11	Mean	3.0	3.1	1.7	1.1	2.1	2.5
			S.D.	1.0	1.0	0.8	0.8	0.7	0.8

*SPK designates superficial punctate keratitis; C, cromolyn; and P, placebo.

tate keratitis associated with cromolyn therapy is seen in Figure 2. The D_1 and D_2 sequence-difference means for superficial punctate keratitis (Table 3) are, however, significantly different as judged by analysis of variance (Table 4), with an F value of 43.49 ($P<.001$).

Itching—The most dramatic differences between cromolyn- and placebo-treated eyes are seen in Figure 3, where mean scores of reported itching severity are plotted. A prompt and marked de-

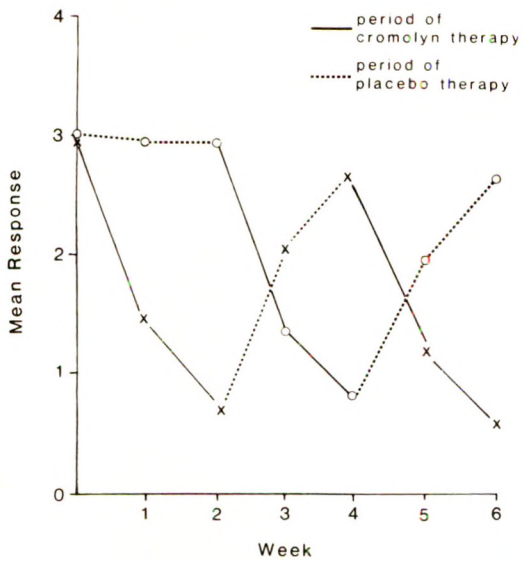


Fig. 1 (Foster and Duncan). Bulbar conjunctival injection. Graphic plot of the mean conjunctival injection score each week for all eyes receiving cromolyn and for those receiving placebo. Note the prompt decrease in injection scores associated with cromolyn therapy.

TABLE 3

SEQUENCE DIFFERENCE*

Condition	Sequence	No.	Statistic	
Bulbar injection	D_1: CPC	11	Mean	− 1.98
			S.D.	0.77
	D_2: PCP	11	Mean	2.02
			S.D.	0.78
SPK	D_1: CPC	11	Mean	− 0.72
			S.D.	0.70
	D_2: PCP	11	Mean	0.92
			S.D.	0.44
Itching	D_1: CPC	11	Mean	− 2.95
			S.D.	0.70
	D_2: PCP	11	Mean	2.95
			S.D.	0.72
Secretion	D_1: CPC	11	Mean	− 1.94
			S.D.	0.69
	D_2: PCP	11	Mean	1.71
			S.D.	0.87

*SPK designates superficial punctate keratitis; C, cromolyn; and P, placebo.

TABLE 4

ANALYSIS OF VARIANCE*

Condition	Source	Sum of Square	Degrees of Freedom	Mean Square	F	Trial P Value
Bulbar injection	Sequence†	88.00	1	88.000	147.00	<.001
	Error‡	11.973	20	0.599		
SPK	Sequence	14.727	1	14.727	43.49	<.001
	Error	6.773	20	0.339		
Itching	Sequence	191.455	1	191.455	377.08	<.001
	Error	10.155	20	0.508		
Secretion	Sequence	73.091	1	73.091	119.29	<.001
	Error	12.255	20	0.613		

*SPK designates superficial punctate keratitis.
†Sequence: variability between sequences, CPC and PCP.
‡Error: variability within sequences, CPC and PCP.

crease in itching was uniformly associated with cromolyn therapy. The switch from cromolyn to placebo resulted in return of moderate to intense itching within one week. The D_1 and D_2 sequence-difference means for itching (Table 3) were significantly different ($P<.001$).

Mucous secretion—We also found a prominent effect of cromolyn therapy on mucous secretion. This effect was less dramatic and of slower development than the effect of cromolyn on itching, and in many ways resembled the effect seen on conjunctival inflammation (Fig. 4). The D_1 and D_2 treatment sequence-difference means for secretion (Table 3) differed significantly ($P<.001$).

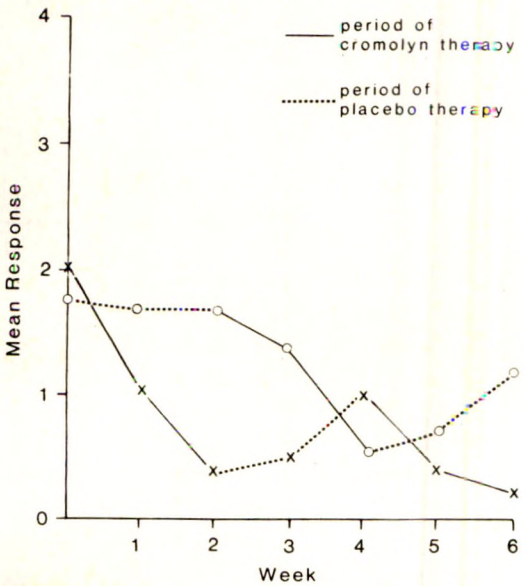


Fig. 2 (Foster and Duncan). Superficial punctate keratitis. An apparent trend toward less superficial punctate keratitis associated with cromolyn therapy is seen. Note the slow redevelopment of superficial punctate keratitis changes, though to lesser degrees, associated with substitution of placebo for cromolyn drops.

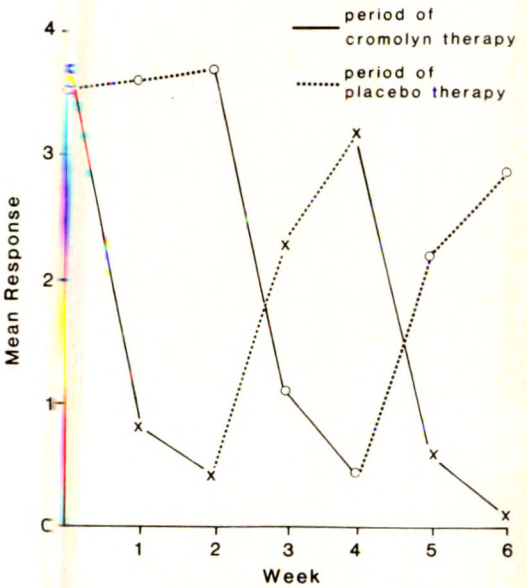


Fig. 3 (Foster and Duncan). Itching. Note the dramatic and prompt decrease in itching scores associated with cromolyn therapy, and the rapid return of high scores with placebo therapy.

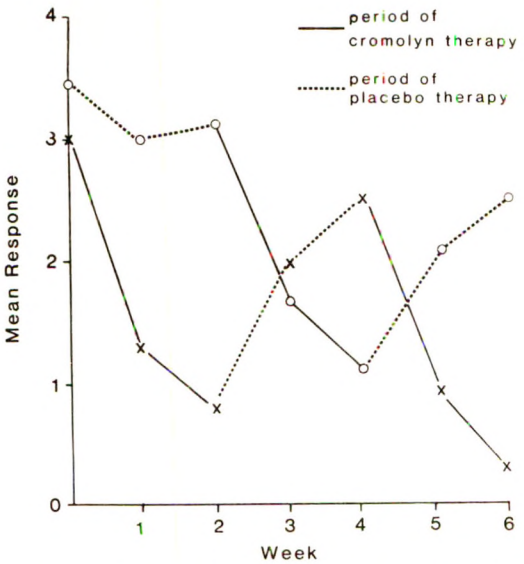


Fig. 4 (Foster and Duncan). Secretion. Note the decreased scores associated with cromolyn therapy.

Discussion

Our results confirm previous findings[6, 8-11] regarding the effectiveness and safety of topically administered cromolyn in the therapy of vernal keratoconjunctivitis. The discrepancy between our results and those of Hyams, Bialik, and Newmann[7] may stem from two important differences in the two studies. First, Hyams, Bialik, and Newmann[7] used 1% cromolyn sodium. In our experience this concentration is not sufficient to achieve a therapeutic effect in most patients. Secondly, and perhaps more importantly, none of the patients in their study had atopic disease, whereas all of our patients and most of the patients from the other studies[6, 8-11] also displayed signs of atopy. We believe that at least two populations of patients with vernal keratoconjunctivitis exist: those with atopy (as distinguished by history of hay fever, asthma, or eczema, and by peripheral blood eosinophilia and increased serum IgE levels) and those without. We also believe that, just as in the treatment of patients with asthma,[15] cromolyn sodium is most effective in patients with a clear atopic IgE-mediated disease mechanism.

There is substantial evidence to suggest that IgE-mediated hypersensitivity reactions play an important role in the disease mechanisms of many patients with vernal keratoconjunctivitis. The concomitant atopic history in many patients, the response of the disease to cromolyn therapy, the increased tear histamine levels,[16] the immunohistopathology, characterized by lymphocyte, plasma cell, and prominent eosinophil infiltration of the conjunctival stroma, and the presence of massive degrees of mast cell degranulation in this tissue all suggest IgE-mediated mechanisms. Type 4 cutaneous basophil hypersensitivity mechanisms may also be involved. Cromolyn presumably affects IgE-mediated reactions by inhibiting mast cell degranulation (possibly through an action on the adenylcyclase-cyclic AMP system calcium fluxes), and in this way effectively suppresses the acceleration of inflammation. We have found this drug to be safe in treating patients with vernal keratoconjunctivitis. Adverse effects have been limited to mild ocular irritation in three patients. Our preparation is not a pure preparation, but one that contains lactose and other excipients present in Intal capsules. Easty, Rice, and Jones[6] found ocular irritation attributable to the thimerisol preservative in their preparation. They also reported one case of cutaneous sensitivity reaction; we have encountered no such instances. Adverse reactions to cromolyn, reported by others (a 2% incidence), include dermatitis, myositis, and gastroenteritis.[17]

We believe that ophthalmologists are no longer limited to seeking only a diminution of symptoms in most patients with vernal keratoconjunctivitis[18]; rather, we have found that most patients (through modulations of their antihistamine, corticosteroid, mucolytic, cromolyn regi-

mens) can have almost total relief of symptoms of vernal keratoconjunctivitis throughout the year. Additionally, we have found cromolyn to be much more effective as a prophylactic agent than as an interrupter of a well-established attack of vernal catarrh. It has been particularly effective in enabling patients with vernal keratoconjunctivitis to significantly reduce or discontinue the use of corticosteroids.

Summary

We performed a controlled, randomized, double-masked, double-crossover clinical trial topically administered cromolyn sodium (4%) in 11 patients with atopy and vernal keratoconjunctivitis. Statistically significant differences were noted in conjunctival injection, superficial punctate keratitis, mucous production, and itching when eyes treated with cromolyn were compared to those treated with a placebo. The drug was well tolerated by all patients.

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SUBRETINAL NEOVASCULAR MEMBRANE AND DISCIFORM SCAR IN BEHÇET'S DISEASE

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Behçet's disease is characterized by three primary components: iridocyclitis with hypopyon, aphthous lesions in the mouth, and ulceration of the genitalia. Erythema nodosum, arthropathy, and thrombophlebitis often accompany these manifestations, but the ocular symptoms may be the most important and serious manifestations of the disease.[1] Central nervous system involvement often precedes death. The frequency of ocular manifestations is 70 to 85% in patients with the disease.[1-3] The underlying disease in all organ systems is an occlusive vasculitis.[4] Although the most common ocular symptom is that of an anterior uveitis, often with hypopyon as a late sign,[5] the presence of necrotizing retinal vascular lesions is well known and often obscured by the severity of the anterior reaction.[6] Retinal lesions have been reported in up to 50% of the cases.[7] We describe a patient with Behçet's disease who had a disciform lesion in the deep outer retinal layers and choroid, secondary to a subretinal neovascular membrane.

CASE REPORT

A 33-year-old woman had a three-year history of aphthous ulceration of the mouth, vaginal ulceration, and an intermittent erythematous tender rash on the shins, consistent with erythema nodosum. Her history began with a recurrent sore throat with tonsillar exudate for which no relief could be found.

In August 1976, during an episode of vaginal sores, "canker sores" in the throat, and the acute initial manifestation of the skin rash on her shins and ankles, she awoke with blurred vision in her right eye. Ophthalmologic consultation at that time provided a working diagnosis of "histoplasmosis or toxoplasmosis" in the left eye. After limited testing failed to show a diagnosis, the patient consulted a local retinal specialist. He performed a fluorescein angiogram and believed it to be consistent with toxoplasmosis, although this was not confirmed by serology. No treatment was administered. The patient had recurrent bouts of stomatitis and aphthi, and vaginal ulcerations that responded to corticosteroid therapy.

The patient was seen by us in February 1979, for ophthalmologic consultation. She was 5 months pregnant, and anxiously concerned about the diagnosis of toxoplasmosis and its possible effect on her offspring.

Visual acuity was R.E.: 6/6 (20/20) with correction and L.E.: 6/120 (20/400). Corneas were clear and conjunctivae were normal without lesions. Anterior chambers were quiet and the lenses were clear. Irides were normal without nodules. Applanation pressure by Goldmann tonometry was R.E.: 19 mm Hg and L.E.: 18 mm Hg. Results of ophthalmoscopic examination were normal in the right eye. The left macula, however, showed a deep disciform lesion centrally, with normal retinal vasculature overlying it. There were several discrete hypertrophic retinal pigment epithelial lesions inferior to this (Fig. 1).

Fluorescent antibody titer, Sabid-Feldman dye titer, and enzyme linked immuno-absorbent assay were all negative for *Toxoplasma* antibodies on undiluted sera. Indirect immunofluorescent antibodies were shown to guinea pig lip epithelial cells (Fig. 2) in a dilution of 1:30.[1,8,9]

Fluorescein angiography, performed eight months later, showed an identical fundus appearance. The early choroidal phase showed a lacy, dot-like hyperfluorescence in the foveal region of the surrounding disciform scar, which in turn blocked the underlying choroidal fluorescence (Fig. 3). As the study progressed, the subretinal hyperfluorescence increased until in the late phases the entire disciform lesion took up fluorescein and stained late (Fig. 4). The overlying retinal vasculature was normal throughout

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Fig. 1 (Michelson, Michelson, and Chisari). Fundus photograph of left eye shows a disciform macular scar. The retinal vessels appear normal overlying the scarred area.

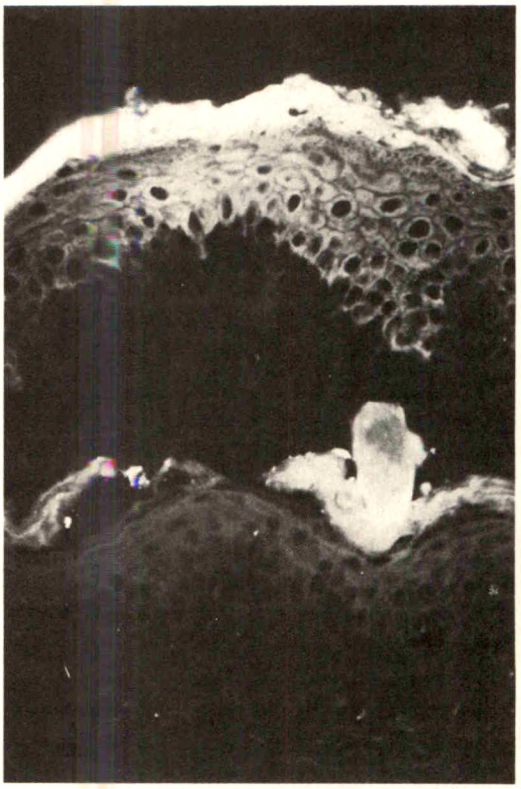


Fig. 2 (Michelson, Michelson, and Chisari). Indirect immunofluorescent analysis of serum of Behçet's antibody. (Substrate, guinea pig lip, ×125). Top, Serum from patient with Behçet's disease. Note brighter fluorescent staining of cytoplasm of epidermis. Nuclei and intercellular bridge areas are negative. Bottom, Serum from normal control. Note absence of epidermal fluorescence. Bright oval in upper right of the panel is a hair shaft.

the study, without leakage or staining of the walls of the vessels.

Material and Methods

Antibodies to cytoplasmic antigens in the stratified squamous epithelium of human and quinea pig lips were identified by indirect immunfluorescence. Cryostat sections were incubated for 45 minutes at 20°C with serial dilutions of patient and control serum (1:3, 1:10, and 1:30 in 0.01M NaH_2PO_4, 0.15M NaCl, pH 7.2, phosphate-buffered solution). They were then washed three times for five minutes in phosphate-buffered solution, dried and incubated with an optimal dilution of a goat antiserum to human immunoglobulins previously conjugated with fluorescein isothiocyanate (0.1 mg/ml, fluorescein/protein ratio = 3.1), according to the method of Clark and Shepard.[8] After 45 minutes' incubation at 20°C, the slides were washed as previously described and the stained sections were examined for fluorescence with a Zeiss RA microscope equipped with an HB-200 mercury arc lamp, a 490-nm interference filter, an NA 1.2 to 1.4 dark field condenser and a 530-nm banner filter. Positive staining was interpreted as apple-green fluorescence restricted to the squamous epthelium of oral mucosa characterized by sparing of epithelial cell nuclei and intercellular bridges (Fig. 2). The positive sera did not stain the epithelial cells of human, rhesus monkey, or guinea pig esophagus or guinea pig

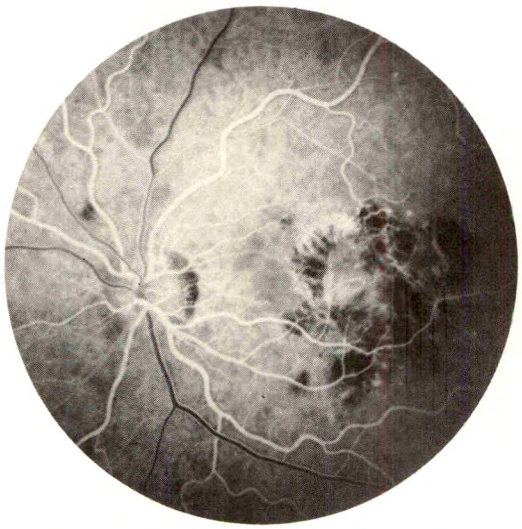


Fig. 3 (Michelson, Michelson, and Chisari). Fluorescein angiogram in the early arteriovenous phase shows the filling of the lacy, subretinal neovascular membrane and the accumulation of dye in the central scarred area.

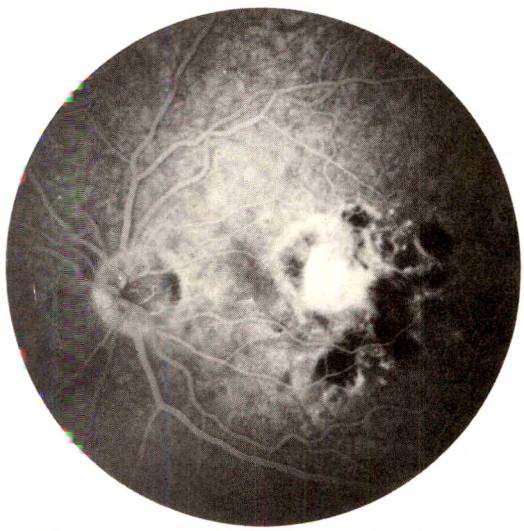


Fig. 4 (Michelson, Michelson, and Chisari). The late phase of the angiogram shows the retinal vessels to be void of dye and the macular scar to be staining late.

tongue and skin, suggesting the existence of anatomically specific antigens. Sera from normal controls and from patients with a variety of other lesions did not stain the substrate.

Discussion

The diagnosis of Behçet's syndrome was established in this woman by the presence of two of the three major components of the disease: oral aphthi and genital ulceration. Erythema nodosum had also been present. Iridocyclitis was never shown clinically.

In our patient, the presence of antibodies against oral mucosa further suggests Behçet's disease.[8,9] A significantly higher percentage of patients with ocular disease in Behçet's syndrome had antibodies to oral mucosa by several techniques, as opposed to those who did not. This factor may prove to be beneficial in the examination of patients with retinal vasculitis, in whom Behçet's syndrome is considered in the differential diagnosis.

Clinical examination, complemented by fluorescein angiography, showed a disciform scar in the deep retina-choroid, with the presence of subretinal neovascular membrane, This has not previously been reported in Behçet's disease.

Because the underlying pathology of Behçet's disease is considered to be necrotizing, occlusive vasculitis, this ocular lesion may have originated from a vasculitis of the choroidal-ciliary circulation.

Subretinal neovascular membranes are found in several conditions in young patients: histoplasmosis, toxoplasmosis, sickle-cell anemia, angioid streaks (with and without pseudoxanthoma elasticum), "lacquer cracks" in Bruch's membrane in myopia, Paget's disease, Ehlers-Danlos' syndrome, and laser treatment. This patient had none of these conditions as a co-existing disorder. Idiopathic subretinal membranes may rarely occur in young patients.

Thus, when a young patient has a disciform scar and subretinal membrane,

Behçet's disease must also be considered in a differential diagnosis of the macular degeneration.

Summary

A 33-year-old woman with a three-year history of aphthous ulceration of the mouth, vaginal ulceration, and erythema nodosum, also had a subretinal neovascular membrane in the left eye. She showed antibodies to guinea pig lip epithelium by indirect immunofluorescence. Thus, Behçet's disease must also be considered in the differential diagnosis of subretinal neovascular membrane in young patients, especially when antibodies to oral mucosa can be shown.

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A NEW METHOD FOR RETROILLUMINATION PHOTOGRAPHY OF CATARACTOUS LENS OPACITIES

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The progressive changes of cataractous lens opacities have been usually documented by written records, sketches, photographs, slit-lamp microscope, and retroillumination techniques. The advantage of retroillumination photography in evaluating the lens opacities is the ability to record the whole pupillary space dilated on one photograph with uniform lighting.[1] It has been performed by using a conventional fundus camera[2] or slit-lamp microscope. However, the undesirable image of the light source for illumination is formed by the corneal reflex near the plane of lens opacities. Its image, superimposed on the lens opacities, probably makes the retroillumination photograph unreliable and less valuable.

We studied a new method of retroillumination photography, hoping to eliminate the specular reflection from the corneal surface.[3] Quantitative evaluation of cataractous lens opacities was done with these photographs.[4] We constructed adaptors suitable for Zeiss or Goldmann-type slit-lamp microscopes.

MATERIAL AND METHODS

In our method, the specular reflection from the corneal surface was reduced by the use of polarization. Figure 1 shows the modified part of the slit lamp. The subject's eye was illuminated by a linearly polarized beam through a Polaroid P_1. The lens opacities were observed and photographed on the same optical axis as

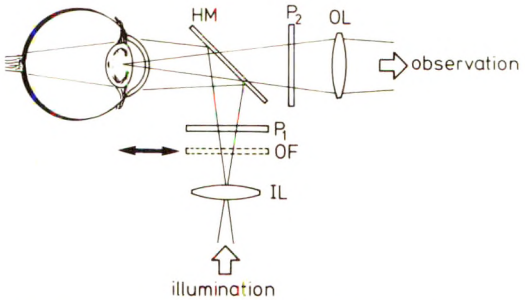


Fig. 1 (Kawara and Obazawa). Schematic diagram of the modified part of a slit-lamp microscope that eliminates specular reflection from the corneal surface in photographing cataractous lens opacities by retroillumination.

that of the illumination beam. Another Polaroid (P_2) was set behind the half mirror. The axis of polarization was at right angles to that inserted in the illumination beam. The specularly reflected light from the corneal surface retained almost the same linear polarization as the illumination light. It could not pass through the Polaroid P_2 set in the observation system and, as a result, the undesirable image mentioned previously was almost eliminated. However, the diffuse reflection scattered by the retina must be depolarized. Assuming complete depolarization, half of the diffusely reflected light passed through the Polaroid P_2. Therefore, only the cataractous lens opacities were recorded as dark shades by retroillumination photography without any undesirable image. Our method needed only a few optical elements, such as linear Polaroids inserted in the illumination and observation optical system, and a glass plate used as a half mirror, which replaced the mirror or prism occupied in the illumination system of the slit-lamp microscope.

Figure 2 shows the adaptors for photo-

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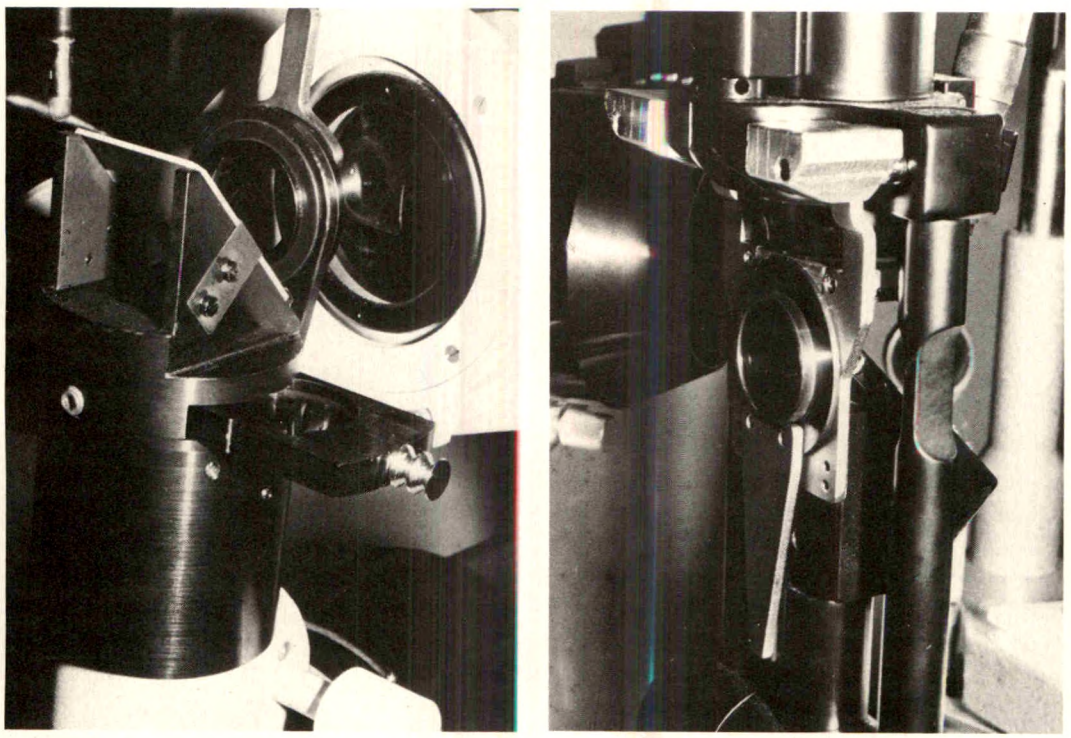


Fig. 2 (Kawara and Obazawa). Appearance of newly devised adaptors for retroillumination photography for Zeiss (left) and Goldmann (right) slit-lamp microscopes.

graphing the cataractous lens opacities by retroillumination. These adaptors were designed for Zeiss (left) and Goldmann-type (right) slit-lamp microscopes. In the retroillumination adaptor for the Zeiss slit-lamp microscope, an orange filter (OF, Fig. 1) was inserted in the illumination beam easily when photographing on a monochromatic film. This filter color-enhanced the contrast between the dark opacities and the orange background in the pupillary area, which was dilated. The photographic condition with the Zeiss slit-lamp microscope was as follows: The magnification on the film was 1.8. The energy of electronic flash was 840 W second of the maximum. The aperture size of the slit lamp was between F32 and F64 in recording on either a color film (Ektachrome ASA 200) without any filter or a monochromatic film (Tri-X) with the orange filter. Both films were developed by an ordinary photographic schedule without sensitization.

Discussion

In retroillumination photographs taken with the new adaptor, the various opacities of senile cataractous lens were clearly visible with good contrast, (Fig. 3). Radial shades (Fig. 3, top left and right), gravel shades (top right), posterior disk shade (bottom left), and vesicle shades (top left, bottom left and right) were resolved satisfactorily.

Most important in photographing opacities by retroillumination is directing the illumination beam onto the optic nerve head of the subject's eye (Fig. 1). We obtained this arrangement by moving the optical system or the fixation light of the slit-lamp microscope to where the diffusely reflected light from the retina was brightest and most uniform on the pupillary plane. Thus, the lens opacities were clearly visible.

In a subject with dense opacities or one who had difficulty fixating, such as a young child, the reflected light from the

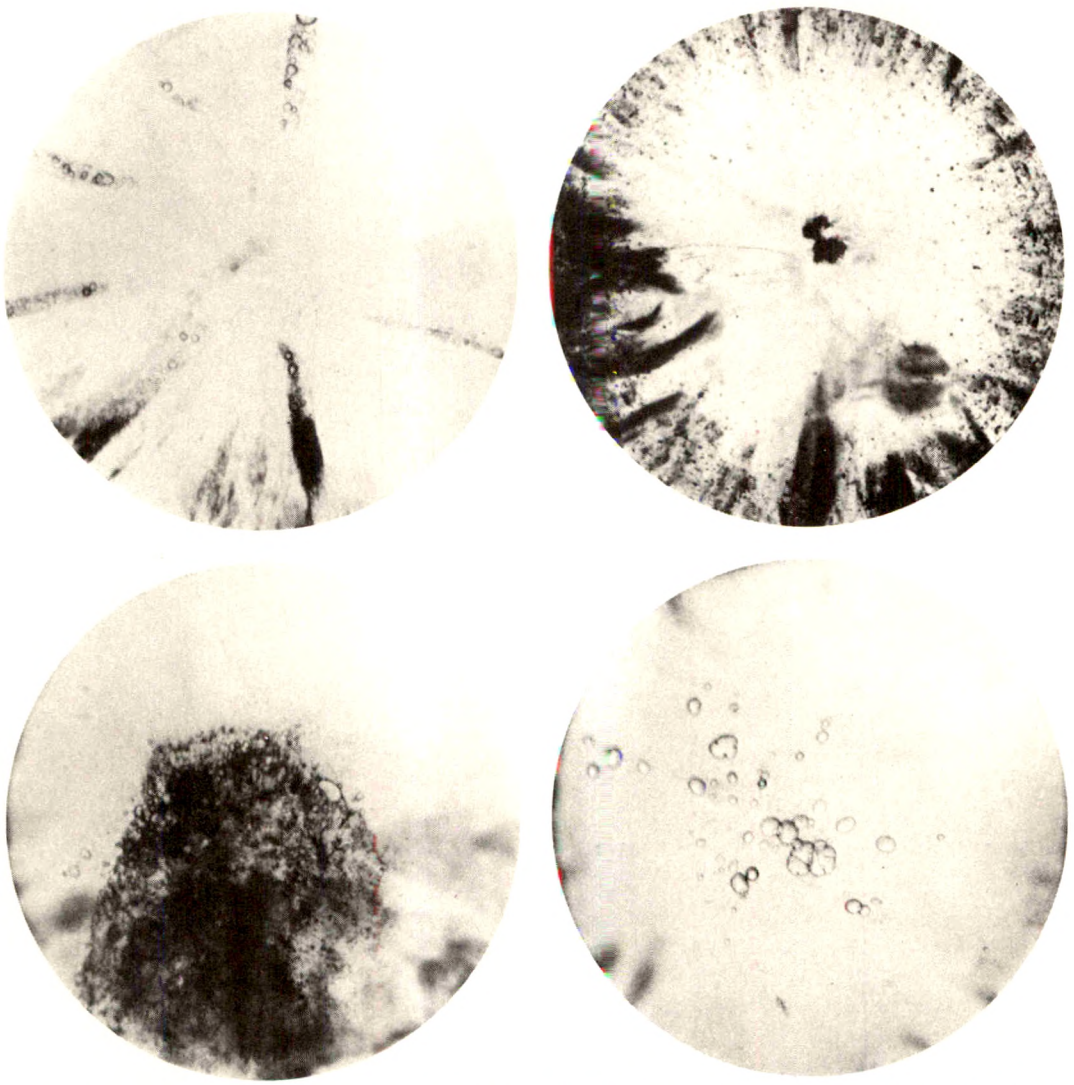


Fig. 3 (Kawara and Obazawa). Retroillumination photographs of senile cataractous lens with various opacities. Radial shades (top left and right), gravel shades (top right), posterior disk shade (bottom left), and vesicle shades (top left, and bottom left and right) are resolved.

retina did not record the opacities satisfactorily. The cross-like dark hyperbola (Fig. 4)[5,6] was conspicuous with this crossed Polaroid method. Its dark figure has been explained as interference between the ordinary and extraordinary ray of corneal birefringence caused by the lamellar structure.[5,6]

If an anisotropic medium is illuminated by circularly polarized light, the cross-like interference disappears because the vibrating plane of the light rotates with time.[6,7] Both the dark hyperbola and the specular reflection are eliminated by the crossed circular polarization. The eye is illuminated by circularly polarized light produced by using a quarter-wave plate inserted behind a Polaroid so that its slow axis is at 45 degrees to the azimuth of the Polaroid. Retroillumination of lens opacities is observed through the identical quarter-wave plate and Polaroid that are used in illumination. The polarization of the light reflected from the corneal sur-

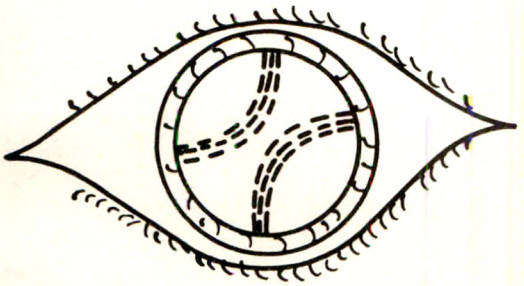


Fig. 4 (Kawara and Obazawa). Schematic figure of the cross-like dark hyperbola caused by corneal birefringence in the worst case.

face specularly is reversed in regard to the illumination light and the light can not pass through the Polaroid following the quarter-wave plate. This method, however, is effective only in the restricted case of monochromatic light because of the dependence of the quarter-wave plate on the wavelength. Even when using crossed-plane Polaroids, the dark hyperbola is invisible in most cases (Fig. 3).

The slit-image photography based on Scheimpflug's principle was developed by Brown,[8] making it possible to record the entire anterior segment of the eye. The sectional figures of cataractous lenses are also recorded and the opacities are evaluated by microdensitometer tracings. However, many sections are necessary to recognize the lens opacities. Retroillumination photography shows the lens opacities with one photograph. Opacities can also be analyzed quantitatively with a photographic density contour map obtained by using a two-dimensional density analyzer.[4]

Because the retroillumination photograph contains only two-dimensional information, we take two photographs that are focused on the anterior and posterior capsule to evaluate opacities.

Summary

We studied a new method for photographing cataractous lens opacities by retroillumination. The slit-lamp microscope was modified with an adaptor to eliminate the specular reflection from the corneal surface. We constructed two kinds of adaptors for the Zeiss and Goldmann-type slit-lamp microscopes. The cataractous lens opacities were recorded both on Ektachrome color films and Tri-X monochromatic films. In using a monochromatic film, an orange filter was inserted in the illumination beam in order to increase the image contrast. The results were satisfactory with high resolution and good contrast. We found the new device to be useful clinically for evaluating the progressive changes of cataractous lens opacities.

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LONG-TERM FOLLOW-UP OF OCCLUSION OF THE CENTRAL RETINAL VEIN IN YOUNG ADULTS

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Occlusion of the central retinal vein is a common ocular vasculopathy among the elderly. Although much has been published on this condition, uncertainties still exist about its pathogenesis, management, and course. A relationship between occlusion of the central retinal vein and systemic illnesses has been emphasized. Because systemic diseases most often occur with advancing age, it is difficult to correlate occlusion of the central retinal vein in the elderly with associated systemic problems. We did a long-term follow-up study of patients patients aged 40 years or younger who had this vasculopathy in order to delineate its course and to determine the frequency of related systemic diseases.

SUBJECTS AND METHODS

Records of patients with occlusion of the central retinal vein who were aged 40 years or younger were retrieved through the computerized medical indexing system of the Mayo Clinic and the photographic file of the Department of Ophthalmology. All patients with documented occlusion of the central retinal vein from 1949 to 1974 were selected for follow-up study. Of the 63 patients who were selected, 42 had follow-up data on morbidity and mortality. Of 27 males and 15 females included in this study, 14 were re-examined here. Follow-up information on the remaining 28 patients was acquired by obtaining the results of the patients' most recent ocular examination done elsewhere.

Re-examination of those patients who returned to the Mayo Clinic included visual acuity, external examination, slit-lamp biomicroscopy of the anterior and posterior segments, intraocular pressure, direct and indirect ophthalmoscopy, and fluorescein angiography. Additionally, their general medical history was reviewed. Every effort was made to obtain similar follow-up data for those patients who were unable to return here.

For the purpose of this study, we defined incomplete and complete occlusion of the central retinal vein by specific criteria. An incomplete occlusion of the central vein was characterized by a visual acuity of 6/18 (20/60) or better, mild-to-moderate blot and flame-shaped intraretinal hemorrhages in all quadrants, venous engorgement in all four quadrants, rare or nonexistent cotton-wool spots, and a normal or minimally swollen optic disk (Fig. 1). A complete occlusion of the central retinal vein was defined as severe visual impairment to 6/60 (20/200) or less, numerous intraretinal hemorrhages, severe venous engorgement in all quadrants, numerous cotton-wool spots, and edema of the optic disk (Fig. 2). This clinical appearance is characteristic of complete central vein occlusion. According to these criteria, 21 patients had incomplete occlusions of the central retinal vein and 21 had complete occlusions. Two patients with central vein occlusion had diabetes mellitus, without evidence of diabetic retinopathy in the fellow eye;

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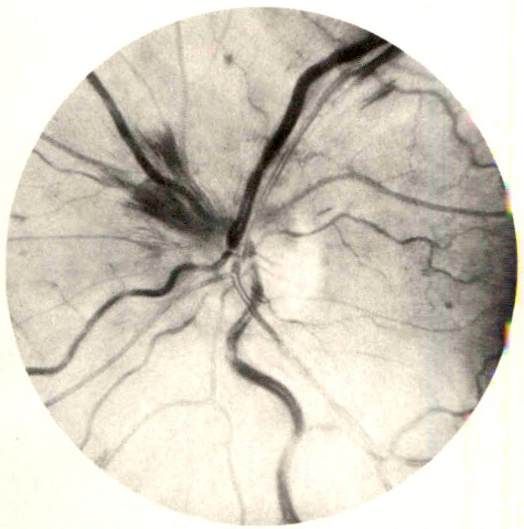


Fig. 1 (Priluck, Robertson, and Hollenhorst). Case 3. Fundus photograph showing typical incomplete occlusion of central retinal vein.

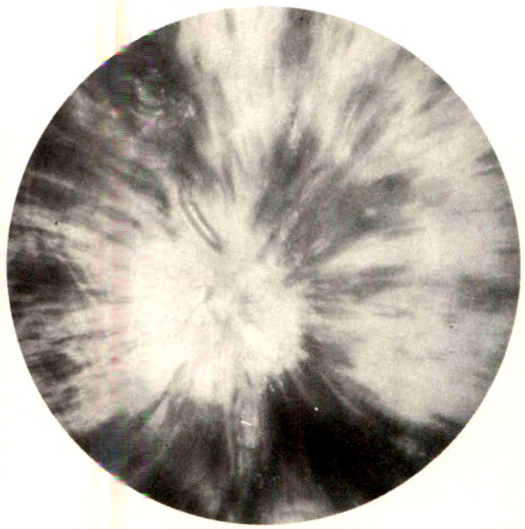


Fig. 2 (Priluck, Robertson, and Hollenhorst). Case 29. Fundus photograph showing typical complete occlusion of central retinal vein.

the unilateral nature of the pathologic changes supported the diagnosis of occlusion of the central retinal vein rather than retinopathy of diabetes mellitus.

Results

The 42 patients ranged in age from 14 to 40 years, with a mean of 31.8 years (Fig. 3). The duration of follow-up was between three and 26 years, with a mean of 14 years. The initial and follow-up results were summarized for all patients with regard to age, sex, visual acuity, systemic disease, ocular findings, and duration of the follow-up period (Table 1).

Mortality—At the conclusion of this study, 37 patients were alive and five had died (12% mortality). Of the five patients who died, three had incomplete occlusion of the central retinal vein and two had complete occlusion. Four deaths resulted from cardiovascular disease, and one death from cerebrovascular disease. The time between the diagnosis of venous occlusion and death ranged from nine to 23 years, with a mean of 15 years.

Associated systemic disorders—Of the 42 patients, eight (19%) had recognized systemic disease at the time of their initial ocular diagnosis and eight additional patients subsequently acquired a generalized chronic illness (Table 2). Three patients (7%) had hypertension and two (5%) had diabetes at the time of the initial diagnosis of occlusion of the

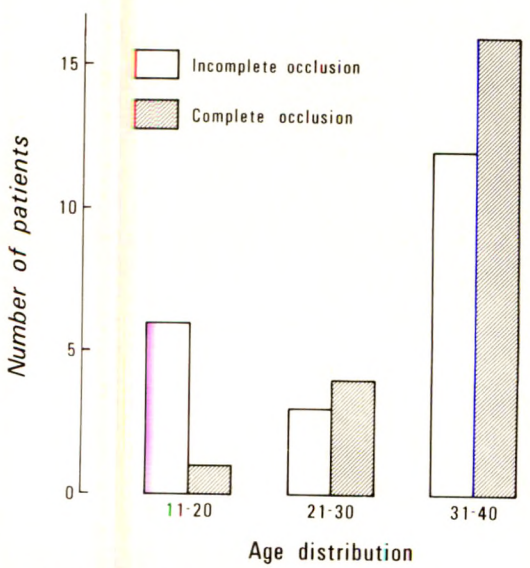


Fig. 3 (Priluck, Robertson, and Hollenhorst). Age distribution of patients with occlusion of central retinal vein.

TABLE 1

INITIAL AND FOLLOW-UP DATA OF PATIENTS WITH OCCLUSION OF THE CENTRAL RETINAL VEIN*

Case No., Sex, Age (yrs)	Type of Occlusion	Initial Visual Acuity	Initial Systemic Diagnosis	Follow-up		
				Years	Ocular Findings	Visual Acuity
1, F, 16†	Incomplete, R.E.	6/12 (20/40)	None	14	Disk vessels, macular mottling	6/6 (20/20)
2, M, 37†	Incomplete, L.E.	6/15 (20/50)	Polycystic kidneys	20	Disk vessels, macular mottling	6/24 (20/80)
3, M, 33†	Incomplete, L.E.	6/6 (20/20)	None	10	Normal	6/6 (20/20)
4, M, 16†	Incomplete, L.E.	6/6 (20/20)	None	5	Disk vessels	6/6 (20/20)
5, M, 25	Incomplete, L.E.	6/6 (20/20)	None	16	Normal	6/6 (20/20)
6, M, 30	Incomplete, R.E.	6/6 (20/20)	None	26	Normal	6/6 (20/20)
7, M, 29	Incomplete, L.E.	6/18 (20/60)	None	22	Optic atrophy, macular mottling	Counting fingers
8, M, 20	Incomplete, L.E.	6/9 (20/30)	None	12	Macular mottling	6/6 (20/20)
9, M, 39	Incomplete, L.E.	6/9 (20/30)	None	10	Normal	6/7.5 (20/25)
10, F, 35	Incomplete, L.E.	6/9 (20/30)	None	24	Normal	6/6 (20/20)
11, F, 40	Incomplete, L.E.	6/18 (20/60)	None	25	Macular mottling	6/9 (20/30)
12, M, 28	Incomplete, L.E.	6/12 (20/40)	None	10	Normal	6/6 (20/20)
13, M, 16	Incomplete, L.E.	6/9 (20/30)	None	8	Normal	6/9 (20/30)
14, M, 40	Incomplete, R.E.	6/12 (20/40)	None	12	Macular hole	6/120 (20/400)
15, F, 20†	Incomplete, L.E.	6/12 (20/40)	None	3	Normal	6/6 (20/20)
16, M, 33	Incomplete, R.E.	6/6 (20/20)	None	4	Normal	6/6 (20/20)
17, M, 19	Incomplete, L.E.	6/12 (20/40)	None	8	Macular atrophy	6/120 (20/400)
18, M, 40‡	Incomplete, R.E.	6/12 (20/40)	None	9	———	———
19, F, 39‡	Incomplete, R.E.	6/6 (20/20)	Hypertension	10	———	———
20, F, 33‡	Incomplete, L.E.	6/9 (20/30)	Hypertension	23	———	———
21, M, 40†	Incomplete, L.E.	6/12 (20/40)	None	7	Macular atrophy	6/90 (20/300)
22, F, 36†	Complete, L.E.	6/90 (20/300)	None	19	Disk vessels, macular drusen	6/6 (20/20)

23, M, 31†	Complete, R.E.	CF	None	12	Disk vessels, macular mottling	6/6 (20/20)
24, M, 31†	Complete, L.E.	6/60 (20/200)	None	23	Disk vessels, macular mottling	6/7.5 (20/25)
25, M, 30†	Complete, R.E.	LP	None	22	Disk vessels	6/6 (20/20)
26, F, 40†	Complete, L.E.	3/120 (10/400)	None	25	Optic atrophy	6/120 (20/400)
27, F, 40†	Complete, R.E.	6/60 (20/200)	Hypertension	3	Optic disk neovascularization, vitreous retinal detachment	HM
28, F, 39†§	Complete, R.E.	NLP	None	20	Vitreous traction retinal detachment	NLP
29, M, 33†	Complete, R.E.	6/60 (20/200)	None	6	Disk vessels	6/21 (20/70)
30, M, 40	Complete, L.E.	6/60 (20/200)	None	15	Optic atrophy	6/60 (20/200)
31, M, 31	Complete, L.E.	6/60 (20/200)	Polymyositis	7	Macular scar	6/90 (20/300)
32, M, 36	Complete, L.E.	6/60 (20/200)	Diabetes	11	Enucleation	———
33, F, 24	Complete, L.E.	6/60 (20/200)	None	25	Macular atrophy	6/30 (20/100)
34, M, 35	Complete, R.E.	6/120 (20/400)	None	10	Enucleation	———
35, M, 38	Complete, R.E.	6/60 (20/200)	None	13	Macular atrophy	6/30 (20/100)
36, F, 14	Complete, L.E.	HM	Diabetes	20	Enucleation	———
37, M, 39	Complete, L.E.	6/60 (20/200)	None	25	Macular atrophy	6/120 (20/400)
38, F, 25	Complete, R.E.	CF	None	12	Macular atrophy	CF
39, M, 25	Complete, R.E.	6/50 (20/200)	None	5	Macular atrophy	HM
40, M, 38‡§	Complete, L.E.	CF	None	19	———	———
41, F, 35‡	Complete, L.E.	CF	Valvular heart disease	15	———	———
42, F, 38	Complete, L.E.	4/200	None	11	Disk vessels	6/7.5 (20/25)

*CF designates counting fingers; LP, light perception; NLP, no light perception; HM, hand movements.
†Patients seen for follow-up examination at the Mayo Clinic.
‡Patients who died.
§Bilateral occlusion of the central retinal vein.

TABLE 2

SYSTEMIC DISEASES ASSOCIATED WITH OCCLUSION OF CENTRAL RETINAL VEIN

Case No.	Systemic Disease (Initial Examination)	Systemic Disease (Follow-up Examination)
2	Polycystic kidneys	Polycystic kidneys
5	None	Hypertension
11	None	Hypertension, diabetes
15	None	Ulcerative colitis
18*	None	Cardiac death
19*	Hypertension	Cardiac death, hypertension
20*	Hypertension	Cerebrovascular death, hypertension
27	Hypertension	Hypertension, angina
30	None	Pancreatitis
31	Polymyositis	Polymyositis
32	Diabetes	Diabetes
36	Diabetes	Diabetes
37	None	Osteoporosis
39	None	Pancreatitis
40*	None	Cardiac death
41*	Valvular heart disease	Cardiac death

*Patients who died.

central retinal vein. During the follow-up period, two additional patients developed hypertension. The mean duration of time between the occurrence of venous occlusion and clinically recognized hypertension for these two patients was approximately 15 years. One of these two patients also acquired diabetes mellitus, and the other developed angina pectoris. Two patients had chronic pancreatitis during the follow-up period; one of them had a history of alcoholism. One patient developed osteoporosis, and another developed ulcerative colitis.

Ocular findings—Follow-up visual acuities of eight patients were not obtained; three had had enucleation for neovascular glaucoma and five had died. Although by definition, the initial visual acuities delineated those patients with incomplete occlusion of the central retinal vein (visual acuity of 6/18 [20/60] or better) from those with complete occlusion (visual acuity of 6/60 [20/200] or worse) at the onset of their ocular disease, a significant overlap in follow-up visual acuity was noted between these two groups (Fig. 4). Of the 21 patients who had complete occlusion of the central retinal vein, nine (43%) had significant visual improvement during the follow-up period, seven (33%) had no change or a decrease in the final visual acuity, three (14%) had enucleation for neovascular glaucoma, and two (10%) had died. Of the 21 patients who had incomplete venous occlusion, 13 (24%) showed a decrease in the final visual acuity, and three (14%) had died. None of the patients with incomplete occlusion of the central retinal vein developed neovascular glaucoma. The presumed cause for poor final visual acuity in patients with incomplete venous occlusion was chronic cystoid macular edema.

Significant ophthalmoscopic findings observed at the follow-up examinations included anomalous vasculature on the optic disk and changes of the macular pigment epithelium. Anomalous vessels on the optic disk were present in six patients with complete occlusion of the central retinal vein and in three patients with incomplete occlusion. Fluorescein angiography showed the venous nature of these vessels (Fig. 5). We were not always able to determine definitively

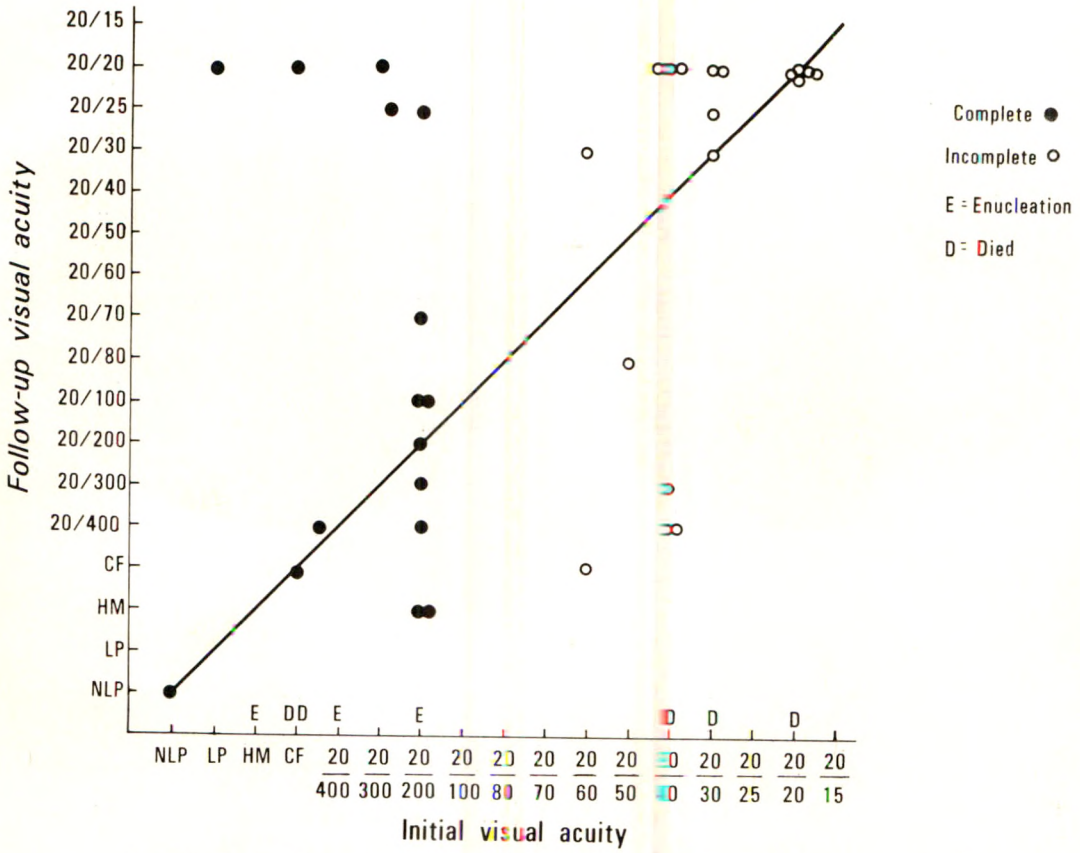


Fig. 4 (Priluck, Robertson, and Hollenhorst). Initial and follow-up visual acuity for all patients in this study. Eight patients did not have follow-up vision testing. D indicates died (five patients); E indicates enucleation for neovascular glaucoma (three patients). Patients with complete occlusions designated by solid circles; those with incomplete occlusion by open circles. Patients whose vision is noted on or below the line had decreased or unchanged follow-up visual acuities. NLP indicates no light perception; LP, light perception; HM, hand movements; and CF, finger counting.

whether these vessels were dilated capillaries that functioned as collateral channels or were true opticociliary veins. Figures 6 to 8 show the evolution of dilated collateral channels in the optic disk of a 31-year-old man who is not otherwise included in this study. The optic disk as initially photographed appeared normal (Fig. 6). The disk is shown as it appeared with an incomplete vein occlusion (Fig. 7) and six months later when anomalous disk vasculature had developed. (Fig. 8).

Disruption of the macular pigment epithelium which developed after long-term cystoid macular edema was seen in nine patients (43%) with complete venous occlusion and in seven patients (33%) with incomplete occlusion (Fig. 9). Other ocular findings included a macular hole (one patient), optic disk neovascularization with recurrent vitreous hemorrhage (one patient), nonrhegmatogenous retinal detachment secondary to vitreous traction (one patient), and optic atrophy (three patients). Each of the three patients who developed optic nerve atrophy initially had the typical appearance of acute venous occlusion, were followed up from 15 to 25 years, and retained visual

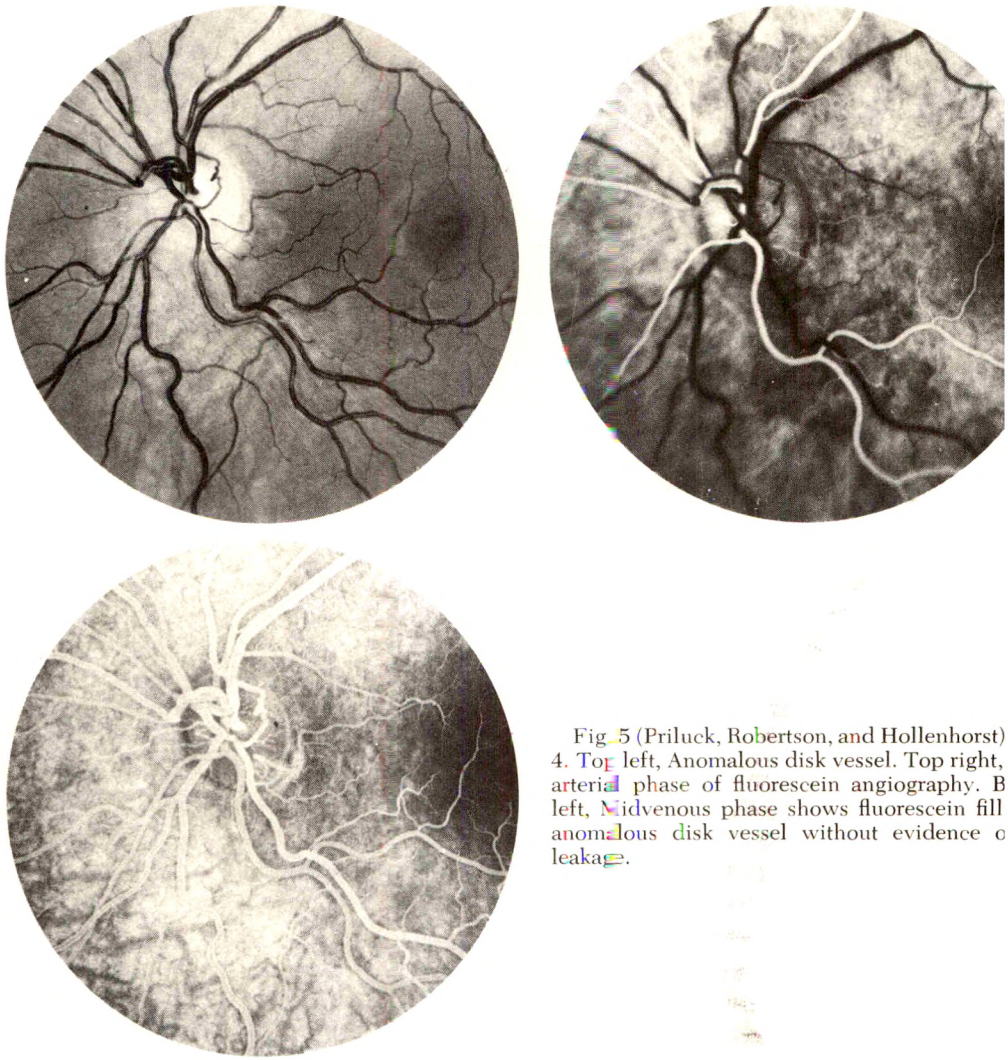


Fig. 5 (Priluck, Robertson, and Hollenhorst) 4. Top left, Anomalous disk vessel. Top right, arterial phase of fluorescein angiography. B left, Midvenous phase shows fluorescein fill anomalous disk vessel without evidence o leakage.

acuity of counting fingers or better in the absence of systemic disease. We believe the optic nerve atrophy in these cases developed as a consequence of the local vascular insult.

The incidence of involvement of the second eye was minimal. Only two patients had occlusion of the central retinal vein in the fellow eye. One (Case 28) had an incomplete occlusion of the central retinal vein of the fellow eye seven years after the initial complete occlusion. The other (Case 40) initially had a com occlusion of the central retinal vein c left eye and an incomplete occlusi the central vein of the right eye. patient had no known systemic disea the time of his initial ocular diagnosi died of a myocardial infarction 19 later at age 57 years.

No other ocular diseases occurre association with occlusion of the ce retinal vein. No patients had ch open-angle glaucoma at their initia

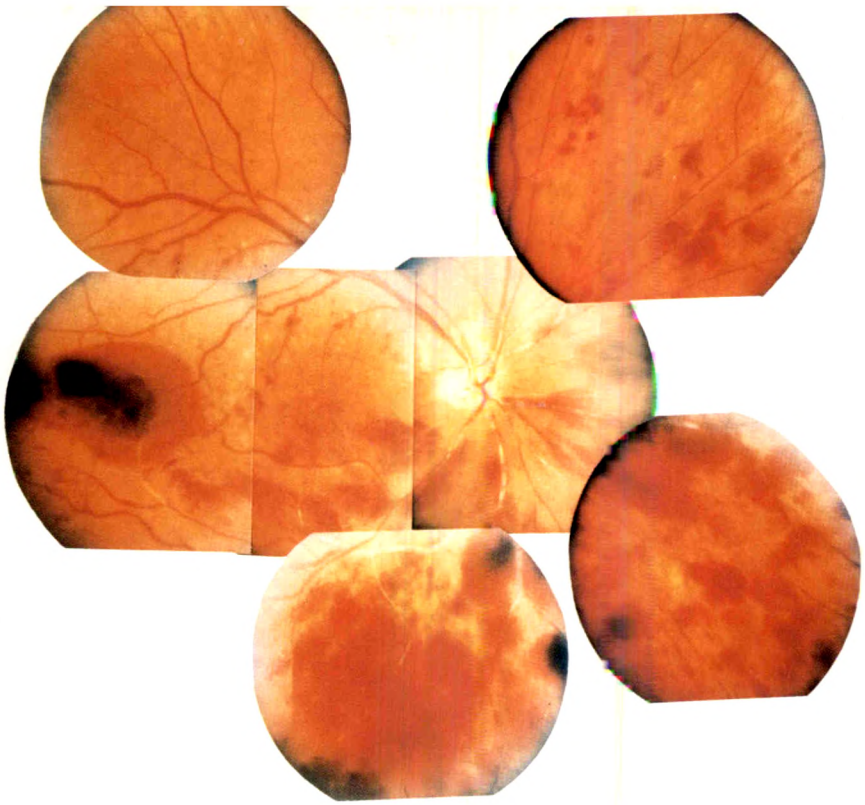


Fig. 1 (Appen, de Venecia, and Ferwerda). Montage of funcus appearance of the right eye on Aug. 10, 1977, showing optic atrophy, diffuse retinal hemorrhages, sheathing of the retinal arterioles, and distention of the retinal veins.

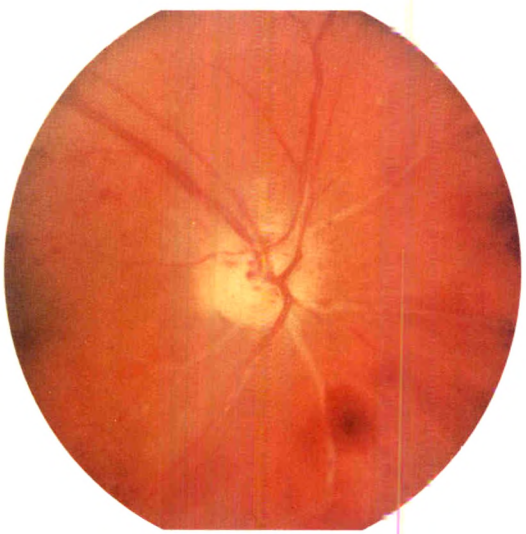


Fig 3 (Appen, de Venecia, and Ferwerda). The right fundus on Aug. 29, 1977, six weeks before enucleation.

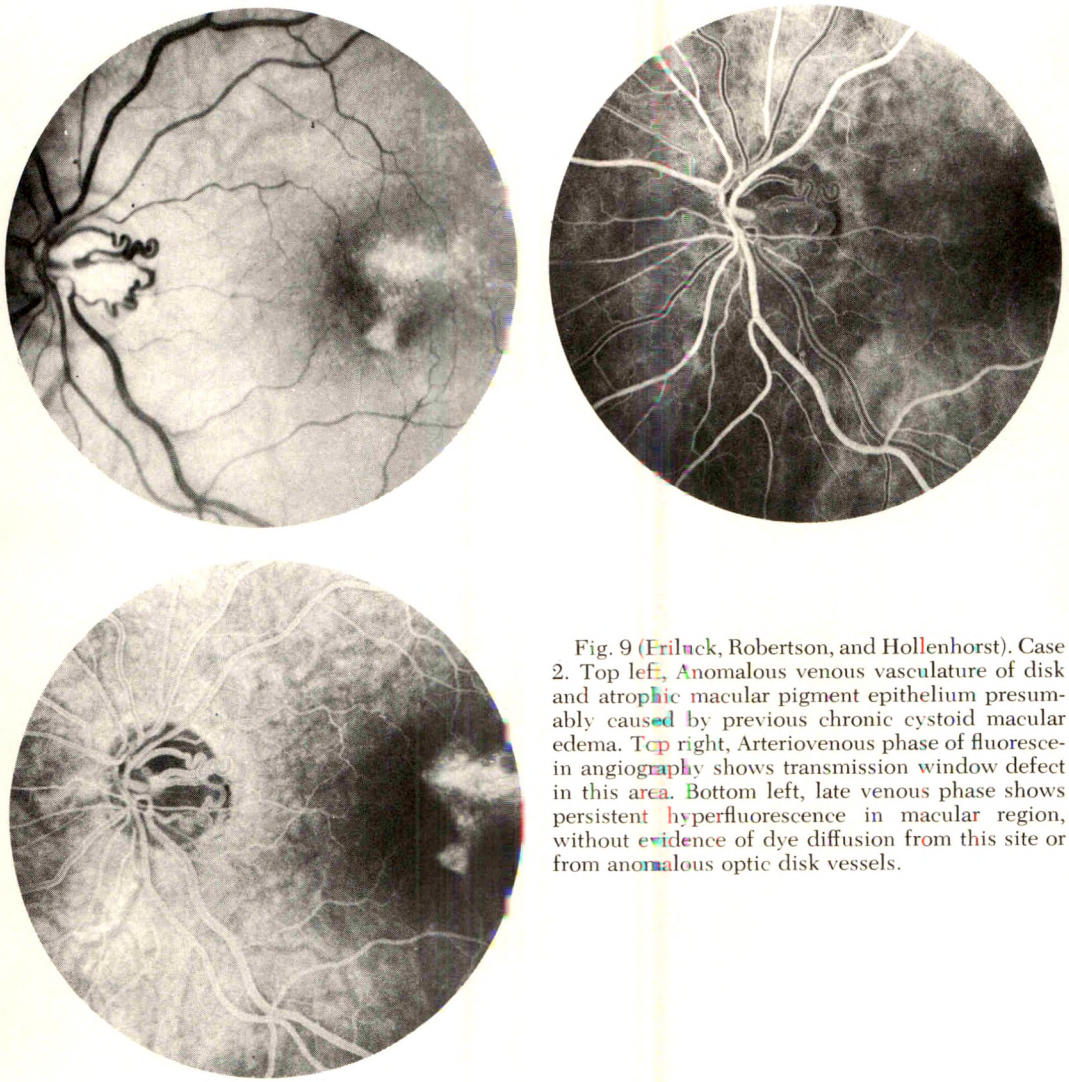


Fig. 9 (Friluck, Robertson, and Hollenhorst). Case 2. Top left, Anomalous venous vasculature of disk and atrophic macular pigment epithelium presumably caused by previous chronic cystoid macular edema. Top right, Arteriovenous phase of fluorescein angiography shows transmission window defect in this area. Bottom left, late venous phase shows persistent hyperfluorescence in macular region, without evidence of dye diffusion from this site or from anomalous optic disk vessels.

amination, and none of them developed open-angle glaucoma during the follow-up period.

Discussion

The precise pathophysiology of occlusion of the central retinal vein has remained an enigma since its first description by Michel[1] in 1878. Causal mechanisms that have been proposed for ophthalmic venous occlusive disease include the following: (1) primary disease of the venous wall, either inflammatory or degenerative; (2) compression of the vein by an adjacent sclerotic arteriole; and (3) hemodynamic alterations predisposing to vascular stagnation and thrombosis.[2,3]

There are many reports of simultaneously occurring systemic diseases associated with and predicated as the cause of retinal venous occlusion. Paton, Rubenstein, and Smith[4] reported that 56% of men and 70% of women with retinal vein thrombosis had hypertension, with dia-

stolic pressures greater than 100 mm Hg. Most patients in his study were between 50 and 70 years old. Cassady[5] reported that 46 (85%) of 54 patients had severe arteriosclerosis or hypertension or both. Additionally, retinal venous occlusion has been associated with a myriad of generalized processes including diabetes,[6] dysproteinemia,[7] hypercoagulable states,[8] and anemia.[9] Because of the many possible associated conditions, it is often difficult for the clinician to decide on the proper diagnostic studies in the absence of subjective premonitory symptoms. In our study, we determined the prevalence of associated systemic illnesses during long-term follow-up of young patients with this ocular disease. Only three patients (7%) had hypertension at the time of their initial diagnosis, and two additional patients developed hypertension during the follow-up period. Because only 42 patients were included in our study and because 5.9% of all males and 2.2% of all females between 16 and 30 years old have diastolic blood pressure levels greater than 95 mm Hg,[10] we cannot draw a significant statistical correlation between hypertension and occlusion of the central retinal vein in these young patients. Additionally, these small numbers preclude inferences as to the future development of hypertension in young persons with this vasculopathy. More importantly, few patients acquired significant systemic disease during the follow-up period. Similarly, no relationships between occlusion of the central retinal vein and the subsequent development of pancreatitis, osteoporosis, and ulcerative colitis could be postulated.

With respect to mortality, there is an apparent correlation between occlusion of the central retinal vein occurring in young persons and early death. Five patients died during the follow-up period (12%). The time between initial ocular diagnosis and death ranged from nine to 23 years, with a mean of 15 years. Two of the five patients had hypertension, and one had valvular heart disease at the initial diagnosis of occlusion of the central retinal vein. The causes of death were cardiovascular in four cases and cerebrovascular in one case. The significant mortality noted in this study is not as remarkable as the ten-year mortality of 33.3% noted by Cassady[5] or the life expectancy of 5.3 years after occlusion of the central retinal vein reported by Moore.[11] However, the latter two studies did not strictly involve young persons.

Hayreh[12,13] categorized occlusion of the central retinal vein into those with and those without associated arterial ischemia. Apparently, patients without arterial ischemia tend to be younger, have less visual impairment, and have a better visual prognosis than do patients who have both venous obstruction and concomitant arterial disease. Other investigators[4,14] have also supported the supposition that arterial ischemia is an important factor in the pathogenesis of retinal venous occlusion.

In our study, the patients were subdivided into two groups: those with complete and those with incomplete occlusion of the central retinal vein. Their initial clinical appearances clearly distinguished these two populations of young patients. In only one patient with a complete occlusion of the central retinal vein was there clinically apparent arterial ischemia at diagnosis, as evidenced by a branch arteriolar occlusion. Three other patients had optic atrophy at their follow-up examinations that we believed had developed as a consequence of the local vascular ischemia.

Although our two groups were objectively easily distinguished at the initial diagnosis, we found wide crossover between the two populations with respect to final visual acuity and ocular findings. Nine (43%) of the 21 patients with com-

plete occlusion showed improved visual acuity during this study. Other authors[5,15] have also reported useful visual acuity after retinal thrombosis. Seven (33%) of the 21 patients with incomplete occlusion showed improved visual acuity. However, five (24%) of the 21 patients with a complete and five of the 21 with incomplete occlusion showed a decrease in their visual acuities at the final examination. Anomalous vessels on the optic disk were present in some eyes of each group. All six improved patients whose initial diagnosis was that of a complete occlusion of the central retinal vein showed anomalous vasculature on the optic disk. Five of these six patients attained visual acuities of 6/7.5 (20/25) or better. We speculate that these vessels are dilated collateral shunts. Their presence seems to be a favorable sign for visual improvement in patients with complete venous occlusion. Derangement of macular pigment epithelium was commonly observed in both groups of patients. Its presence reflects previous disruption of normal macular structure associated with chronic cystoid macular edema. The greater the macular pigment derangement, the poorer was the final visual acuity.

Our study supports the findings of others[12,13] that the two types of occlusion of the central retinal vein generally can be clearly categorized by the initial clinical appearance. However, in young patients, the prognosis for final visual acuity and follow-up ocular findings, including collateral disk vasculature and disruption of macular pigment epithelium, could not be predicted on the basis of the initial clinical appearance.

This retrospective investigation was intentionally restricted to include only patients who were aged 40 years or less in an attempt to eliminate some systemic diseases such as hypertension, diabetes, and chronic open-angle glaucoma, which are so common in older age groups. Limiting the population studied according to age not only supports other reports that both complete and incomplete occlusions occur in young persons,[14] but also helps define the course of this disorder with respect to systemic disease. Our findings suggest that occlusion of the central retinal vein in young persons is not a totally benign and isolated ocular disorder. A significant number of patients had accompanying cardiovascular disease, including hypertension (7%) and diabetes (5%), at the time of their venous occlusion. Except for these two major categories of systemic diseases, there appears to be no significant association between venous occlusion and generalized illnesses. Thus, in the absence of signs or symptoms of the previously mentioned disorders, we do not recommend extensive searching for unusual systemic diseases that may be correlated with occlusion of the central retinal vein.

Summary

We did a long-term follow-up study of 42 patients aged 40 years and younger who had occlusion of the central retinal vein in order to learn its course and determine the frequency of related systemic disorders. Two groups of venous occlusion (complete and incomplete) were delineated by initial ocular findings. Final visual prognosis could not be predicted by the severity of the venous occlusion at the time of diagnosis. The presence of anomalous disk vessels closely correlated with a favorable prognosis in patients who had complete occlusion of the central retinal vein. None of the patients with incomplete central vein occlusion developed neovascular glaucoma; three (14%) of the 21 patients with complete venous occlusion developed neovascular glaucoma, which resulted in enucleation. Significant associated systemic maladies included cardiovascular disease and diabetes mellitus. An appar-

ent correlation exists between occlusion of the central retinal vein and early death.

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DIABETIC PAPILLOPATHY

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Ophthalmoscopic observation of papilledema, that is, swollen optic disks caused by increased intracranial pressure,[1] generally indicates a serious neurologic disorder. Optic nerve abnormalities that resemble the appearance of papilledema, but which are not secondary to increased intracranial pressure, include the following: pseudopapilledema, typified by optic nerve drusen or congenital optic disk anomalies without visible drusen[2]; and ocular disorders causing swelling of the optic disks, such as papillitis,[3] ischemic optic neuropathy,[4-6] papillophlebitis,[7,8] toxic optic neuropathies,[9] ocular hypotony,[10] and neoplastic infiltration of the optic nerve.[11]

Swollen optic nerves resembling papilledema can occur in association with diabetes mellitus.[12,13] We describe two patients who show what may be termed diabetic papillopathy, a syndrome in which young, juvenile diabetics develop transient edema of the optic disks with minimal impairment of the function of the optic nerves.

CASE REPORTS

Case 1—A 13-year-old girl had had diabetes mellitus for ten years, and required daily therapy of 75 units of NPH insulin. In September 1978, during a routine ocular examination, she had edema of the optic disks. She had not noted headache, visual blurring, diplopia, limb weakness, or paresthesias, but, seven months before, she had developed sudden loss of hearing in the left ear. Otologic examination showed substantial high-frequency hearing loss in the left ear, considered secondary to end-organ disease, the nature of which could not be ascertained. During the next year, moderate improvement in hearing occurred, though a significant impairment persisted.

Visual acuity was R.E.: 6/4.5 (20/15) and L.E.: 6/6 (20/20). Refractive findings were R.E.: plano $+0.50 \times 15$ degrees and L.E.: $-1.00 + 1.00 \times 175$ degrees. Ocular motility, pupil reactions, and examination of the anterior segments were normal. Intraocular pressure was 18 mm Hg in each eye. Visual fields showed enlargement of both blind spots, and a scotoma inferonasal to central fixation in the left eye (Fig. 1). Ophthalmoscopy bilaterally showed edematous optic disks, elevated 2 to 3 diopters, with moderate venous engorgement (Fig. 2). Spontaneous venous pulsations were absent. There were no new vessels at the disk, but a few fine microaneurysms were present in the macular areas. There was no macular edema and no posterior hyaloid detachment. Fluorescein angiography showed dilation of the capillaries on the disk. In the late phase of the angiogram, the disks were hyperfluorescent and the margins were blurred, indicating leakage of fluorescein into the edematous disk tissue. There was good perfusion of the capillary bed in the macular area, and a few fine microaneurysms were present.

Results of complete physical and neurologic examinations were normal except for marked loss of hearing in the left ear. Brachial blood pressure was 118/90 mm Hg. Skull roentgenograms and computed axial tomography of the brain were normal. Specifically, the size of the cerebral ventricles was normal by computed tomography.

One month after the initial examination, without treatment, the optic nerve swelling had decreased; three months later, there was only 1.0 diopter of disk elevation and the retinal veins were less engorged. Her visual acuity remained stable; the blind spots had decreased in size; and the scotoma in the left eye had resolved (Fig. 3). In November 1979, 14 months after her first examination, the patient remained well and the optic disks were flat (Fig. 4).

Case 2—A 20-year-old man had had diabetes mellitus for 17 years. His diabetes was considered to be well controlled. He was receiving 44 units of NPH insulin daily. In August 1977, he noted brief intermittent blurring of vision of both eyes.

Visual acuity was R.E.: 6/6 (20/20) and L.E.:

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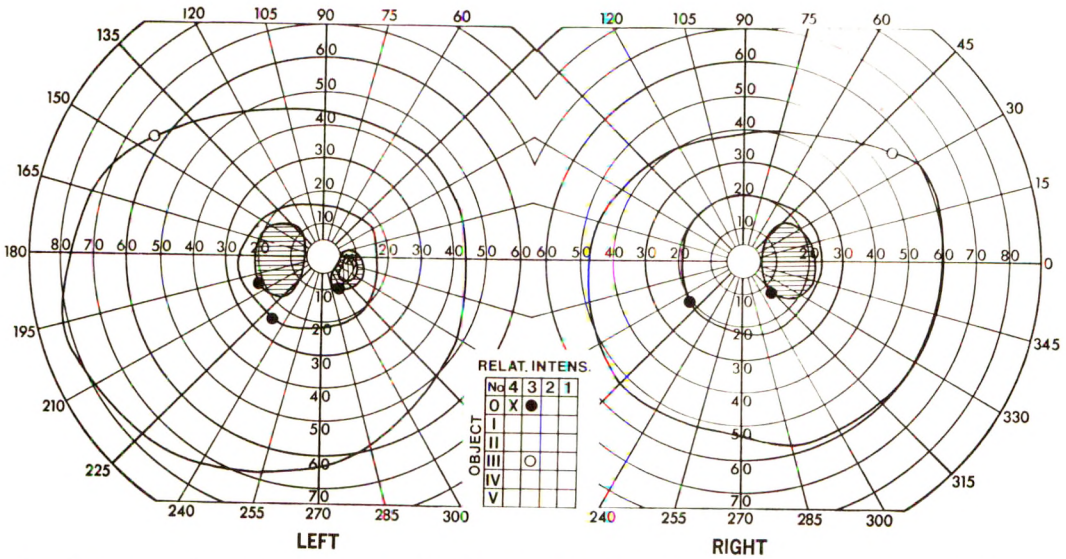


Fig. 1 (Appen and associates). Case 1. Visual fields by kinetic Goldmann perimetry on Sept. 23, 1978.

6/7.5 (20/25). Ocular motility, pupillary reactions, and anterior segment examination of both eyes were unremarkable. Intraocular pressure was 24 mm Hg in each eye. Brachial blood pressure was 112/78 mm Hg. Both fundi showed 1 to 2 diopters of disk swelling, associated with peripapillary hemorrhages and dilated retinal vessels, more marked in the right eye (Fig. 5). Otherwise, the retinas were normal. Visual fields showed enlargement of the blind spot in each eye.

One month later, without treatment, the disk swelling of the right eye had resolved; the fundus appearance of the left eye was unchanged. Two months thereafter, the abnormalities of the left fundus had resolved as well. Fourteen months after the original examination, visual acuity was R.E.: 6/6

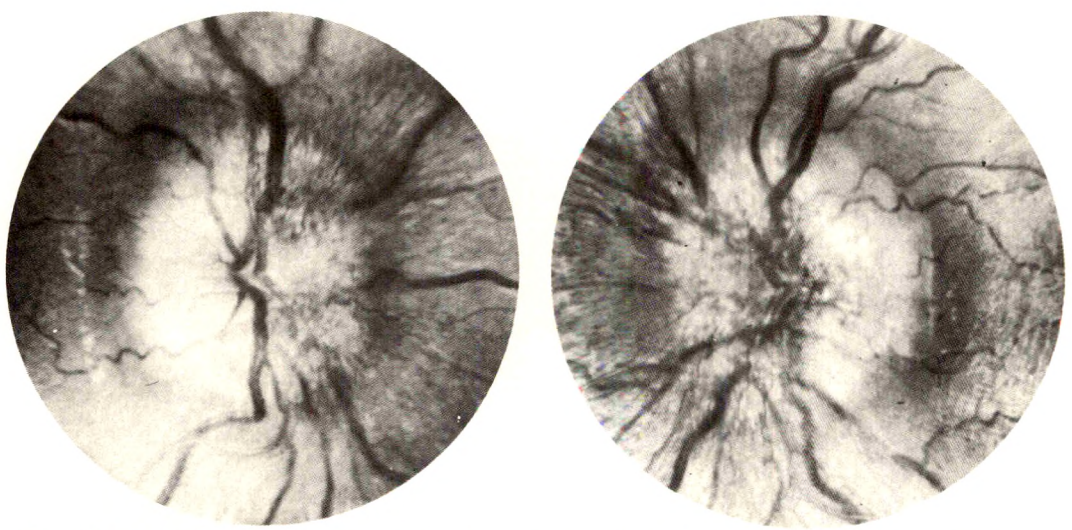


Fig. 2 (Appen and associates). Case 1. Left, Right optic disk. Right, Left optic disk on Sept. 14, 1978. There is moderate swelling of both disks with obscured vessels at disk margins and congested optic disk vasculature.

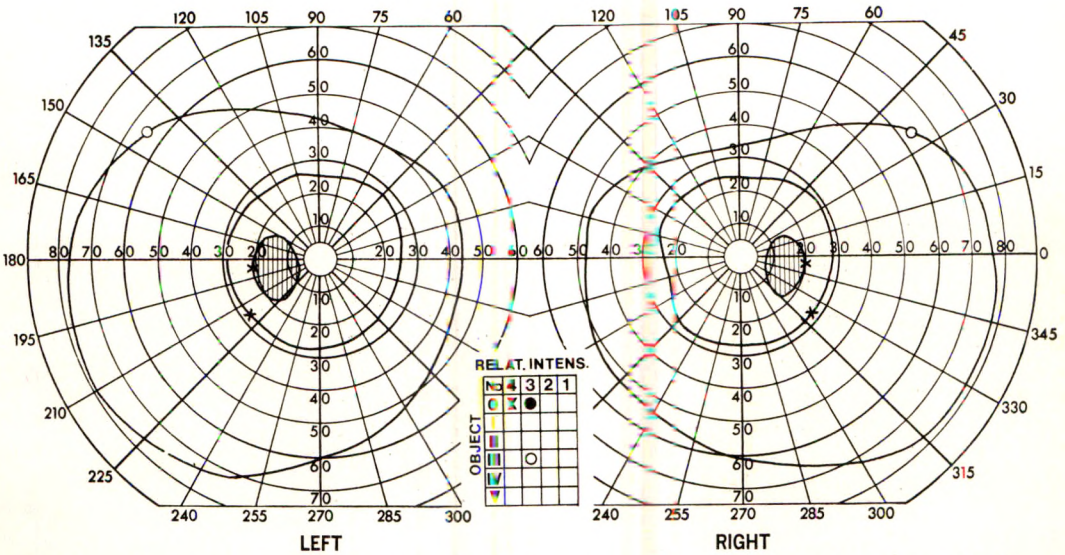


Fig. 3 (Appen and associates). Case 1. Visual fields on Dec. 21, 1978, show diminution in size of blind spots and resolution of the scotoma in the left eye.

(20/20) and L.E.: 6/12 (20/40). The visual field of the right eye showed a small paracentral scotoma in the inferior nasal quadrant, and there was mild peripheral contraction involving the inferior nasal field of the left eye (Fig. 6). The optic disks had mild pallor (Fig. 7). There was no posterior hyaloid detachment of the vitreous.

Discussion

Optic disk swelling of this type in diabetics has been attributed to ischemic optic neuropathy.[12,14] Ischemic optic neuropathy[4-6] (when not caused by giant cell arteritis) occurs in middle-aged or older adults as an abrupt onset of usually monocular visual impairment. Associated findings on examination are a sluggish pupil, a nerve fiber bundle or altitudinal visual field defect, and often segmental optic nerve swelling. Subsequently, optic

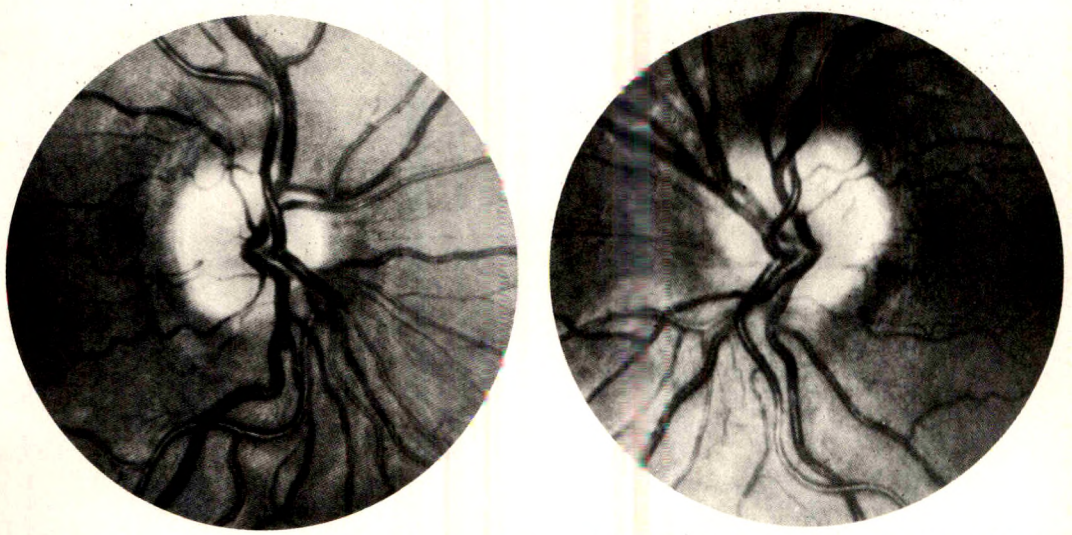


Fig. 4 (Appen and associates). Case 1. Left, Right optic disk. Right, Left optic disk on Nov. 2, 1979. The disk swelling has resolved.

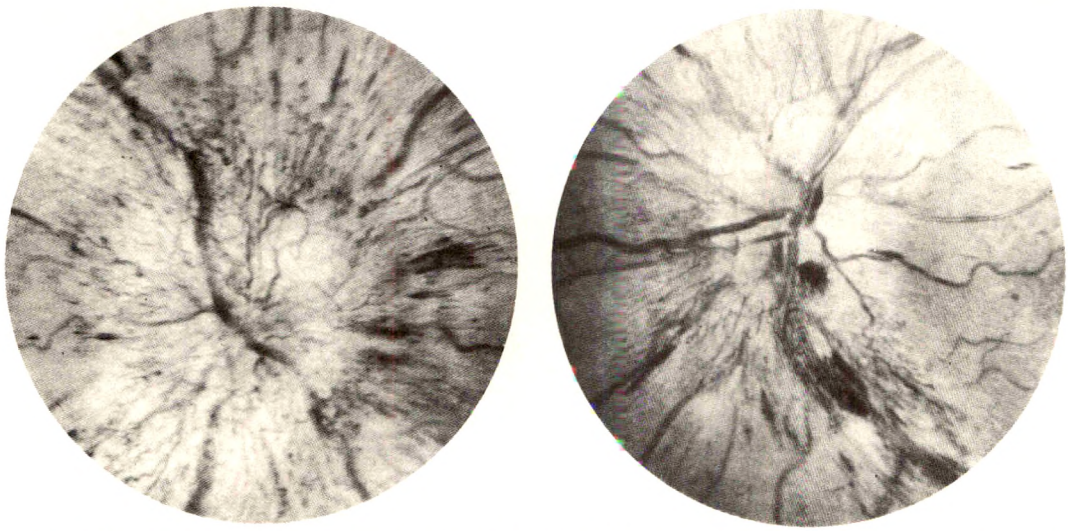


Fig. 5 (Appen and associates). Case 2. Left, Right optic disk. Right, Left optic disk on Aug. 27, 1977. Peripapillary hemorrhages and distended small vessels of the optic disks are evident.

atrophy occurs, but the visual field defect persists. The two cases reported by Freund, Carmon, and Cohen[14] in 1965 as "papilledema and papillitis in diabetes" are undoubtedly examples of ischemic optic neuropathy. However, several characteristics of our patients are atypical for this diagnosis. Our patients were much younger than is typical for patients with ischemic optic neuropathy. Additionally, ischemic optic neuropathy rarely occurs simultaneously bilaterally. Moreover, one of our patients showed no visual field loss aside from enlarged blind spots, and the other only relatively minor scotomas, contrasting with the more extensive altitudinal or nerve fiber bundle defects that occur with ischemic optic neuropathy.

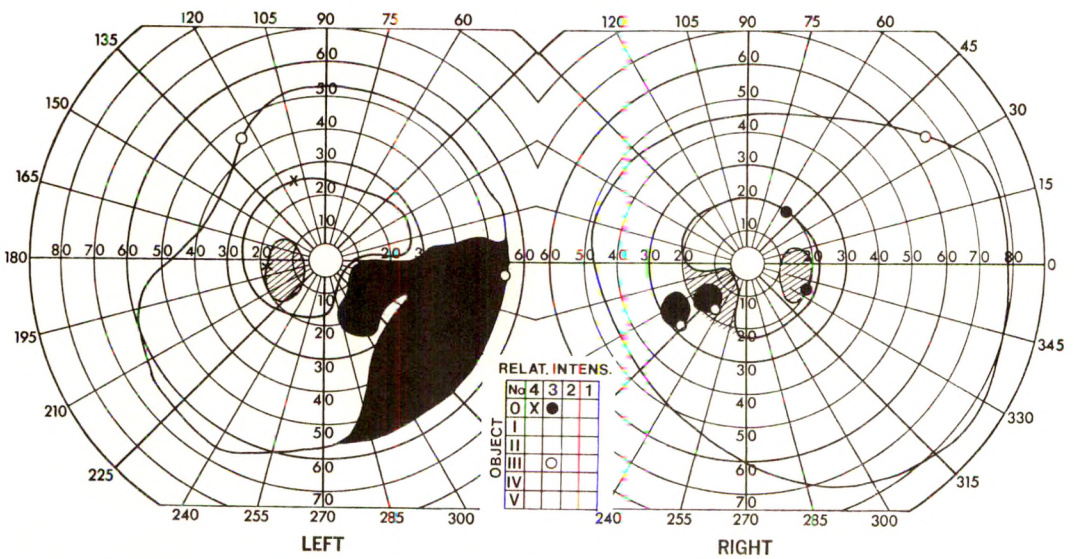


Fig. 6 (Appen and associates). Case 2. Visual fields by Goldmann kinetic perimetry on Oct. 5, 1978.

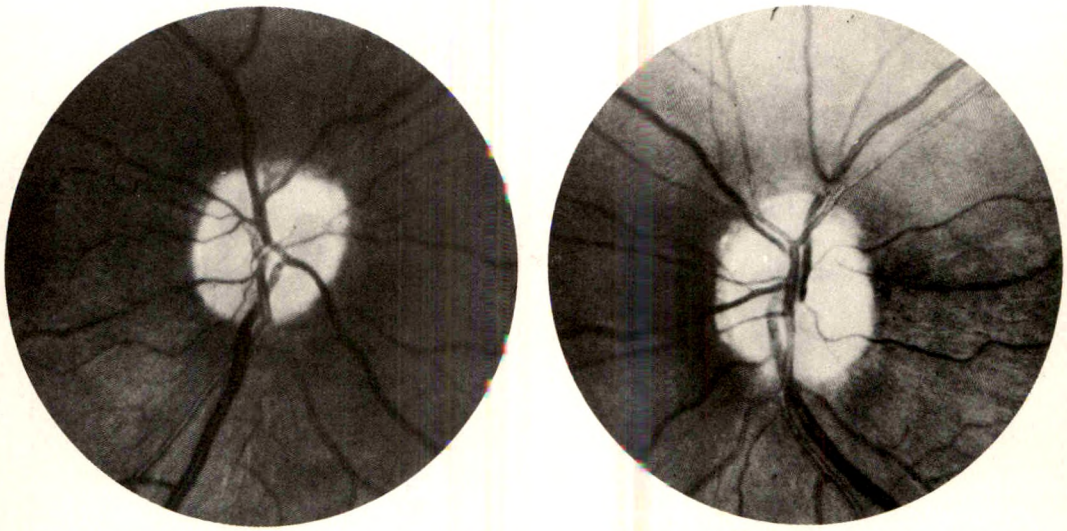


Fig. 7 (Appen and associates). Case 2. Left, Right optic disk. Right, Left optic disk on May 5, 1978. The previous optic disk swelling has resolved, with moderate residual pallor of the left optic disk.

In 1971, Lubow and Makley[12] described three patients with what they called "pseudopapilledema of juvenile diabetes mellitus," and suggested that the disease is a type of ischemic optic neuropathy. However, two of their patients (Cases 2 and 3) were essentially identical to ours, with good visual acuity and no visual field defects aside from enlarged blind spots. The first patient of their series had poor visual acuity in one eye, but optic nerve and macular edema were present. The pupil and visual field examinations were not mentioned. That patient had good recovery of visual acuity during 15 months' time. The visual field defects of ischemic optic neuropathy rarely improve so profoundly, and one suspects that the poor visual acuity was secondary to macular edema rather than to the optic neuropathy. Though visual acuity of 6/6 (20/20) is common in ischemic optic neuropathy, visual field defects other than an enlarged blind spot are invariable, and generally recovery of vision is not substantial.[4-6] Although the residual visual field defects in our second case are suggestive of ischemic optic neuropathy, the diabetic papillopathy syndrome appears to have more differences from than similarities to the usual ischemic optic neuropathy. We suspect that the pathogeneses of the two conditions are not identical.

The lack of central visual field defects and resultant impairment of visual acuity, the normally reactive pupils, and the lack of substantial symptoms of impaired vision indicate that the optic nerve changes in our patients are not a variant of demyelinizing or inflammatory papillitis. Papillophlebitis appears to be an unlikely explanation for diabetic papillopathy, because of the bilateral occurrence and lack of diffuse retinal hemorrhages. The lack of a pertinent history, the findings on examination, and the course of the condition would preclude ocular hypotony and neoplastic affections as the cause.

Pseudotumor cerebri is more difficult to exclude in retrospect, but the lack of the symptom of headache in either of our patients, and the normal size of the cerebral ventricles by computed tomography in Case 1, suggest that the intracranial pressure was not increased in our patients. In Case 2 of the patients described by Lubow and Makley,[12] the pa-

tient had "transient obscurations" of vision typical of papilledema, but is said to have had a "normal" lumbar puncture, although the exact intracranial pressure was not mentioned. In our second case, the resolution of the optic disk edema in the right eye while the left remained swollen is strong evidence against the explanation that increased intracranial pressure resulted in the optic disk changes, and indicates that the disorder is caused by a local optic disk abnormality.

Diabetic papillopathy may be distinguished from the more common diabetic retinopathy with optic disk neovascularization. During the early proliferative phase of diabetic retinopathy, fine new vessels on the disk may resemble the congested preexisting vessels seen in our patients. Fluorescein angiography cannot be used to distinguish between neovascularization of the disk and diabetic papillopathy, because the vessels in both of these conditions are permeable to fluorescein. The new vessels at the optic disk usually appear in patients with more advanced diabetic retinopathy, that is, microaneurysms, blot retinal hemorrhages, hard exudates, or cotton-wool spots, whereas these signs are minimal in patients with diabetic papillopathy. Moreover, there generally is less edema of the disk itself accompanying the new vessels in diabetic retinopathy than that present in patients with diabetic papillopathy.

Diabetics can develop accelerated hypertension with associated papilledema. Although these patients have signs of diabetic retinopathy, they generally may be distinguished from our patients by the stigmata of hypertensive retinopathy, that is, arteriovenous crossing changes, localized arteriolar narrowing, and sclerosis of vessels, as well as a markedly increased brachial blood pressure.

We suspect that these young patients with diabetic papillopathy sustain a local vasculopathy of the optic disk, presumably related to the diabetes mellitus, resulting in a transient leakage of fluid into the optic disk and swelling of the optic nerve head. There has been recent interest in derangements of axoplasmic flow as a cause of optic nerve disorders. We consider that the ophthalmoscopically visible vascular engorgement on and adjacent to the optic disk in patients with diabetic papillopathy indicates that the primary dysfunction is vascular leakage, although there could be a secondary effect on the optic nerve axoplasm dynamics.

This abnormality could be related to the retinal vascular disturbance in diabetics with retinopathy that results in macular edema. Resolution of the process in diabetic papillopathy may leave no residual impairment in the optic nerve function, as in our first case, or minimal visual field defects, as in our second. The mechanisms for the production of the vasculopathy and its resolution are still unknown. It is important for the ophthalmologist to recognize this condition so that an extensive neurologic examination of the patient may be avoided and that the condition not be misinterpreted as requiring retinal photocoagulation.

Summary

A 13-year-old girl and a 20-year-old man with juvenile-onset diabetes mellitus developed transient bilateral optic disk edema with minimal impairment of optic nerve function and with minimal diabetic retinopathy. This condition, termed diabetic papillopathy, appears to be a local optic disk vasculopathy, and generally resolves without treatment. Diabetic papillopathy should not be mistaken for papilledema or for the optic disk neovascularization of diabetic retinopathy.

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MACULAR EDEMA AFTER SCATTER LASER PHOTOCOAGULATION FOR PROLIFERATIVE DIABETIC RETINOPATHY

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Panretinal (scatter) photocoagulation appears to be beneficial for certain stages of proliferative diabetic retinopathy from the randomized clinical trial of the Diabetic Retinopathy Study.[1] In some cases visual acuity decreases after scatter photocoagulation, but usually improves after several weeks.[2] What causes this is not known for certain. Macular edema has been observed after panretinal photocoagulation but it is not clear whether this is related to the photocoagulation or is coincidental.[2] Serous detachment of the macula can also develop after scatter photocoagulation.[2] I studied the fluorescein angiographic findings of three cases of macular edema, which appeared directly related to the scatter photocoagulation for proliferative diabetic retinopathy.

SUBJECTS AND METHODS

Three patients with proliferative diabetic retinopathy underwent scatter laser photocoagulation and developed decreased visual acuity postoperatively. Fluorescein angiography and detailed ocular examinations were done to investigate the pathophysiology of the decreases in visual acuity after photocoagulation in these cases.

CASE REPORTS

Case 1—A 51-year-old man who had had insulin-dependent diabetes mellitus for 22 years was seen on July 24, 1979. He complained of a haze over the left eye, which had lasted for one week. Best corrected visual acuity was 6/7.5 (20/25) in both eyes. Results of anterior segment examination were normal in both eyes. Applanation pressures were 16 mm Hg in both eyes and no rubeosis was seen. In the left eye a vitreous hemorrhage was present slightly inferior to the disk with two flat neovascular fronds along the inferotemporal arcade. In the right eye there was a flat neovascular patch, one disk diameter in size, along the inferotemporal arcade. There were exudates and hemorrhages in the macular areas of both eyes. No vitreous separation or macular edema was observed on biomicroscopic examination in either eye. In two sessions two weeks apart the left eye had focal laser treatment to the neovascular fronds and scatter laser photocoagulation to the entire retina. A total of 1,543 burns at 0.2 second with 200- and 500-μ spot sizes at 300 to 400 mW were done. Proparacaine was used for topical anesthesia. Two weeks after completion of the laser treatment best corrected visual acuity was 6/7.5 (20/25) in the left eye with slight macular edema on biomicroscopic examination. No choroidal detachment was seen with indirect ophthalmoscopy. The patient had also started taking spironolactone for hypertension at this time.

On Oct. 17, 1979, best corrected visual acuity was 6/7.5 (20/25) in both eyes. In the left eye there was complete regression of the inferotemporal neovascular fronds, there was no macular edema seen on biomicroscopic examination, and the vitreous hemorrhage was resolving. In the right eye two small new flat neovascular patches superior to the disk and two new flat neovascular fronds along the inferotemporal arcade were observed. The old neovascular patch farther distal along the inferotemporal vein had enlarged to two disk diameters in size (Fig. 1). Because of progression of the neovascularization in the right eye, scatter laser photocoagulation was recommended. A prelaser fluorescein angiogram of the right eye on Oct. 17, 1979, showed no leakage of dye in the macula (Fig. 2).

On Oct. 19, laser photocoagulation was done using 2% lidocaine without epinephrine for retrobulbar anesthesia. The right eye had focal treatment to the neovascular patches along the inferotemporal arcade and scatter treatment to the inferior half of the retina using 903 burns at 0.2 second duration with 200- and 500-μ spot sizes at 300 to 350 mW. On Oct. 22, best corrected visual acuity was R.E.: 6/15 (20/50) and L.E.: 6/7.5 (20/25). In the right eye there was moderate foveal edema on contact lens examination. Stereoscopic fluorescein angiography showed perifoveal capillary leakage of dye without early subretinal leakage of dye in the macula (Fig. 3). No choroidal detachment was seen with indirect ophthalmoscopy. On Nov. 1, 1979, best corrected visual acuity was 6/12+3 (20/40+3) in the right eye with some foveal edema and perifoveal capillary leakage

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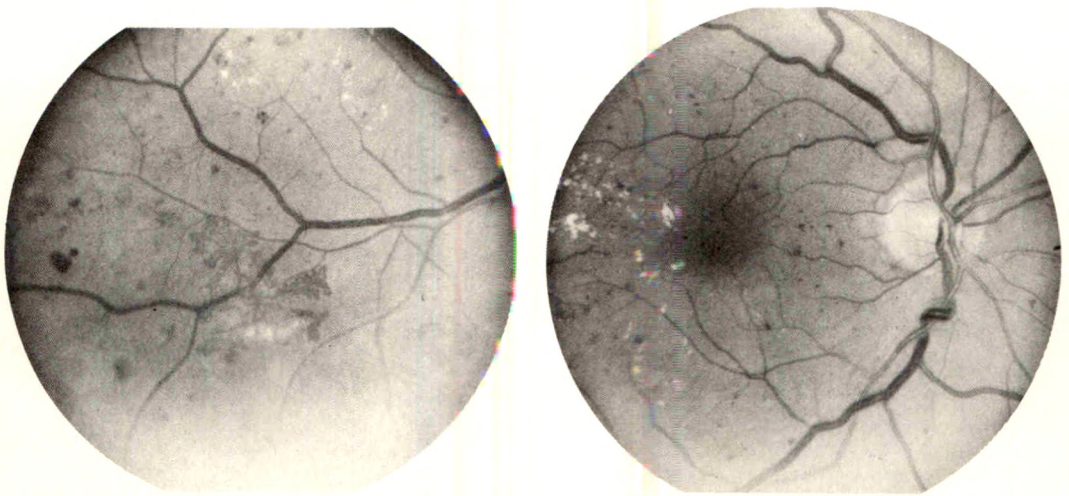


Fig. 1 (Meyers). Case 1. Right eye with moderate neovascularization along inferotemporal arcade with exudates and hemorrhages in the macula (left and right, respectively).

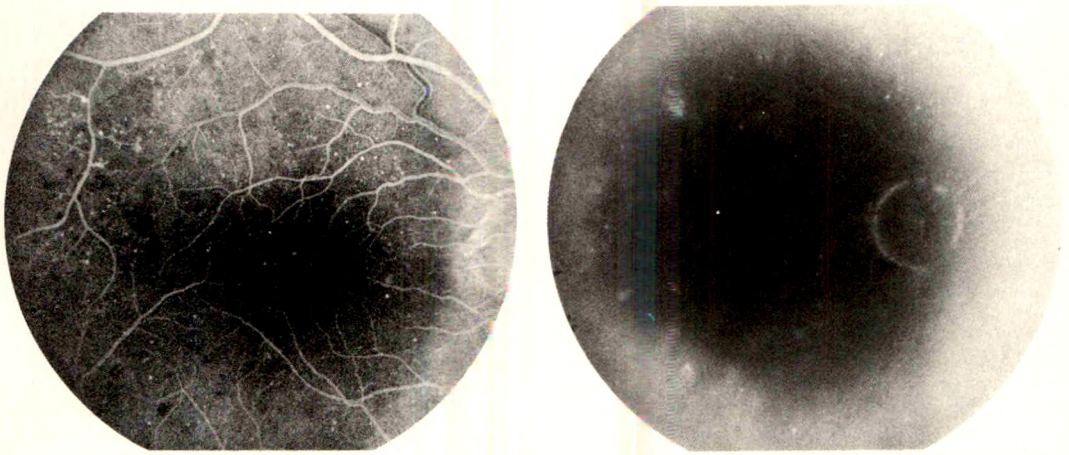


Fig. 2 (Meyers). Case 1. Prelaser fluorescein angiogram of the right eye without leakage of dye in the macula in the early (left) or late (right) phases.

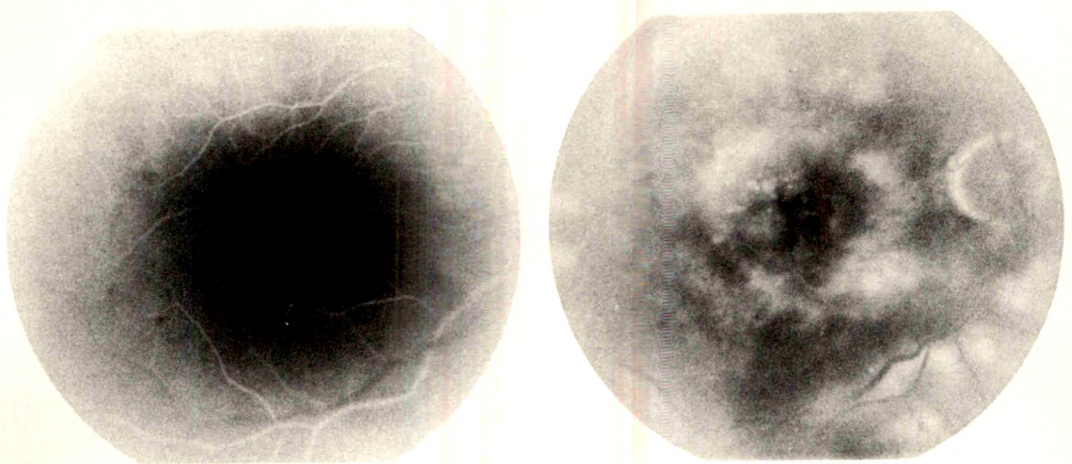


Fig. 3 (Meyers). Case 1. Postlaser fluorescein angiogram of the right eye without leakage of dye in the macula in the early arteriovenous phase (left) but with perifoveal leakage of dye in the late phase (right).

on fluorescein angiography. The neovascular fronds along the inferotemporal arcade were almost completely regressed. On Nov. 15, best corrected visual acuity was 6/12+3 (20/40+3) in the right eye with a small amount of foveal edema. Laser photocoagulation to the right eye was done on Nov. 15, using topically administered proparacaine. Focal treatment was applied to the neovascular patches superior to the disk and scatter treatment to the superior half of the retina using 554 burns at 0.1 second with 200- and 500-μ spot sizes at 400 to 450 mW. On Nov. 27, best corrected visual acuity was 6/9 (20/30) in the right eye with complete regression of the neovascular fronds superior to the disk. A small amount of foveal edema with perifoveal capillary leakage on fluorescein angiography was still present in the right eye.

When the patient was last seen on Dec. 18, 1979, visual acuity was R.E.: 6/9 (20/30) and L.E.: 6/7.5 (20/25). In the right eye there was complete regression of all the neovascular fronds with a slight amount of edema in the temporal part of the fovea on contact lens examination. Ophthalmoscopic examination of the left eye showed no neovascularization or macular edema and further resolution of the vitreous hemorrhage inferior to the disk.

Case 2—A 56-year-old man who had had insulin-dependent diabetes mellitus for 22 years was first seen on July 24, 1978, for proliferative retinopathy in the left eye. The patient had diabetic nephrosclerosis and had been undergoing hemodialysis since November 1976. The patient was taking insulin, but was taking no other medications. The patient previously had had scatter laser photocoagulation in the right eye in 1973 by another ophthalmologist. On July 24, 1978, best corrected visual acuity was R.E.: counting fingers at 2 feet and L.E. 6/7.5 (20/25). Results of anterior segment examination were normal, except for mild nuclear sclerosis and posterior subcapsular lens changes in the left eye. Applanation pressures was 14 mm Hg in both eyes, and no rubeosis was seen. The fundus of the right eye had photocoagulation scars in all quadrants with avascular fibrous tissue emanating from the disk and a glistening preretinal membrane over the macula. The left eye had flat disk neovascularization (Fig. 4) without macular edema on examination with the Goldmann contact lens. On Aug. 4, 1978, the left eye had scatter laser photocoagulation to the inferior half of the retina using topically administered anesthesia (proparacaine). A total of 891 laser burns were placed using the 500-μ spot size at 0.1 second duration and 700 to 750 mW. On Aug. 14, 1978, visual acuity was 6/9 (20/30) in the left eye. There was no fluid seen in the macula with the Goldmann contact lens. The disk neovascularization had partially regressed. The patient deferred completion of the laser treatment until Oct. 17, 1978, at which time the left eye had scatter laser photocoagulation to the superior half of the retina using topically administered proparacaine. A total of 983 laser burns were placed using the 500-μ spot size at 0.1 second duration and 600 to 700 mW. On Oct. 20, the patient complained of decreased vision with best corrected visual acuity of counting fingers at 6 feet in the left eye. There was fluid in the left macula on contact lens examination. No choroidal detachment was seen with indirect ophthalmoscopy. There was leakage of dye from perifoveal retinal capillaries without early subretinal leakage on the stereoscopic fluorescein angiogram (Fig. 5). On Dec. 4, 1978, the best corrected visual acuity in the left eye was 6/15 (20/50) without macular edema on biomicroscopic examination and nearly total regression of the disk neovascularization. On Jan. 29, 1979, visual acuity in the left eye was 6/12 (20/40). The macula had no edema on contact lens examination. On April 2, 1979, visual acuity was 6/12 (20/40) in the left eye with depigmented spots in the fovea. A tiny remnant of the neovascular tuft on the disk was unchanged from the previous examination. On July 23, 1979, visual acuity was 6/12 (20/40) in the left eye and results of an examination of the left fundus were unchanged.

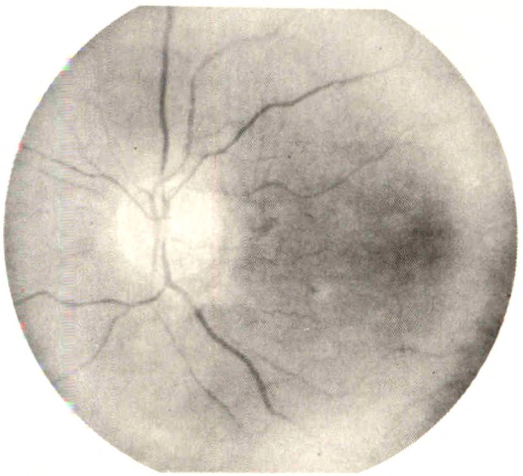


Fig. 4 (Meyers). Case 2. Left eye with neovascularization extending past the temporal edge of the disk.

Both laser treatments to the left eye were done on the mornings of days when the patient was not undergoing hemodialysis.

Case 3—A 64-year-old man was referred for evaluation of diabetic retinopathy. The patient had had insulin-dependent diabetes mellitus for 18 years but had been taking insulin for only ten years. The patient also had congestive heart failure and was taking furosemide (Lasix) and digoxin. The patient was first seen on Feb. 2, 1979. Best corrected visual acuity was R.E.: 6/7.5-1 (20/25-1) and L.E.: 6/6-2 (20/20-2). Results of examination of the anterior segment were normal. Ophthalmoscopic examination showed mild and moderate neovascularization of the disk in the right eye and left eye respectively (Fig. 6), with vitreous hemorrhage inferiorly in the right eye and cotton-wool lesions near the disk in the left eye. No macular edema was present in either eye on examination with the Goldmann contact lens. On

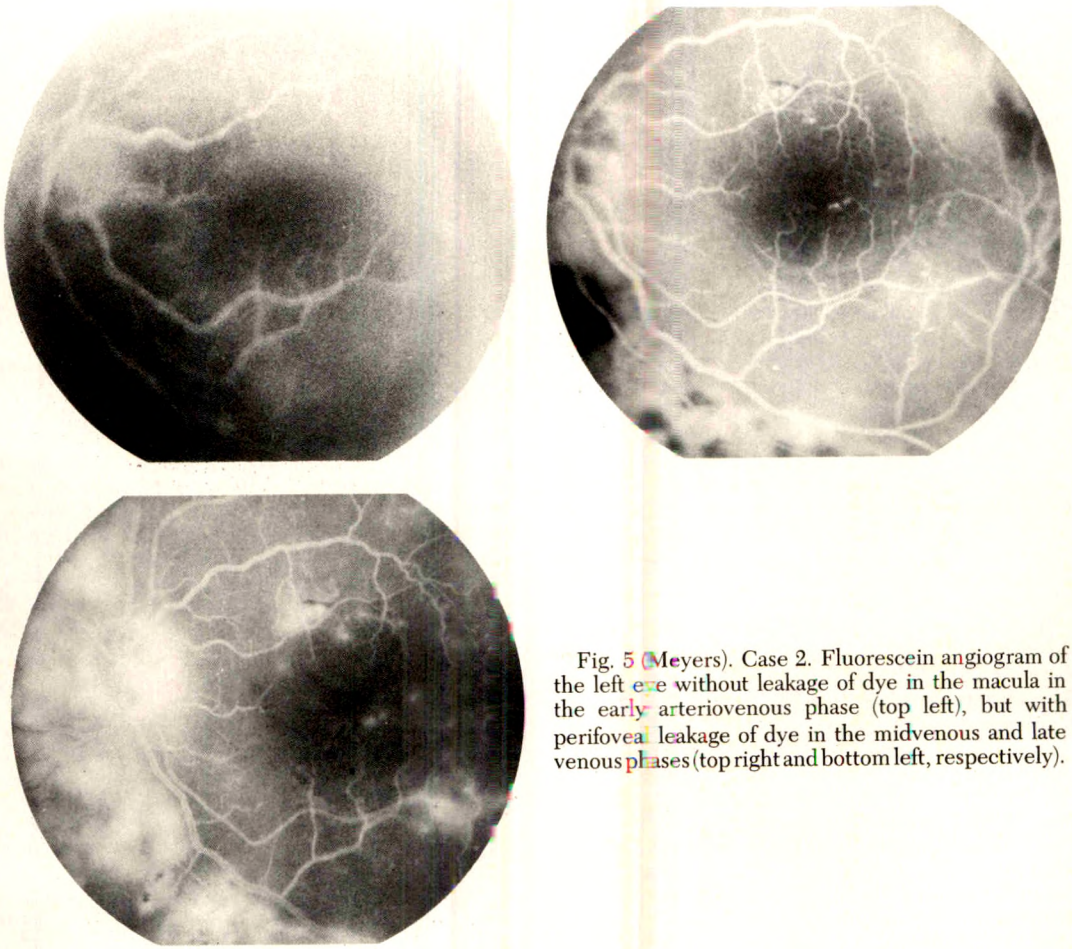


Fig. 5 (Meyers). Case 2. Fluorescein angiogram of the left eye without leakage of dye in the macula in the early arteriovenous phase (top left), but with perifoveal leakage of dye in the midvenous and late venous phases (top right and bottom left, respectively).



Fig. 6 (Meyers). Case 3. Right eye with mild neovascularization in the inferior and central areas of the disk (left), and left eye with moderate neovascularization of the inferior temporal and superior temporal areas of the disk (right).

Feb. 5, 1979, the right eye had scatter laser photocoagulation to the inferior half of the retina using retrobulbar anesthesia with 2% lidocaine without epinephrine. A total of 966 laser burns were placed using the 500-μ spot size at 0.1 second duration and 700 to 800 mW. On Feb. 13, visual acuity decreased to 6/15 (20/50) in the right eye with edema in the fovea and inferior to the fovea in a two-disk-diameter area. No choroidal detachment was seen with indirect ophthalmoscopy. Stereoscopic fluorescein angiography showed leakage of dye from perifoveal retinal capillaries without early subretinal leakage (Fig. 7). The neovascularization of the disk had almost regressed. On Feb. 27, visual acuity was 6/21 (20/70) in the right eye with persistent foveal edema, but there was resolution of the retinal edema inferior to the fovea. Stereoscopic fluorescein angiography again showed perifoveal capillary leakage. On March 27, visual acuity was R.E. 6/9-2 (20/30-2) without macular edema on Goldmann contact lens and L.E.: 6/7.5 (20/25). The disk neovascularization had almost regressed in the right eye. Because of the moderate disk neovascularization and cotton-wool lesions, the left eye had scatter laser photocoagulation on March 27, to the nasal area of the retina from the equator to within one-half disk diameter of the disk. A total of 550 laser burns were placed at 0.2 second duration using the 500-μ spot size at 300 to 400 mW. Topically administered proparacaine was used for anesthesia.

On April 25, 1979, best corrected visual acuity was R.E.: 6/7.5-2 (20/25-2) and L.E.: 6/6-3 (20/20-3) without macular edema on biomicroscopic examination in either eye. The left eye had laser photocoagulation with topically administered proparacaine at 0.2 second duration with the 500-μ spot size at 350 mW. A total of 787 burns were placed inferiorly and inferotemporally. On April 27, best corrected visual acuity was 6/9 (20/30) in both eyes. There was macular edema with a foveal cyst in the left eye and no macular edema in the right eye on biomicroscopic examination. Stereoscopic fluorescein angiography showed perifoveal leakage of dye from retinal capillaries without early subretinal leakage in the left eye (Fig. 8).

On May 10, 1979, visual acuity was 6/9 (20/30) in both eyes. Because of a persistent small tuft of neovascular tissue on the disk in the right eye, additional scatter laser photocoagulation with topically administered proparacaine was done. Laser burns totalling 404 burns at 0.2 second using the 500-μ spot size at 400 mW were placed temporally and superotemporally. On May 15, visual acuity decreased to R.E.: 6/15-2 (20/50-2) and L.E.: 6/12-1 (20/40-1). On biomicroscopic examination there was macular edema in both eyes. Fluorescein angiography showed perifoveal retinal capillary leakage of dye in both eyes. The patient was taking 325 mg of aspirin orally three times daily. On May 29, visual acuity was R.E.: 6/12 (20/40) and L.E.: 6/9 (20/30). In the right eye the disk neovascularization had entirely regressed and no macular edema was seen with the Goldmann contact lens. In the left eye the foveal edema and the cyst persisted and disk neovascularization was still present. The aspirin was discontinued, and no further photocoagulation was recommended at that time. On July 27, 1979, visual acuity was R.E.: 6/9-2 (20/30-2) and L.E.: 6/7.5-2 (20/25-2). In the right eye there was no disk neovascularization, and no macular edema was seen on biomicroscopic examination. In the left eye the foveal edema and cyst were gone but residual disk neovascularization was still present. Close observation was recommended.

Discussion

The stereoscopic fluorescein angiograms in these patients showed leakage from the perifoveal retinal capillaries

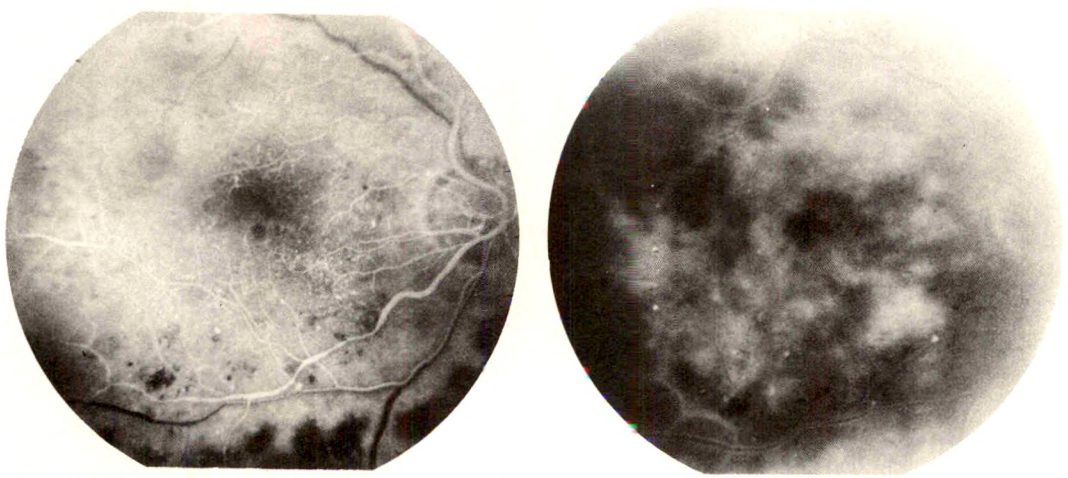


Fig. 7 (Meyers). Case 3. Fluorescein angiogram of the right eye, without leakage of dye in the macula in the early arteriovenous phase (left) but with perifoveal leakage of dye in the late phase (right).

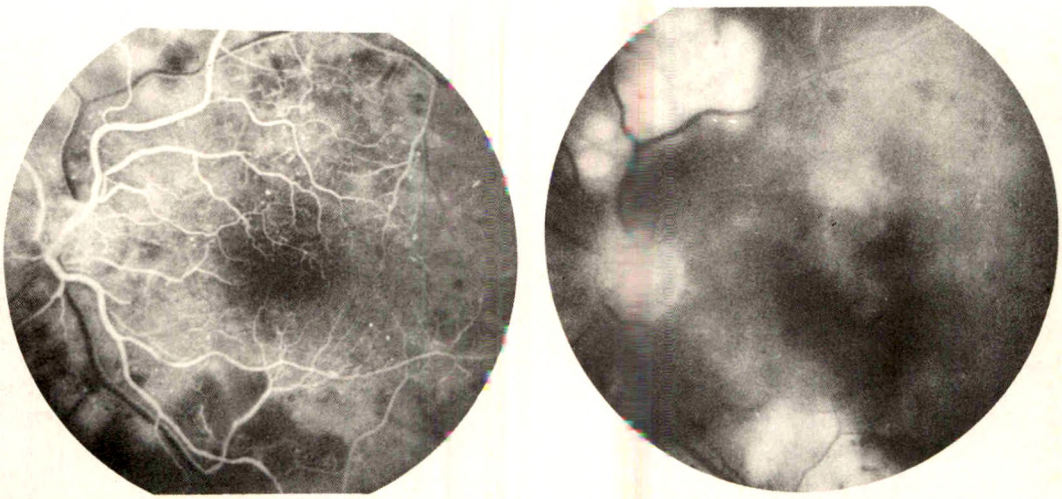


Fig. 8 (Meyers). Case 3. Fluorescein angiogram of the left eye without leakage of dye in the macula in the early arteriovenous phase (left), but with perifoveal leakage of dye in the late phase (right).

without early subretinal leakage of fluorescein in the macula. The type of anesthesia used, topically administered or retrobulbar, did not seem to be important. The visual acuity in these patients improved over several weeks.

A prelaser fluorescein angiogram in the right eye in Case 1 showed no perifoveal leakage. In the other cases and in the left eye in Case 1 no macular edema was seen before laser treatment on Goldmann contact lens examination, although prelaser fluorescein angiograms were not done. Even if there were a slight amount of subclinical macular edema before laser treatment in the left eye in Cases 1 to 3, the dramatic appearance of macular edema on biomicroscopic examination with perifoveal capillary leakage on fluorescein angiography, a few days after scatter laser photocoagulation, suggests that the scatter laser treatment was an important factor in the pathogenesis of the macular edema. In the left eyes in Cases 2 and 3 the macular edema occurred only after the second laser treatment. Additionally, in Case 3, macular edema occurred in the right eye after both laser treatments.

A number of factors may have been responsible for the macular edema. Perhaps, predisposed, labile perifoveal retinal capillaries react to the "photocoagulation-induced inflammation" with leakage caused by further endothelial dysfunction. This may be analogous to cases of macular edema after retinal detachment surgery in phakic and aphakic eyes.[3,4] Similarly, other investigators have theorized that aphakic cystoid macular edema may be related to retinophlebitis.[5] However, this hypothesis does not seem to explain completely the occurrence of macular edema in the left eyes in Cases 2 and 3 only after the second laser treatment, and not after the first treatment. Although there were somewhat more laser burns used in the second treatments in the left eyes in Cases 2 and 3, macular edema occurred after both laser treatments in the right eye in Case 3 despite a significantly reduced number and intensity of the laser burns in the second treatment. Furthermore, the effect of aspirin, a drug with some anti-inflammatory actions, was equivocal in Case 3—the macular edema resolved in the right eye but persisted in the left eye after two weeks of aspirin therapy.

Alternatively, the hemodynamics of the

retinal circulation may possibly be altered at times after photocoagulation in certain retinal areas, resulting in perifoveal capillary leakage. Diddie and Ernest[6] hypothesized that photocoagulation may induce an acute hyperemia to account for the acutely increased retinal oxygen levels over laser lesions in monkeys.[6]

All three patients had other cardiovascular and renal abnormalities. In Case 2 the patient was undergoing hemodialysis and had a more severe decrease in visual acuity after the second laser treatment than occurred in the other cases. However, it is not known whether patients with cardiovascular and renal disorders are more likely to develop macular edema after scatter laser photocoagulation or whether patients undergoing hemodialysis are predisposed to more severe reduction in visual acuity if macular edema occurs after scatter laser photocoagulation.

The decrease in visual acuity after scatter laser photocoagulation for proliferative diabetic retinopathy does not seem to occur in most patients, and, if visual acuity does decrease, it usually returns almost to pretreatment levels. Altering the intensity, duration, and number of photocoagulation burns for subsequent treatment may be considered in those patients who developed macular edema after scatter laser photocoagulation, although this did not appear to be effective in the right eye in Case 3. However, decreasing the number and intensity of laser burns in the second treatment of the right eye in Case 3 may have lessened the severity of the subsequent macular edema. Alternatively, postponing further laser photocoagulation in certain patients should be considered.

Summary

Fluorescein angiography showed perifoveal capillary leakage in three cases of macular edema after scatter laser photocoagulation for proliferative diabetic retinopathy. The scatter laser photocoagulation in these cases may have been an important factor in the pathogenesis of macular edema.

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CHOROIDORETINAL VASCULAR ANASTOMOSES

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Both arteriolar and venous choroidoretinal anastomoses have been documented.[1-4] The associated etiologic conditions include a variety of congenital and acquired diseases. We studied three cases: two of acquired venous choroidoretinal anastomosis, and one of a congenital arterial anastomosis. All represent, to our knowledge, previously unreported congenital and acquired causes and all allow pertinent observations on the vascular dynamics relating to the development and progression of such vascular aberrations.

CASE REPORTS

Case 1—A 33-year-old woman was referred to one of us (M.M.S.) when she moved to North Carolina in 1977. Before that time, her ocular problem had been diagnosed as presumed ocular histoplasmosis. In 1975, she had required photocoagulation for a paramacular subretinal neovascularization in the right eye occurring at the site of an atrophic histoplasmosis spot.

Her best obtainable visual acuity was 6/6 (20/200) in the right eye and 6/6 (20/20) in the left eye. Our initial examination included a rapid-sequence fluorescein angiogram to assess the cicatrix in the right macula. We also found an unusual choroidoretinal venous anastomosis in the left inferior retina (Fig. 1). The proximal segment of the involved vein between the histoplasmosis spot and the major arcade vein was of slightly smaller caliber than the distal segment (Fig. 1, top left). Ten months later, fundus examination showed marked atrophy of that same segment of vein (Fig. 1, middle left). Fluorescein angiography later verified the clinical impression of decreasing vascular flow through the vessel segment and suggested progressive enlargement of the venous anastomosis and increased vascular flow through the choroidoretinal anastomotic vessel (Fig. 1, middle right and bottom left).

Case 2—A 45-year-old woman was referred to the Bowman Gray Medical Center Ophthalmic Clinic in November 1974 for evaluation of her diabetic retinopathy. Radiologic and endocrine studies had established that she had adult-onset diabetes mellitus with empty-sella syndrome and related systemic hypertension.

Her best visual acuity was 6/15 (20/50) in the right eye and 6/15 (20/60) in the left eye. Fundus photographs and fluorescein studies indicated a bilateral background diabetic retinopathy (Fig. 2, top left and top right).

In an effort to reduce the cystoid macular edema in the right eye, we applied argon laser photocoagulation along the arcade vessels and temporal to the macula in a C shape. From that time through 1979, there was a worsening of her systemic hypertension and slow deterioration of her vision. Fundus photographs (Fig. 2, middle left) taken in 1979 showed marked elevation of the macular area, wrinkling of the internal limiting membrane, many old photocoagulation marks along the superior arcade vessels, and a large tortuous vein filling proximally from the dependent perimacular capillary bed. By the midvenous phase of fluorescein angiography (Fig. 2, bottom left), that vessel was rapidly draining into the choroidal circulation through an enlarged avascular zone in the foveal capillary network.

Case 3—A 37-year-old woman with pseudoxanthoma elasticum and peripapillary angioid streaks was referred to one of us (M.M.S.) for examination. Because of a large cicatricial lesion involving the macula, visual acuity in her right eye was only finger-counting at one foot. Rapid-sequence fluoroangiography of the left eye, performed to delineate the distribution of angioid streaks around the optic disk and establish the absence of subretinal neovascularization, showed an unusual arterial vascular pattern in which the arterial branch associated with the disk was assumed to be a cilioretinal artery. The hemodynamics of this anastomosis were not clarified until the patient was reexamined for metamorphopia in the left eye approximately three years later (Fig. 3, top left). At that time, fluorescein angiographic studies, performed to evaluate the extent of a subretinal neovascular complex superior to the fovea, clearly showed arterial retinal flow from a proximal branch of the superior temporal retinal artery into the choroidal vasculature by way of an anastomosis through the optic disk (Fig. 3, top right, bottom left, and bottom right).

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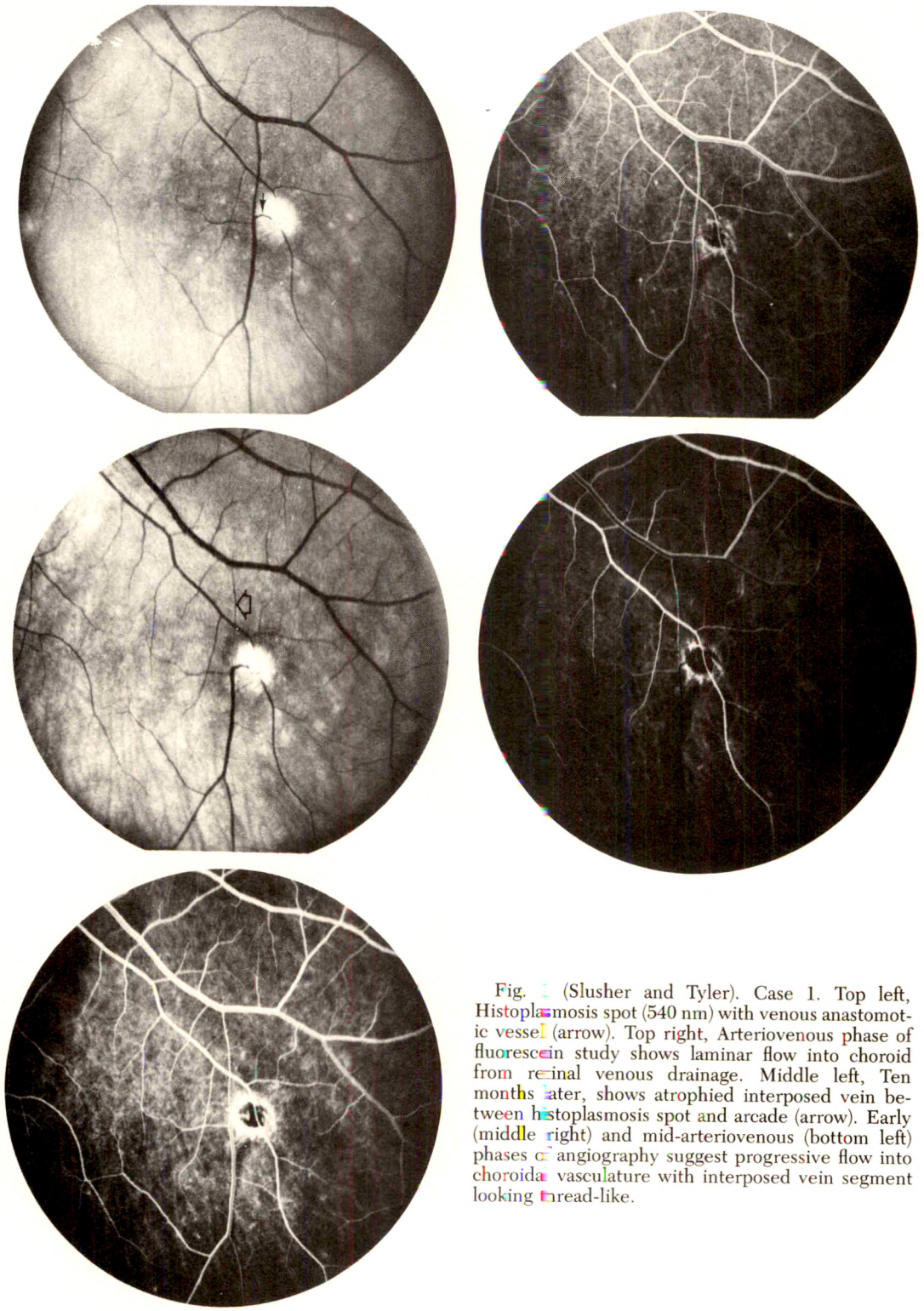


Fig. 1 (Slusher and Tyler). Case 1. Top left, Histoplasmosis spot (540 nm) with venous anastomotic vessel (arrow). Top right, Arteriovenous phase of fluorescein study shows laminar flow into choroid from retinal venous drainage. Middle left, Ten months later, shows atrophied interposed vein between histoplasmosis spot and arcade (arrow). Early (middle right) and mid-arteriovenous (bottom left) phases of angiography suggest progressive flow into choroidal vasculature with interposed vein segment looking thread-like.

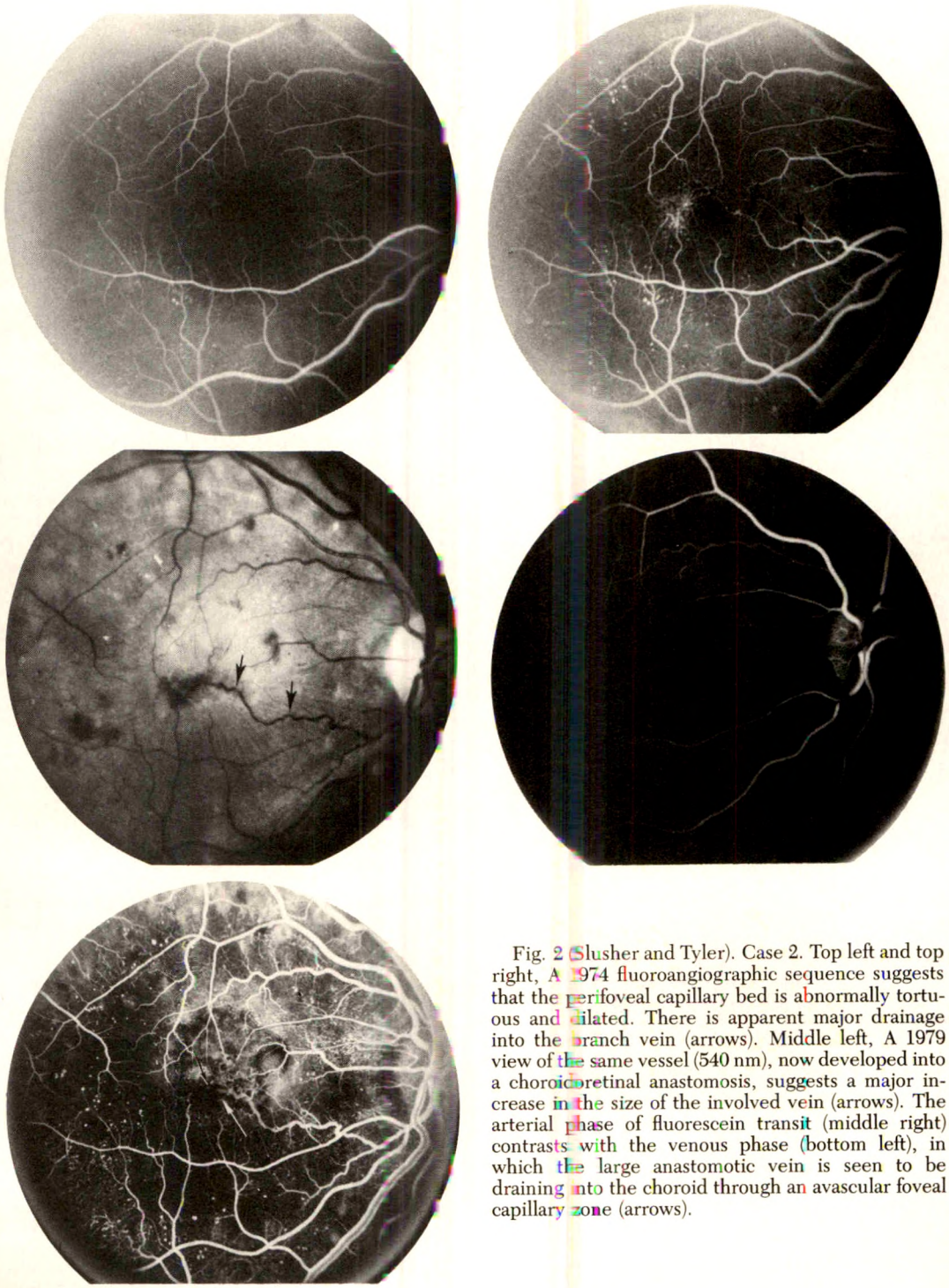


Fig. 2 (Slusher and Tyler). Case 2. Top left and top right, A 1974 fluoroangiographic sequence suggests that the perifoveal capillary bed is abnormally tortuous and dilated. There is apparent major drainage into the branch vein (arrows). Middle left, A 1979 view of the same vessel (540 nm), now developed into a choroidoretinal anastomosis, suggests a major increase in the size of the involved vein (arrows). The arterial phase of fluorescein transit (middle right) contrasts with the venous phase (bottom left), in which the large anastomotic vein is seen to be draining into the choroid through an avascular foveal capillary zone (arrows).

Discussion

In most reports on the formation of acquired venous choroidoretinal anastomoses, the common pathophysiologic factor is a break in, or damage to, Bruch's membrane.[1-6] That is probably true in our Cases 1 and 2, although we have not seen presumed ocular histoplasmosis or chronic hypertensive diabetic maculopathy cited previously as etiologic conditions.

The prospective follow-up of Cases 1 and 2 made it possible for us to observe an important characteristic of these aberrant vascular connections: they may progress in size over a period of time, allowing an increased volume of blood to be shunted into choroidal vascular channels. In Case 2, the size of the anastomotic vein increased markedly over a four-year period (Fig. 2), and late phases of fluorescein transit indicate a substantial volume of retinal venous blood draining into the choroid. In Case 1, the same progression of hemodynamic features was evident, but over a shorter period (ten months). Additionally, progressive atrophy occurred in the proximal segment of vein interposed between the histoplasmosis spot and the inferior major venous arcade (Fig. 1). This particular anastomotic development suggests a type of choroidal venous steal, manifested on fundus photography and rapid-sequence fluorescein angiography as increasing amounts of retinal venous blood being channeled into the choroid. In Kennedy and Wise's[3] case, associated with toxoplasmic chorioretinitis, the clinical picture was similar, but the hemodynamics were not demonstrated angiographically.

The experimental and clinical observations of many authors[3-6] have led them to theorize that the pattern of venous flow from retina to choroid is consistent with the gradient of pressure (or resistance to flow) that exists in the involved vessels between the retinal and choroidal systems. In order to accept a gradient of vascular resistance, such as that proposed by Owens, Goldberg and Busse,[1] in which "choroidal artery > retinal artery > retinal vein > choroidal vein," one must assume that the anomalous arterial anastomosis in Case 3 between the superior temporal artery and the retrolaminar or choroidal system represents a type of arteriovenous shunt.

In a review of 2,000 eyes in 1,000 subjects, Justice and Lehmann[7] established the incidence of cilioretinal arteries as 49.5%. They also pointed out that, on fluorescein injection, a cilioretinal artery classically fills simultaneously with the choroidal circulation, and always shows the presence of dye before the proximal branches of the central retinal artery are filled. This would certainly eliminate our arterial shunt as a cilioretinal vessel. Moreover, if this patient's anastomosis is simply a loop between two branches of the central retinal artery, those branches would be expected to fill simultaneously, which they clearly do not do (Fig. 3). Studying the incidence of "non-aneurysmal congenital anomalies of the retinal arteries" in 2,100 consecutive healthy patients, Awan[8] established cilioretinal arteries as the most commonly occurring vascular anomalies of the retina.

However, none of the arterial anastomoses reported by those authors were similar to that in our Case 3. Indeed, we are unable to find a comparable arterial anastomosis cited in published ophthalmic reports.

If one accepts that the arterial branch exiting through the disk communicates with some portion of the choroidal circulation, the angiographic findings suggest that this represents flow from the central retinal arterial branches to the choroidal venous system. Although this is consistent with current theories of vascular resistance in the retinal and choroidal

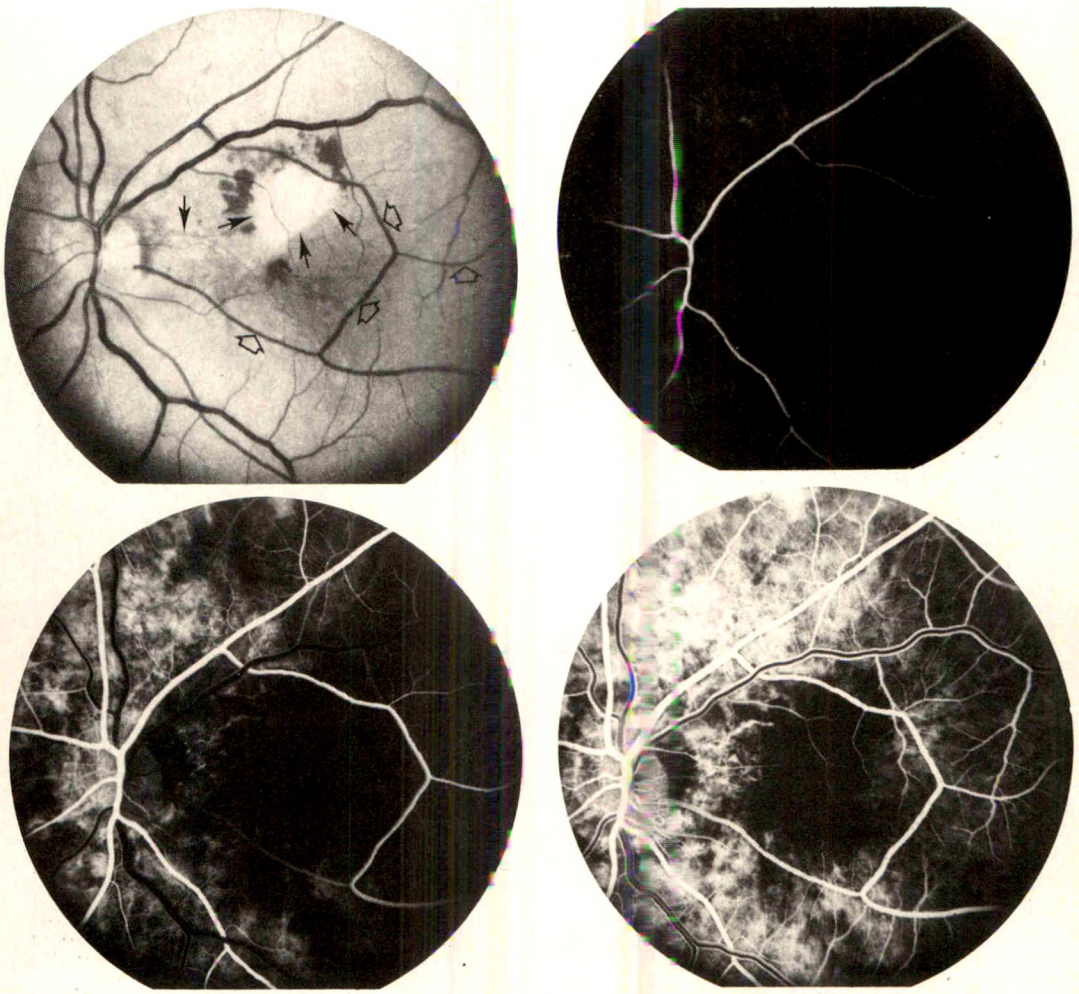


Fig. 3 (Slusher and Tyler). Case 3. Top left, Unusual arterial choroidoretinal anastomosis (540 nm) in which the anomalous arteriole, after circumscribing the fovea, bifurcates into a distal branch and a branch exiting through the disk (open arrows) and presumably entering the choroidal circulation. Solid arrows, angioid streaks and a photocoagulated subretinal neovascular net. The arterial phases of fluorescein transit (top right, bottom left, and bottom right) clearly indicate flow from the proximal branch of the superior temporal retinal artery into the retrolaminar space.

systems, it cannot be proved conclusively. Indeed, the prospective photographic and angiographic studies of all three of the cases presented here suggest that many basic hemodynamic features of the retinal and choroidal blood supplies are incompletely understood.

Summary

Three cases of choroidoretinal anastomosis had unusual etiologic and hemodynamic features. In two cases the anastomoses, one caused by ocular histoplasmosis and one by diabetic maculopathy, shunted retinal venous drainage into the choroid. In the third case, the congenital arterial choroidoretinal anastomosis should probably be termed retinochoroidal arteriovenous anastomosis. Prospective photographic and fluorangiographic studies of these patients suggest that our understanding of many basic

features of retinal-choroidal circulatory hemodynamics is incomplete.

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Ophthalmic miniature

The eyes of other people are the eyes that ruin us. If all but myself were blind, I should want neither fine clothes, fine houses, nor furniture.

Benjamin Franklin

LATE CYSTOID MACULAR EDEMA IN PSEUDOPHAKIA

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Aphakic cystoid macular edema is a syndrome characterized clinically by decreased central visual acuity and pathologically by the accumulation of serous fluid in the outer plexiform layer of Henle.[1] The cause is unknown but inflammation, vitreous traction, and generalized vascular incompetence have been proposed as pathogenic factors. Yanuzzi and associates have implicated prostoglandins as the direct mediator of the noxious stimulus.[2] Aphakic cystoid macular edema may occur at any time; however, the onset is most frequently four to 16 weeks after cataract extraction.[3] The term late onset is proposed to describe cystoid macular edema that first appears one year or more after cataract extraction.

CASE REPORTS

Case 1—A 70-year-old man complained of blurred vision in his right eye in January 1976. Visual acuity was 6/60 (20/200) in the right eye and 6/7.5 (20/25-1) in the left eye. There was a dense posterior subcapsular cataract in the right eye and an incipient cataract in the left. The only other abnormality was atrophy of the pigment epithelium in both maculas consistent with minimal "dry" senile macular degeneration.

In February 1976, I performed an uneventful intracapsular cataract extraction on the right eye and implanted a 19.5-diopter medallion intraocular lens. The patient's postoperative course was uncomplicated. A refraction performed two months postoperatively was R.E.:-0.50 + 1.00 × 150 with a visual acuity of 6/12 (20/40+1). The refraction and visual acuity were unchanged 12, 24, and 36 months later. Slit-lamp examination of the right macula at six months disclosed diffuse atrophy of the pigment epithelium. The sensory retina was normal. On Aug. 22, 1979, the patient complained of blurring of his right eye. Corrected visual acuity was 6/24 (20/80) in the right eye and 6/9 (20/30-1) in the left eye. Intraocular pressure was 18 mm Hg in both eyes. External examination of the right eye revealed no perilimbal vascular injection. The cataract wound had healed well. The cornea was clear and the anterior chamber was perfectly clear without cell or flare. The implant was well positioned in the eye and was free of precipitates. The anterior vitreous was clear. The disk showed a small physiologic cup. The macula again showed atrophy of the pigment epithelium. Additionally, biomicroscopic examination of the retina disclosed multiple cystoid spaces.

I performed a photostress test to determine the magnitude of the physiological defect.[4] Photostress recovery time was 90 seconds in the right eye and 49 seconds in the left eye. The results of a fluorescein angiogram were consistent with the diagnosis of cystoid macular edema.

The patient began taking 25 mg of indomethacin, three times each day. One month later his visual acuity had improved to 6/12 (20/40-) but cystoid macular edema was still present. Three months later the visual acuity was 6/15 (20/50-) and a fluorescein angiogram remained positive for cystoid macular edema.

Case 2—A 74-year-old woman complained of difficulty in reading in March 1978. Visual acuity was 6/24 (20/80) in the right eye and 6/12 (20/40) in the left eye. There were posterior subcapsular cataracts present in both eyes. Results of the ophthalmological examination were otherwise within normal limits

On May 18, 1978, I performed an intracapsular cataract extraction on the right eye, implanting an 18.5-diopter Binkhorst Fyodorov lens. The patient's postoperative course was uneventful. Two months postoperatively the visual acuity in the right eye was 6/7.5 (20/25+2) with a correction of −2.75 +2.50 ×175. Nine months postoperatively the visual acuity was unchanged.

On Sept. 12, 1979, the patient complained of sudden deterioration of the vision in the right eye. Visual acuity was 6/18 (20/60) in the right eye and 6/24 (20/80-) in the left eye. An external examination showed nothing unusual. Slit-lamp examination showed no sign of inflammation. The implant was well situated in the eye but there appeared to be excessive pseudophakodenesis. Biomicroscopic examination of the retina disclosed microcystic changes in the right macula. Photostress recovery time was 74 seconds in the right eye and 29 seconds in the left eye.

I diagnosed aphakic cystoid macular edema and began topically administered corticosteroid every two hours. Nine days later the visual acuity had improved to 6/12 (20/40). Six months later the visual acuity was 6/9 (20/30+2) in the right eye, and the photostress recovery time was 30 seconds in the right eye. A careful examination of the right macula showed complete resolution of the cystic edema.

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Case 3—A 70-year-old man came in for a routine examination in April 1978. I noted that his best corrected visual acuity had deteriorated from 6/12 (20/40) to 6/60 (20/200) in the left eye. The right eye was correctable to 6/15 (20/50). Both eyes had nuclear cataracts. Results of the ophthalmological examination were otherwise within normal limits

On June 20, 1978, I performed an intracapsular cataract extraction on the left eye, inserting a 16.0-diopter Binkhorst Fyodorov lens. The surgery was uneventful. Three months postoperatively the visual acuity in the left eye was 6/6 (20/20) with a correction of -2.00 $+0.50$ $\times 180$. One month later the lens spontaneously dislocated into the anterior chamber, necessitating surgical repositioning. This was done without incident. Five months postoperatively the visual acuity was 6/9 (20/30+) in the left eye. Sixteen months postoperatively it was 6/7.5 (20/25+) with the same correction. Eighteen months postoperatively the visual acuity suddenly decreased to 6/120 (20/400) and the patient was aware of a positive central scotoma.

On examination there was 1+ perilimbal injection and a trace flare and cell in the anterior chamber. The implant was properly positioned and free of precipitates. The anterior vitreous was clear. It was not possible to perform a slit-lamp examination of the retina; however, photostress recovery time was more than 120 seconds in the left eye and 22 seconds in the right eye. A fluorescein angiogram documented cystoid macular edema. I injected 6 mg of betamethasone into subtenon's space. To date the edema has not resolved and the visual acuity varies between 6/15 and 6/22 (20/50 and 20/70).

Discussion

The incidence of early cystoid macular edema has been carefully studied. Klein and Yanuzzi[5] performed fluorescein angiograms on 100 consecutive cataract patients, two to seven days postoperatively. Of these patients 5% demonstrated classic cystoid macular edema. Eighteen patients had a second fluorescein angiogram four to six weeks postoperatively. None of this group had demonstrated cystoid macular edema in the first postoperative week. Three of these 18 patients (16.6%) showed cystoid macular edema on the second angiogram.

Irvine and associates[3] reported that the incidence of cystoid macular edema four to 16 weeks after cataract extraction was 40%. Jacobson and Dellaporta[6] demonstrated that macular edema resolves spontaneously in at least 71% of patients with this condition, although it may require six months or longer to improve. They concluded that "no medical or surgical therapy has been demonstrated to improve the rate of natural spontaneous resolution of cystoid macular edema after cataract extraction."

Winslow, Taylor, and Harris[7] showed that there was a progressive reduction in the incidence of cystoid macular edema. At six weeks the incidence was 44%, at six months 29%, and at 12 months 20%. They concluded that the incidence of cystoid macular edema after lens implantation is comparable to that following nonimplant surgery. The Miami Study Group[8] confirmed this observation in patients who had undergone intracapsular cataract extraction with a lens implantation, but found a significantly lower incidence of cystoid macular edema in patients with extracapsular surgery and lens implantation.

Late-onset cystoid macular edema is well recognized, and there are reports of cases appearing long after cataract extraction. The longest documented onset that I could find occurred 13 years after an intracapsular cataract extraction.[9] Unfortunately, the course of late-onset cystoid macular edema is unknown, and it is not possible to compare the rate of occurrence in conventional cataract surgery to that in eyes with intraocular implants. Because I have now seen three patients with intracapsular cataract extraction and lens implantation develop cystoid macular edema, I wonder whether the incidence is greater in the pseudophakic eye.

Since lens implantation has become an important adjunct to cataract surgery, understanding the incidence and causes of late-onset pseudophakic cystoid macular edema is vital.

Summary

Late-onset cystoid macular edema appears one year or more after cataract

extraction. Three patients developed this problem after intracapsular cataract extractions and implantation of intraocular lenses. In two cases the edema did not resolve despite drug therapy. In the third, the edema resolved completely, possibly because of spontaneous improvement rather than treatment. It is not yet possible to determine whether the incidence of cystoid macular edema is greater in eyes that have been implanted with intraocular rather than conventional lenses.

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PARS PLANA VITRECTOMY INFUSION LINE IN RETINAL DETACHMENT SURGERY

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If retinal detachment is complicated by large amounts of subretinal fluid[1] or by choroidal detachment,[2,3] therapy is difficult. Severe bullous retinal detachment may prevent accurate cryoretinopexy and accurate buckle placement, and the hypotony caused by drainage of large amounts of fluid may contribute to choroidal detachment and thus complicate the postoperative course.

Preoperative choroidal detachment may preclude the successful placement of an adhesive because the reaction cannot reach the pigment epithelium to stimulate an inflammatory response.[2] The presence of a choroidal detachment may complicate associated vitreous surgery.

We have successfully operated on several patients with retinal and choroidal detachments by using a pars plana infusion line; specifically, the infusion cannula designed by Conor O'Malley for the Ocutome system.

CASE REPORTS

Case 1—A 68-year-old man had blurred vision in his right eye for about ten days before he saw an ophthalmologist and learned of his retinal detachment. Visual acuity was R.E.: 6/18 (20/60) and L.E.: 6/60 (20/200). Decreased visual acuity in the left eye was caused by a cataract.

Ophthalmoscopy of the right eye showed a large, posterior horseshoe tear at the 11-o'clock position, and a bullous retinal detachment involved the superotemporal quadrant. The macula was shallowly detached. Bed rest was prescribed to encourage resolution of the fluid but when there was no significant settling after two days, surgery was performed.

After a 360-degree conjunctival incision and isolation of rectus muscles, the eye was again examined by scleral depression and indirect ophthalmoscopy. No other breaks were found. We could not accurately localize the hole, partly because of the high retinal elevation. Therefore, we inserted the infusion line.

We made a circumferential sclerotomy 4 mm from the corneoscleral limbus at the 8:30-o'clock position with the micro-vitreo-retinal blade, a two-edged knife designed especially for the line. We sutured the Ocutome infusion cannula in place with a 6-0 Vicryl mattress suture. By setting the 500-ml infusion bottle of balanced saline solution 11 inches above the eye, we were able to maintain normal intraocular pressure.

In the area of the retinal break, we created a radial sclerotomy, preplaced a 5-0 mersilene suture through the lips, and released subretinal fluid (Fig. 1). After the mattress suture had been temporarily tied, we opened the infusion line to restore intraocular volume and pressure. With the infusion line open, we were able accurately to treat the break with cryotherapy and place mattress sutures for a scleral

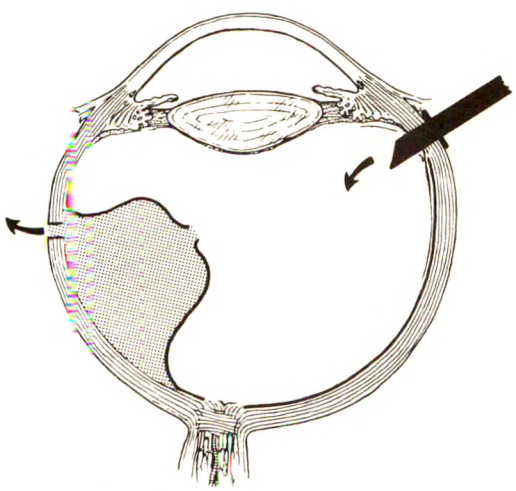


Fig. 1 (Diddie and associates). Subretinal fluid is released from the area of the bullous retinal detachment, while the pars plana infusion line maintains intraocular volume and pressure.

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sponge. The infusion line was removed and the sclerotomy closed with a figure-8 nylon suture.

Case 2—A 10-year-old boy had visual acuity of 1/180 (2/400) in the right eye and external examination showed a port-wine stain involving both upper and lower eyelids on the right side. Results of slit-lamp examination were normal. Ophthalmoscopy showed a normal left eye, but an extensive bullous retinal detachment was present in the right eye. This appeared to be associated with a large choroidal hemangioma superotemporally, an impression that was confirmed by ultrasonography and fluorescein angiography. No retinal breaks were seen.

We treated the hemangioma with xenon photocoagulation after draining the subretinal fluid in an attempt to reattach the retina. After incising the conjunctiva and isolating the rectus muscles, we inserted the infusion cannula 3.5 mm from the corneoscleral limbus in the inferotemporal quadrant and secured it in place with a mattress suture. We then prepared a partial-thickness scleral flap 16 mm posterior to the corneoscleral limbus in the 8-o'clock meridian. The exposed choroid was treated with confluent diathermy. While the eye was being infused at 20 mm Hg with balanced saline solution, the subretinal space was entered and fluid drained without hemorrhage. The infusion cannula maintained normal intraocular pressure. After closure of the scleral flap with 5-0 mersilene suture, xenon photocoagulation to the hemangioma was done. The infusion cannula was removed and the sclerotomy site closed with an 8-0 nylon shoestring suture.

Case 3—A 70-year-old man had undergone cataract extraction and placement of an intraocular lens in his right eye four weeks before he was seen by us. He had visual acuity of R.E.: 6/60 (20/200) and L.E.: 6/9 (20/30). Slit-lamp examination showed a Binkhorst iridocapsular intraocular lens in place. Ophthalmoscopy showed a large nasal and inferior rhegmatogenous retinal detachment with vitreous condensation and periretinal proliferation inferiorly. This was further complicated by a 360-degree choroidal detachment.

Because of the periretinal proliferation, we operated without waiting for the choroidal detachment to resolve. After incising the conjunctiva and isolating the rectus muscles, we placed an extra-long, 4-mm infusion cannula through the pars plana 3.5 mm from the corneoscleral limbus inferotemporally. After determining that the cannula was in the vitreous cavity, we prepared a radially oriented sclerotomy 7 mm posterior to the corneoscleral limbus. Copious amounts of dark red fluid were released from the subchoroidal space (Fig. 2). Intraocular volume was maintained by the infusion cannula. With the choroidal detachment drained, we then performed vitrectomy, membrane peeling, cryoretinopexy, scleral buckling, and gas injection.

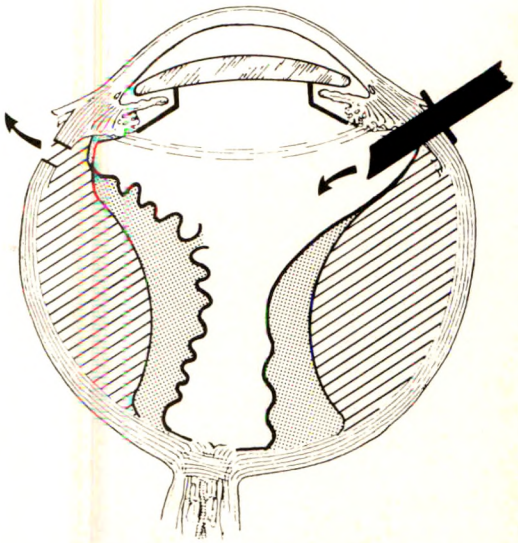


Fig. 2 (Diddie and associates). The pars plana infusion line maintains normal intraocular relationships and helps to expel fluid from the choroidal detachment.

Discussion

The problems with highly bullous detachments are well known to retinal surgeons.[1] Some have advocated drainage of subretinal fluid before localizing, treatment, and buckling. The release of large amounts of subretinal fluid during retinal detachment surgery can result in hypotony and its attendant complications, that is, problems in suturing or incising a soft eye and enhancement of the possible development of choroidal detachment or intraocular hemorrhage.[2,4] Extensive choroidal detachment can contribute to retinal adhesions and closure of the anterior chamber angle with synechiae. Severe intraocular hemorrhage is a more serious complication and is virtually untreatable without vitrectomy techniques. We believe that the maintenance of intraocular volume and pressure during surgical decompression of the eye, such as the drainage of considerable amounts of subretinal fluid, should reduce the incidence of these complications. It should also be helpful in treating the eye with preexisting choroidal separations and preventing their recurrence during the ope-

rative procedure. Our experience with the pars plana infusion cannula indicates that it can be useful in these situations. Our procedure was designed for eyes that have already had vitrectomy,[5] but we have extended and adapted it to non-vitrectomy situations.

This technique emphasizes that surgical methods for treating retinal detachment must remain flexible. Thus, although usually the retinal break is localized and treated and the sclera prepared for buckling before drainage of subretinal fluid, there may be instances, as in our cases, when the drainage may be the initial step.

Treatment of preoperative choroidal detachment is not entirely satisfactory. Corticosteroids have been recommended,[3,6,7] but it is not always possible to wait for medical resolution before scleral buckling. Drainage of choroidal detachments has been recommended before scleral buckling, and our technique should simply make this safer by maintaining normal intraocular volume and pressure. Hemorrhagic choroidal separation can be managed with the infusion cannula as shown in Case 3. If the hemorrhagic choroidal detachment is fresh, red blood will be drained, whereas drainage of older detachments will produce so-called "black oil."

We encountered two problems with this technique. In one patient, despite initial flattening of the retina, the vitreous became incarcerated in the sclerotomy site and the retinal break was held open with this traction. Vitrectomy reattached the retina. Strict attention to wound-closure techniques, avoiding excessive vitreous incarceration, should decrease the incidence of this problem.

The second problem we have encountered has been retinal incarceration during simultaneous drainage of subretinal fluid and intraocular infusion. This can be avoided by careful monitoring of retinal position with the indirect ophthalmoscope during the technique. Also, an infusion bottle height more than that required to maintain normal intraocular pressure (about 11 inches above the eye) should be avoided.

Summary

We used new applications for a pars plana infusion cannula in three patients with retinal and choroidal detachments. Because the cannula maintained intraocular volume and form, it was helpful in treating bullous retinal detachment and choroidal detachment.

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LENS CAPSULAR CYST

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AND GORDAN K. KLINTWORTH, M.D.
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Cysts within the anterior and posterior chamber are well-known complications of epithelial implantations secondary to penetrating injuries or certain intraocular surgical procedures. Epithelization of the anterior chamber may also result in cysts of the iris.[1] Other cysts in the anterior segment of the eye include the nonpigmented iris stromal cysts,[2] the deeply pigmented iris neuroepithelial cysts,[3] iris cysts associated with melanoma,[4] parasitic cysts,[5] and the stromal cysts of the cornea.[6] We describe a patient with a cyst that originated from the crystalline lens. To the best of our knowledge, this is the first case to be documented.

CASE REPORT

A 33-year-old man was seen by his primary ophthalmologist after a piece of nail he was hammering caused an acute penetrating injury to his left eye. Uncorrected visual acuity at that time was R.E.: 6/9 (20/30) and L.E.: 03/6 (1/200). No other abnormalities were noted in the right eye. External examination of the left eye showed a pupil that was central and round. When viewed with the slit lamp, a perforating wound of the peripheral portion of the nasal cornea was evident at the 8-o'clock position. The anterior chamber was deep, with a small hyphema and a metallic foreign body nestled on the anterior surface of the iris. The lens and fundus were not clearly visible because of fibrinoid material within the anterior chamber. A keratotomy at the entry site of the foreign body was performed for magnetic removal of a 2 × 2-mm intraocular foreign body. During the procedure a portion of iris prolapsed and was excised. The corneal wound was closed with several interrupted 8-0 silk sutures. Four months later the patient's best corrected visual acuity was R.E.: 6/9 (20/30) and L.E.: 6/12 (20/40). An irregular pupil with a nasal iridectomy defect, corneal scar, and a small opacity of the peripheral nasal lens cortex was evident in the left eye at this time. Visual acuity remained unchanged until 1975 when it slowly began to diminish without pain. An optometrist told the patient that a cataract was present in the left eye, but that it was not ready to be removed. Two years later a family practitioner on a routine examination also noted a cataract in that eye. In June 1979 when the patient was examined elsewhere, a dense sclerotic lens and a 10 × 0.8-mm clear fluid-filled anterior chamber cyst were evident. This was the first time anyone had noted an intraocular cyst. A month later the patient was referred to the Durham Veterans Administration Hospital where physical examination showed a corrected visual acuity of R.E.: 6/6 (20/20) and L.E.: light perception with inaccurate projection. Intraocular pressures were R.E.: 18 mm Hg and L.E.: 21 mm Hg by applanation tonometry. Slit-lamp examination showed a 10 × 0.8-mm clear cyst extending from the nasal corneoscleral limbus to the temporal pupillary margin and lying adjacent to the corneal endothelium (Fig. 1). Other than a small corneal scar in the peripheral corneoscleral limbus in the 8-o'clock position, the cornea was clear. No interruptions were identified within Descemet's membrane. There was a 5 × 7-mm coloboma of the iris inferonasally. The anterior chamber was of normal dimensions and lacked flare or cellular deposits. The

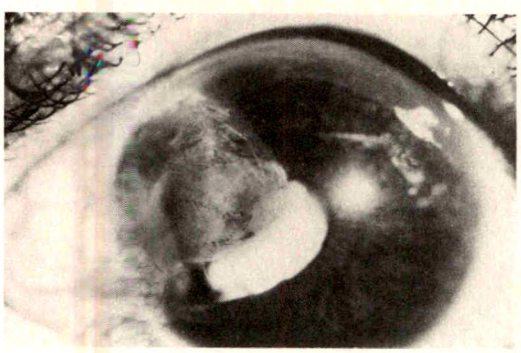


Fig. 1 (Salisbury, Foulks, and Klintworth). Slit-lamp view of the clear anterior chamber cyst extending from the nasal corneoscleral limbus and the cataractous lens changes.

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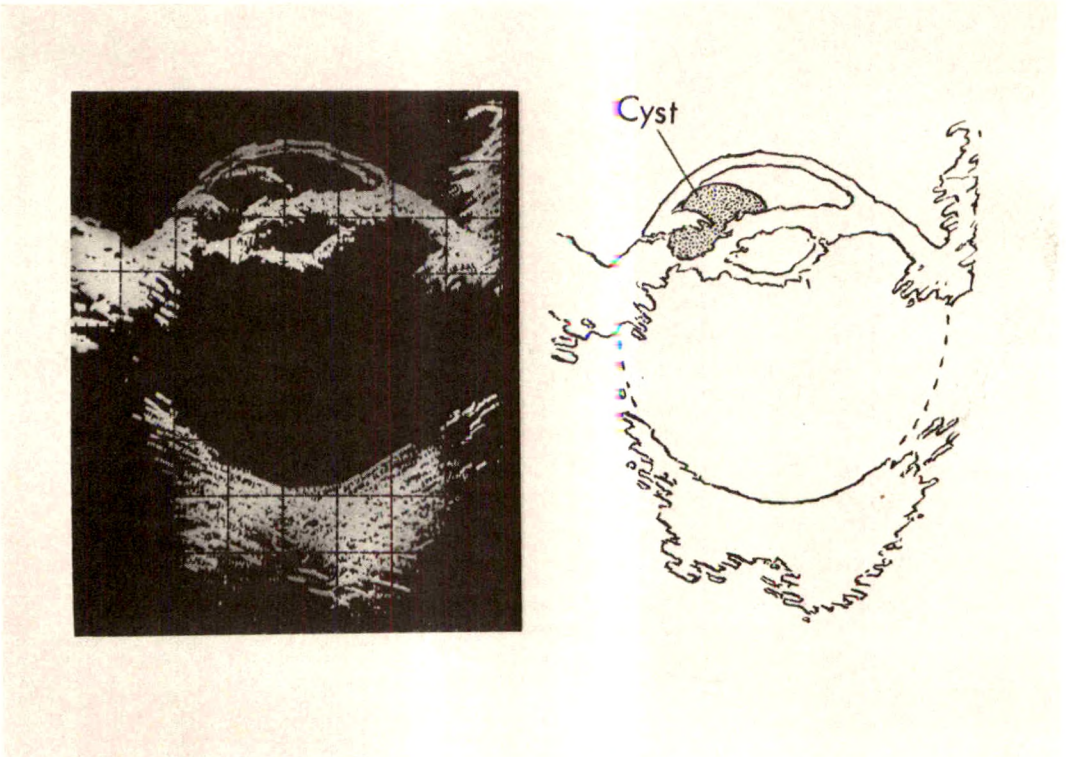


Fig. 2 (Salisbury, Foulks, and Klintworth). Ultrasonography defines the cyst which extends into both the anterior and posterior chambers and which is constricted by the iris, producing a dumbbell shape.

lens was shrunken with dense cataractous changes and wrinkling of the anterior surface. The fundus could not be seen. Ultrasonography outlined the cyst in the anterior chamber and showed an extension into the posterior chamber (Fig. 2). The cyst, which was constricted at the pupillary margin giving it a "dumbbell" shape, extended anteriorly to lie behind the cornea and posteriorly toward the ciliary body.

With the patient under local anesthesia, the anterior segment was entered through a fornix-based conjunctival flap. The cornea was then retracted and the cyst was easily dissected from its attachments to the cornea and ciliary body. No connection between the cyst and lens was noted. While it was being taken from the eye a portion of the cyst wall broke and remained near the ciliary body. After excision of as much of the cyst as possible, the cataractous lens was removed without difficulty, but a small amount of vitreous prolapsed. During an anterior vitrectomy an inferior retinal dialysis was noted and repaired.

Results

After their excision both the cyst and the cataractous lens were fixed in 10% buffered formalin and processed for light microscopy according to standard methods. Paraplast-embedded sections showed the cyst to have a smooth, homogenous, eosinophilic and markedly PAS-positive capsule about 10 nm thick (Fig. 3). Part of its inner surface was lined by a cuboidal epithelium, and abundant fibrous tissue was located adjacent to these cells and the hyaline capsule. Epithelial cells resembling those of cornea or conjunctiva were not observed. Within the cyst wall were scant cells with small rounded eccentric nuclei and foamy cytoplasm and larger cells with the morphologic attributes of the bladder cells of Wedl (Fig. 4). The cataractous lens measured 7 mm in diameter and 2 mm in thickness, and multiple step sections through it failed to show any surrounding capsule or capsular epithelium. A few macrophages were situated between some of the superficial lens fibers at the equator of the lens (Fig. 5). To learn more about the nature of the hyaline capsule of the cyst, a

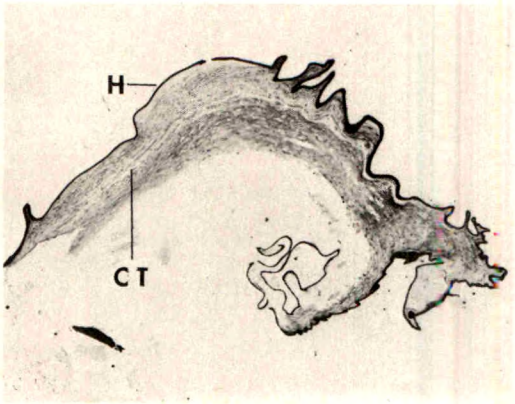


Fig. 3 (Salisbury, Foulks, and Klintworth). The cyst wall is covered by a markedly PAS-positive capsule (H), and its inner surface contains collagenous tissue (CT) (PAS, ×32).

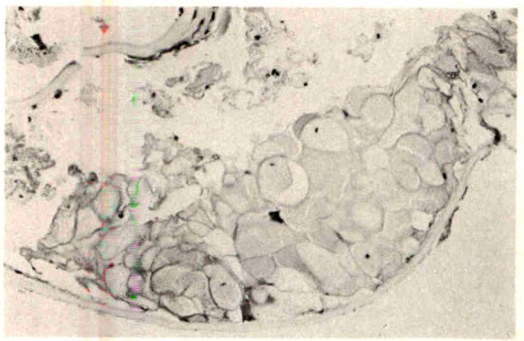


Fig. 4 (Salisbury, Foulks, and Klintworth). Within the cyst wall are cells with small rounded eccentric nuclei and foamy cytoplasm and larger cells with the morphologic attributes of the bladder cells of Wedl (Masson trichrome, ×250).

representative portion of the Paraplast-embedded cyst wall was soaked in xylol to remove the embedding material and then rehydrated through graded concentrations of ethanol to water. The tissue was then fixed in 1% osmium tetroxide before dehydration through graded alcohols and placement in propylene oxide and embedding in Epon. Thick sections (500 mμ) were examined with an electron microscope at 60 kV. Transmission electron microscopy showed the outer hyaline portion of the cyst capsule to be composed of numerous closely packed lamellae of delicate fibrillogranular material with epithelial cells located adjacent to the interior surface (Fig. 6). The membrane lacked the ultrastructural features of Descemet's membrane such as the characteristic 100-nm banded fetal layer. The inadequate preservation of the tissue for electron microscopy precluded a detailed study of cellular morphology.

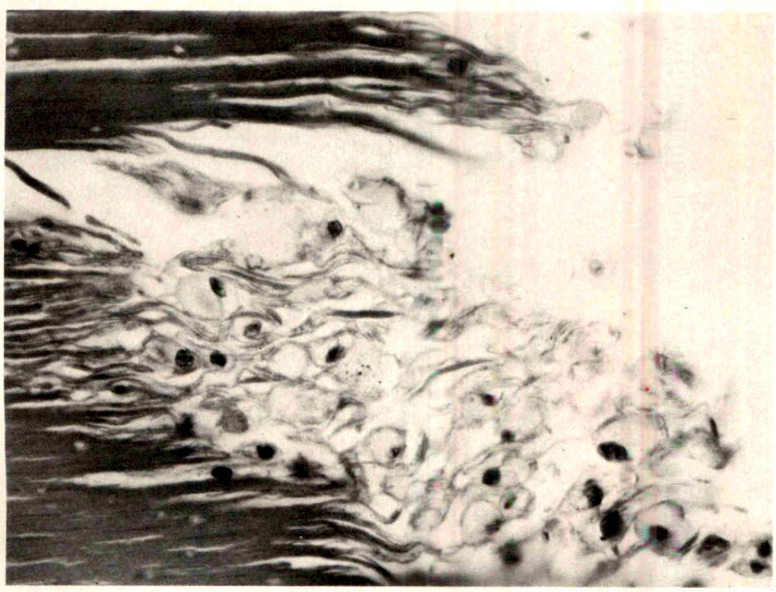


Fig. 5 (Salisbury, Foulks, and Klintworth). Macrophages situated between some of the superficial lens fibers at the equator of the cataractous lens (Masson trichrome, ×400).



Fig. 6 (Salisbury, Foulks, and Klintworth). Electron micrograph of the hyaline capsule of the cyst wall showing numerous closely packed lamellae of delicate fibrillogranular material consistent with normal lens capsule (×14,400).

Discussion

Before surgical excision, ultrasonography showed the cyst to be adjacent to a sclerotic lens and to extend forward into the anterior chamber and posteriorly toward the ciliary body. Although this intraocular cyst that developed after a penetrating ocular injury was suspected preoperatively of being an epithelial implantation cyst, histologic examination proved this not to be the case. Surprisingly, the cyst was lined by a hyaline capsule, epithelial cells, bladder cells, and collagenous tissue. The lens rudiments within the cyst wall resembled those found in Elschnig pearls, lentoids, or Soemmering's ring cataract, which commonly develop after extracapsular cataract extraction.[7]

Although Descemet's membrane and

the lens capsule may closely resemble each other by light microscopy, it seemed unlikely that Descemet's membrane formed the cyst wall because this structure was intact clinically and corneal edema was absent. Light microscopy supported the impression that the hyaline membrane was lens capsule, because the associated cells had morphologic features of reactive lens epithelium and not of corneal endothelial cells. That the lens capsule did indeed form the cyst wall was confirmed by transmission electron microscopy. Despite its processing under suboptimal conditions, ultrastructural characteristics of lens capsule were evident, whereas morphologic features of Descemet's membrane, including the anterior banded fetal layer, were conspicuously absent.

The cyst was clearly derived from the lens that had been injured several years earlier by a penetrating wound, but the reason for its late appearance remains perplexing. The thickness of the hyaline capsule of the cyst (10.3 μ) was comparable to what one would expect of a lens capsule from a person this age. This suggests that the cyst wall consisted of a dislodged lens capsule and was not produced by displaced lens epithelial cells with newly formed capsular material. Presumably, the lens capsule was stripped from the lens with associated epithelial cells. The latter had not only differentiated into lens fibers and into the bladder cells of Wedl, but had produced collagen as is seen in anterior polar cataracts.[8]

The small opacity of the peripheral nasal lens cortex, which was evident four months after the penetrating injury, followed the wound to the lens capsule and formed the nidus for the subsequent more extensive cataract. The presence of macrophages between some of the lens fibers of the cataractous lens is evidence of a past break in the lens capsule because this structure forms a natural barrier to cellular infiltrates. Despite this morphologic evidence of a ruptured lens capsule, the patient failed to manifest clinical evidence of iritis, phacolytic glaucoma, or phacoanaphylactic endopthalmitis. The usual progression of localized lens opacities resulting from small penetrating lenticular injuries, such as that received by our patient, is to remain nonprogressive. Others, as in the present case, may evolve into an intumescent cataract. Perhaps such a swollen cataractous lens caused a previously weakened and partially disrupted lens capsule to separate from the underlying cortex with apparent cyst formation.

Summary

A 33-year-old man had a unique lens capsular cyst that became clinically evident eight years after a penetrating ocular injury in which the lens capsule was disrupted. Microscopic examination indicated that the cyst may have arisen from displaced lens capsule and epithelial cells that became separated from the lens.

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REFRACTIVE POWER OF PREMATURE CHILDREN AT INFANCY AND EARLY CHILDHOOD

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Several studies in the past have pointed out that premature babies tend to have refractive errors.[1-3] The mode and power of that error are still unknown. The purpose of this study was to find in children born prematurely, during their first years of life, if there is any correlation between birthweight and refractive power, the distribution of refractive power, and the trend of change of the refractive power.

SUBJECTS AND METHODS

All 380 children born in Jerusalem, Israel, between the years 1974 and 1976 with a birthweight of 2,000 g or less were scheduled for examination in July 1977. Of these, only 236 were examined. Five mothers refused to bring their children because they were completely healthy. The mother of a boy who was blind because of retrolental fibroplasia objected to any further examination of her child. The rest of the children who did not come for examination belong to families who changed their address and it was impossible to locate them.

Each child underwent the following examinations: Hirschberg light reflex test, cover test, cover-uncover test, ocular movements in eight directions, and pupil reflex to light (direct and consensual). Then each child received cyclopentolate HCL (Cyclogyl) 0.5 to 1.0% in two doses, ten minutes apart. The children under 1 year of age received the lower concentration. Twenty minutes after the second drop was instilled, skiascopy with streak retinoscopy at a working distance of two-thirds of a meter with the lens 12 mm in front of the cornea, anterior segment biomicroscopy with Goldmann slit lamp, and ophthalmoscopic examination with indirect ophthalmoscope were performed in that order. Of the 236 children examined, four had retrolental fibroplasia, five had cataract, two had esotropia, and 27 had pigmentary changes of the fundus. These 38 children with ocular disease were excluded from this study. The remaining 198 children were divided into six groups according to age.

The refraction was calculated by adding to the sphere result one half of the cylinder correction of the eye. All examinations were carried out by the same physician (A.S.). Statistical analysis was done by using the one-way analysis of variance test and the Student *t*-test when appropriate.

RESULTS

The correlation coefficeints between birthweight and refraction or astigmatism when tested in all the children or separately in each of the six age groups were always below 0.28.

The mean, standard deviation, median and range of birthweight, refraction, sphere, and cylinder of all the children and of each of the six age groups are given in Table 1. No statistically significant

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CHILDREN BORN WITH WEIGHT OF 2,000 g OR LESS—MEAN, STANDARD DEVIATION, MEDIAN, AND RANGE OF BIRTHWEIGHT, REFRACTION, SPHERE, AND CYLINDER

	All Children (6 mos-3½ yrs)	6 mos-1 yr	1-1½ yr	1½-2 yr	2-2½ yrs	2½-3 yrs	3-3½ yrs
No. of Eyes	396	22	48	58	54	78	136
Birthweight (g)							
Mean	1,649.5	1,707.3	1,669.6	1,690.0	1,675.9	1,614.8	1,624.9
S.D.	240.8	225.8	256.6	268.6	216.7	249.3	231.8
Median	1,696.7	1,800.0	1,700.0	1,797.5	1,680.0	1,645.0	1,695.0
Minimum	850	1,300	850	1,020	1,150	900	1,040
Maximum	2,000	1,960	1,970	1,980	1,980	2,000	2,000
Refraction (diopters)							
Mean	+1.222	+1.491	+1.268	+1.500	+1.118	+1.030	+1.195
S.D.	1.140	0.839	1.426	0.973	0.611	1.403	1.134
Median	+1.258	+1.601	+1.481	+1.450	+1.031	+1.125	+1.474
Minimum	−3.75	−0.25	−3.50	−2.25	−0.25	−3.75	−3.125
Maximum	−5.00	+3.00	+5.00	+4.25	+2.50	+5.00	+4.750
Sphere (diopters)							
Mean	+1.439	+1.685	+1.458	+1.694	+1.343	+1.269	+1.419
S.D.	1.128	0.816	1.371	1.051	0.707	1.391	1.088
Median	+1.496	+1.695	+1.518	+1.500	+1.344	+1.292	1.514
Minimum	−3.75	0.00	−3.00	0.00	0.00	−3.50	−2.75
Maximum	+5.50	+3.00	+5.00	+5.00	+3.00	+5.00	+5.50
Cylinder (diopters)							
Mean	−0.434	−0.386	−0.380	−0.388	−0.499	−0.478	−0.449
S.D.	0.434	0.517	0.375	0.494	0.374	0.464	0.429
Median	−0.442	−0.250	−0.453	−0.200	−0.477	−0.443	−0.451
Minimum	−2.50	−1.75	−1.50	−1.50	−1.50	−2.50	−2.00
Maximum	0.00	0.00	0.00	0.00	0.00	0.00	0.00

change was found between the groups in any of the above-listed factors. In all the groups the mean and median of the refractive error were above +1.0 diopter.

The mean, standard deviation, median, and range of birthweight, refraction, sphere, and cylinder of the children with birthweights of 1,750 to 2,000 g, divided into six age groups, are given in Table 2. No statistically significant changes were found between the groups in any of the factors. In all the groups the mean and median of the refraction were above +1.0 diopter.

The correlation coefficients when calculated between the two eyes of each child for sphere correction, cylinder correction, and the axis of the cylinder were 0.930, 0.833, and 0.694, respectively, with a significance of .001.

The distribution in percentage of the refraction, sphere, and cylinder correction is given in Table 3. A similar distribution exists in all age groups.

DISCUSSION

In this study a low correlation coefficient (less than 0.28) was found between the birthweight and refractive power, in normal healthy children with birthweight of 2,000 g or less. The mean and median refractive powers in all ages were between +1.0 and +1.610 diopter (Tables 1 and 2).

In previous studies different and contradictory results were found. Fletcher

CHILDREN BORN WITH WEIGHT OF 1,750 TO 2,000 g—MEAN, STANDARD DEVIATION, MEDIAN, AND RANGE OF REFRACTION, SPHERE, AND CYLINDER

	All Children (6 mos-3½ yrs)	6 mos-yr	1-1½ yrs	1½-2 yrs	2-2½ yrs	2½-3 yrs	3-3½ yrs
No. of Eyes	160	12	20	32	22	26	48
Refraction (diopters)							
Mean	+1.328	+1.563	+1.400	+1.609	+1.114	+1.000	+1.328
S.D.	0.951	0.360	1.086	1.088	0.469	1.306	0.841
Median	+1.375	+1.563	+1.475	+1.438	+1.031	+1.125	+1.500
Miminum	−1.50	+1.125	−0.875	+0.25	+0.25	−1.50	−1.50
Maximum	+4.25	+2.125	+2.75	+4.25	+1.75	+3.75	+2.75
Sphere (diopters)							
Mean	+1.533	+1.708	+1.650	+1.820	+1.352	+1.192	+1.516
S.D.	0.974	0.292	1.101	1.228	0.464	1.288	0.820
Median	+1.510	+1.625	+1.562	+1.500	+1.375	+1.250	+1.563
Minimum	−1.50	+1.50	−0.50	+0.50	+0.50	−1.50	−1.00
Maximum	+5.00	+2.25	+3.00	+5.00	+2.00	+4.00	+3.00
Cylinder (diopters)							
Mean	−0.409	−0.292	−0.500	−0.422	−0.477	−0.385	−0.375
S.D.	0.360	0.292	0.425	0.445	0.344	0.348	0.319
Median	−0.454	−0.250	−0.475	−0.313	−0.479	−0.400	−0.450
Minimum	−1.50	−0.75	−1.50	−1.50	−1.25	−1.00	−1.00
Maximum	0.00	0.00	0.00	0.00	0.00	0.00	0.00

TABLE 3

DISTRIBUTION OF REFRACTION, SPHERE, AND CYLINDER CORRECTION, IN PERCENTAGE, OF THE CHILDREN DIVIDED INTO THE DIFFERENT AGE GROUPS

	REFRACTION				SPHERE				CYLINDER			
	Minus	0.00 to +1.00	+1.25 to +3.00	Above +3.00	Minus	0.00 to +1.00	+1.25 to +3.00	Above +3.00	Less Than −1.00	−0.625 to −1.00	−0.125 to −0.500	Without a Cylinder
All children (6 mos-3½ yrs)	8	32	57	3	5	29	62	4	6	18	45	31
6 mos-1 yr	9	9	82	0	0	18	82	0	9	9	46	36
1-1½ yrs	8	25	63	4	4	29	63	4	4	17	42	37
1½-2 yrs	3	35	59	3	0	34	56	10	10	14	31	45
2-2½ yrs	4	52	44	0	0	44	56	0	4	18	48	30
2½-3 yrs	13	36	46	5	10	31	54	5	5	18	56	21
3-3½ yrs	9	28	62	1	4	25	68	3	4	23	45	28

and Brandon[1] found that infants of birthweights more than 1,700 g had a mild degree of myopia ranging between zero and −6.0 diopters, which decreased to a range of +1.0 to −3.0 diopters at 6 months of age. The infants with birthweights of 1,250 g or less showed a myopia from −10.0 to −20.0 diopters, with a residual myopia of −2.0 to −6.0 diopters at 6 months of age. Graham and Gray,[2] in their study that included full-term and premature infants during the first week of life, proved a definite tendency toward myopia in premature infants. Wagner[3] showed in children aged 3 to 5 years who had been born prematurely that the more the refraction differed from emmetropia, the lower was the average birthweight. Wagner[3] concluded that hypermetropia should be expected in children of low birthweight who suffered from anemia or received oxygen therapy. Scharf, Zonis, and Zeltzer[4] did not find a higher percentage of myopia in infants with birthweights of 1,000 to 2,000 g when compared to infants with birthweights of 2,000 g or more. These premature children showed a change toward emmetropia when examined at 6 months of age. This tendency towards emmetropia continued up to the age of 7 years.[5] Kalina[6] in his study on the refraction of 37 low-birthweight children, aged 2 months to 6 years, found a percentage of myopia similar to that found in our study.

Possibly the reason for these variable findings and the differences with our study result from the different criteria for including children in each research. In the present study only children with no ocular disease were included. Even such a disease as abnormal retinal pigmentation was a reason to exclude a child. The ophthalmologically normal children who were born prematurely had a refraction that was similar to the one found in normal full-term children of similar age.[7] The low correlation coefficients that were found in our study between birthweight and refraction or astigmatism tested in all premature children and in the six different age groups are another indication of the disparity between refraction or astigmatism and birthweight, when the child reaches the age of 6 months.

The number of children with the different diseases found in our study was too small to draw any conclusions about the refraction in these diseases. However, it is likely that in this group, the incidence of refraction errors is higher.

Statistical analysis between the six age groups of premature children, 6 months to 3½ years old (Table 1), using the one-way analysis of variance test, indicated that with the increasing age there is no statistical significant change in refraction or astigmatism, and no trend toward emmetropia. In order to eliminate any possible influence of birthweight on the statistical evaluation, the same statistical test was used on a more homogeneous group of children, those born with birthweights of 1,750 to 2,000 g (Table 2). The same results were obtained with this group: no change of refraction or astigmatism between 6 months and 3½ years of age. Table 3 shows the same distribution of refraction, sphere, and cylinder correction in the six age groups, which is another indicator that there is no change in these factors between 6 months and 3½ years of age.

Fledelius,[8] in his research on children 8 to 11 years old, found almost the same results we found in our study. He stated that the refraction pattern was fairly uniform in premature and full-term born children. Our study indicates that this uniformity starts at the age of 6 months.

Summary

We evaluated the refraction of 198 children born prematurely with birthweights of 2,000 g or less, without any

ocular disease. Between the ages of 6 months to 3½ years these children had a mild hypermetropia. The refractive error did not change between the ages of 6 months and 3½ years. The refraction of the prematurely born children was similar to that found in children born at full term.

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Ophthalmic miniature

I had decided long before that the best way to get the attention of a scientific meeting was to assume that everyone knew nothing outside his subject; I made a point of making it as explicit and easy as I could – rather, in fact, as though instead of addressing scientists I was confronted by their wives. This was the way of all the best scientific expositors, I found: I copied it, and it paid.

C.P. Snow, *The Search*
Penguin, 1958

CYCLOPLEGIC REFRACTIONS IN INFANTS AND YOUNG CHILDREN

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Deprivation of a good retinal image can adversely affect the development of vision.[1] Cataracts of early childhood degrade the retinal image and can leave the eye amblyopic even after surgery clears the media. In some situations refractive errors are associated with refractive amblyopia[2] and astigmatic errors play a particular role in the development of meridional amblyopia.[3] Recent reports stating that more than 45% of normal infants have astigmatism of 1.0 diopter or more, which is about five times the incidence in adults, raised questions as to what effect, if any, such astigmatic errors had on the developing visual system.[4,5] Both of the studies[4,5] which reported a high incidence of infantile astigmatism used noncycloplegic techniques for refracting. We reviewed the results of other studies to determine if the observed astigmatism was related to the noncycloplegic state or an artifact.

Santonastaso[6] reported a 60% incidence of astigmatism in 80 children (with normal eyes and no systemic disease) refracted without cycloplegia and a 50% incidence when the same infants were refracted 24 hours after administration of one drop of atropine 1%. Forty-four infants were refracted within the first ten postnatal weeks and the remainder over the course of the first year. Not only did the author note that cycloplegia in some babies was incomplete with the amount of atropine used, but the number of infants refracted toward the end of the first year was very small (six infants aged 30 to 40 weeks, one aged 40 to 50 weeks, and two aged 50 to 60 weeks). Therefore, these data are not comparable with those obtained in the recent noncycloplegic studies.

Ingram and associates[7-11] refracted large numbers of 1-year-old children with atropine or cyclopentolate cycloplegia, or both. They also found a higher occurrence of astigmatism (13.2% to 17.5% with ≥ 1.5 diopter cylinder) in 1-year-old children than in 3½-year-old children (7.4% with ≥ 1.5 diopter cylinder) and than others found in adults. However, no data are presented for refractive errors during the first postnatal year.

The data available in these and a large number of other reports, summarized by Banks,[12] are not sufficient to indicate the incidence and characteristics of astigmatism in the early postnatal years. To determine the incidence of astigmatism, measured under cycloplegia, throughout the first postnatal year and to look for a decline in postnatal years two and three, we have reviewed the retinoscopic results of cyclopentolate-induced cycloplegia for a large number of youngsters.

Secondly, this study was undertaken to determine if results of refractions differ between normal and esotropic or amblyopic eyes. Infantile astigmatism may not

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be important to visual performance in the first three postnatal years because the earliest meridional amblyopia that has been detected is just before the end of the third postnatal year.[4] However, it is not known if cylindrical or spherical errors or both differ between normal and esotropic or amblyopic eyes of infants and children up to age 3 years (the age when most children can have vision tested with the Allen picture cards or similar test materials). Significant differences could then be used to screen[7,8] youngsters for esotropia or amblyopia, the most frequent ophthalmic disorders for our healthy patients in this age range.

Subjects and methods

The data were taken from 5,042 consecutive charts of patients examined by three staff ophthalmologists at the Children's Hospital Medical Center between 1968 and 1978. One of us (R.A.P.) performed 95% of the retinoscopies reported; two other examiners performed the rest. In a previous study of refractive errors in 143 infants and children there was no difference in the results between refractionists.[13] We included only patients who had been full-term infants and who had no systemic abnormalities; their ages ranged from birth to 160 weeks when first refracted. This report excludes patients with any ophthalmic disorders other than esotropia or amblyopia or both. The diagnoses of patients included the following: normal eyes, esotropia, and amblyopia. We made the diagnosis of normal eyes only if a full ophthalmic examination showed no abnormalities. Patients with normal eyes had been brought to the ophthalmologists because of a family history of an ocular problem or a suspected ocular problem (for example, strabismus), but were found to have no abnormality. Children with both congenital and acquired esotropia were included under the diagnosis of esotropia. The Hirschberg and Krimsky methods were used to make the diagnosis of esotropia in most patients; for some infants and cooperative young children cover-uncover and alternate-cover tests were used. For infants and preverbal children, we based the diagnosis of amblyopia on a consistent preference of one eye for fixation. Unequal visual acuity (assessed by Allen picture cards), in addition to fixation preference, supported the diagnosis in many of the 2½- to 3-year-old patients. We included both strabismic and refractive amblyopia. The esotropic and amblyopic groups were not mutually exclusive; 50 of the 61 children with amblyopia also had esotropia.

In all cases, the cycloplegic agent was cyclopentolate 1%. When administered as recommended,[11,13] cyclopentolate uncovers about 0.40 diopter less hyperopia[11,13] (mean spherical equivalent) and from 0.10 to 0.19 diopter less astigmatism (mean cylindrical error)[11] than atropine refractions of infants and young children. All retinoscopies were performed in a dimly lighted room using a Copeland streak retinoscope; the retinoscopic reflexes were evaluated when the youngster was fixating the light of the retinoscope. The refractionists used minus cylinder convention. The retinoscopic results reported here were those recorded in the chart on the patient's first visit. Usually it was the first refraction ever done on the youngster, and, thus, the examiner was not influenced by previous retinoscopic findings. For the purposes of this report, we defined astigmatism as 1.0 diopter or more difference in refractive power of orthogonal meridia.

Computer-aided analyses were performed on data coded for age at time of retinoscopy, ophthalmic diagnosis, power of spherical and cylindrical components, and axis of cylindrical components for each eye. We calculated spherical equivalents for each eye, and determined

right-left eye differences of spherical equivalent for each patient. As Mohindra and associates[4] did in a study of infant refractions, we classified the axis of any cylindrical component as with-the-rule (direct) if the minus cylinder axis was at 180 degrees ± 15, against-the-rule (indirect) for minus cylinder at 90 degrees ± 15 or oblique (other than with- or against-the-rule) for the normal patients and those with esotropia and amblyopia.

The results of refractions are given for right eyes only. The results for left eyes were similar. The mean spherical equivalent and the mean cylindrical power of the right eye and left eye within each diagnostic group differed by less than 0.34 diopter.

Results

Spherical equivalents—The distributions of the power of the mean spherical equivalent for the first three postnatal years for each of the diagnostic groups were similar in each of the three postnatal years (Fig. 1). In the range of severe hyperopia, that is, +2.76 diopters or more, we found 67 (33%) children with esotropia and 19 (31.5%) children with amblyopia, but only 32 (8.6%) normal children. Thus, for spherical equivalents of +2.76 diopters or more, patients in this population were much more likely to have esotropia or amblyopia than to have normal eyes. However, if we had relied only on hyperopic errors of +2.76 diopters spherical equivalent or more to identify esotropia or amblyopia, we would not have detected 136 (67%) children with estotropia and 42 (68.5%) with amblyopia. For spherical equivalents of less than +2.76 diopters, the distributions of normal children and children with esotropia and amblyopia overlapped broadly; in this range of spherical equivalents the distributions of the three diagnostic groups were indistinquishable.

The mean spherical equivalent for 376 normal children was 1.27 diopters (S.D. = 1.81 diopters), for 203 children with esotropia it was +2.0 diopters (S.D. = 1.24 diopters), and for 61 children with amblyopia it was +1.74 diopters (S.D. = 2.6 diopters). With less than 1.0 diopter difference in the means, and standard deviations of these magnitudes, even if measurements could be made with great precision in this age group, retinoscopy could not distinguish be-

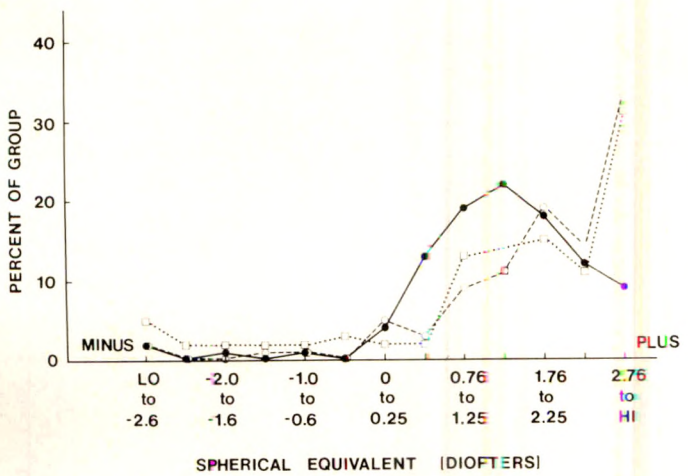


Fig. 1 (Fulton and associates). Distribution of spherical equivalents for normal children (closed circles) and those with esotropia (open circles) and amblyopia (open squares). For each diagnostic group the distribution of spherical equivalents for each of the first three postnatal years is similar to that plotted for the entire age range from birth to 3 years.

tween these three diagnostic groups. The variability among the present examiners when refracting the same youngster was usually less than 0.5 diopter spherical equivalent.

No difference of spherical equivalent between left and right eyes was present in 333 (88.5%) of the normal children, 154 (75.8%) of the children with esotropia, and 38 (62.5%) of the children with amblyopia (Fig. 2). However, the distributions of anisometropia for each of the three diagnostic categories were similar. There were not enough individuals with unilateral severe myopia or hyperopia and refractive amblyopia and esotropia to change the shape of the distributions.

Astigmatism—The distribution of astigmatism across the first three postnatal years was not uniform for each of the three diagnostic groups (Figs. 3 and 4). Of the normal children, the 30- to 40-week-old and the 40- to 50-week-old groups, that is, 7- to 12-month-old infants, had the highest incidence of astigmatism, 36 (25%) and 34 (23.5%) respectively.

Our data obtained for the first postnatal year are summarized and compared with other refraction studies in the Table. As shown in the Table, the two studies [4,5] of noncycloplegic refractions of infants up to 1 year of age found a higher proportion of astigmatic infants and an earlier peak incidence of astigmatism than we did. Mohindra and associates[4] found a peak incidence of astigmatism in 11- to 20-week-old infants; about 55% had more than 1.0 diopter of cylinder. In Howland and associates' study,[5] more than 50% of 1- to 3-month-old infants had 1.0 diopter or more of astigmatism.

From age 50 weeks onward, the percentage of normal youngsters with astigmatism decreased (Fig. 3, top). The percentage of these normal youngsters with astigmatism was significantly different from adults ($\chi^2 = 23.3$; d.f. = 7; $P<.01$). Various reports indicate 6% to 10% of young adults between the ages of 20 and 30 years have astigmatism that does not change.[5,14,15] For purposes of analysis we have assumed that 10% of young adults have more than 1.0 diopter of astigmatism regardless of age.

For children with esotropia, the distri-

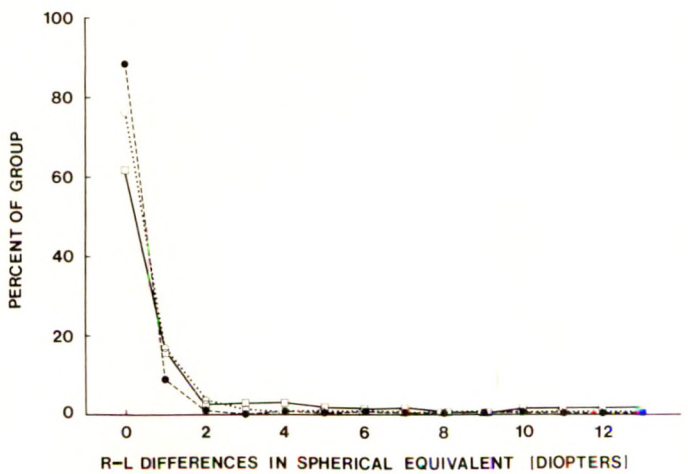


Fig. 2. (Fulton and associates). Differences in spherical equivalents between the right and left eyes of normal children (closed circles) and those with esotropia (open circles) and amblyopia (open squares). There is no difference between right- and left-eye spherical equivalents for 333 (88.5%) normal children, 1554 (76%) children with esotropia, and 38 (62.5%) children with amblyopia.

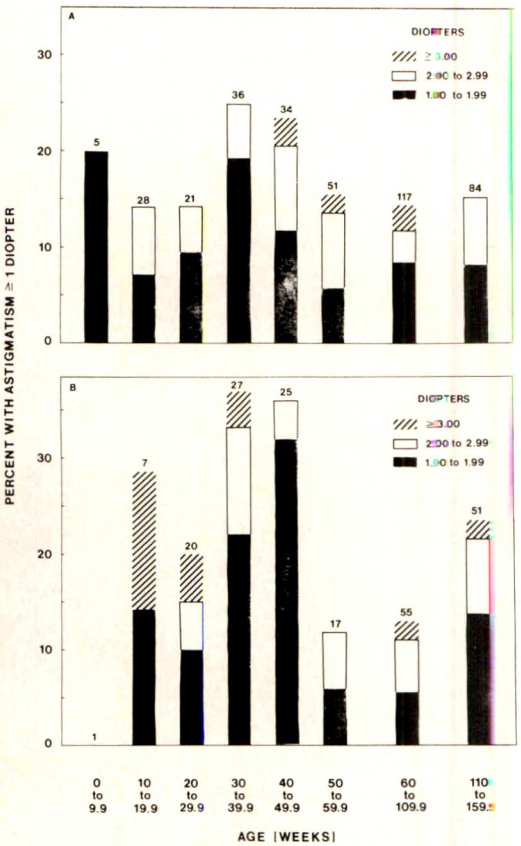


Fig. 3 (Fulton and associates). Distribution of astigmatism among normal children (top) and children with esotropia (bottom). Numbers at the tops of bars indicate the number of individuals in each age group. There is no significant difference between the two groups (χ^2 test).

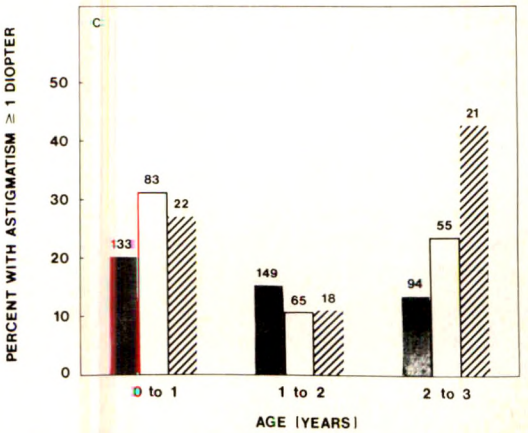


Fig. 4 (Fulton and associates). Percentage of normal children (closed bars) and children with esotropia (open bars) and amblyopia (shaded bars) with 1.0 diopter cylinder. The distribution of astigmatism for children with amblyopia is significantly different from that for normal children (χ^2) test).

bution of astigmatism was not uniform (Fig. 3, bottom). The greatest percentage of astigmatism occurred toward the end of the first year in 30- to 40-week-old and the 40- to 50-week-old (7 to 12 months) groups; 27 (36%) and 25 (34%) individuals in these age groups had astigmatism. The distribution of astigmatism in youngsters with esotropia is also significantly different from adults ($\chi^2 = 50.9$; d.f. = 7; $P < .001$), but not different from normal youngsters ($\chi^2 = 7.8$; d.f. = 7; $P = .5$).

The distribution of astigmatism across the first three years for children with amblyopia is shown in Figure 4. The data for children with amblyopia are presented in one-year age groups because the total number of amblyopic children was small (No. = 61). For comparison the data for normal and esotropic children are also shown in one-year groups. There was a statistical difference ($\chi^2 = 29.3$; d.f. = 2; $P < .001$) between normal children and those with amblyopia. However, by the third postnatal year, when the difference between normal youngsters and those with amblyopia was greatest (Fig. 4), visual screening, which is done routinely in this community, and the onset of accommodative esotropia modify the selection of the patient population. In the third postnatal year, 21 children with amblyopia were examined for the first time. Of these 21, ten also had esotropia; the strabismus had been noticed by the family or pediatrician and led to the child's visit to the ophthalmologist. Each of the remaining 11 had failed visual screening tests and this led to the pa-

tient's first visit. Each of the 40 children with amblyopia examined before the age of 2 years also had esotropia, and ophthalmological consultation had been sought because of the strabismus.

The number of normal youngsters with astigmatism in the first year was 71 (19%). Refractionists who have used noncycloplegic techniques report two to three times this incidence of astigmatism (Table). Of the previous cycloplegic studies, only Santonastaso[6] found such a high incidence of astigmatism (50%); however, this author remarks that cycloplegia was incomplete in many of the babies refracted 24 hours after instillation of a single drop of atropine 1%. The other cycloplegic investigations summarized in the Table indicated that the incidence of astigmatism in 1-year-old children is similar to that found in this report. None of the previous cycloplegic studies (except Santonastoso's) examined the refractive status of younger infants over the course of the first postnatal year.

When the data are combined over the first three years, 63 (16.8%) normal children, 47 (23%) with esotropia, and 17 (28%) with amblyopia were astigmatic. The mean cylindrical power for normal children was 0.37 diopter (S.D. = 0.7, for those with esotropia, 0.46 diopter (S.D. = 0.8 diopter), and for those with amblyopia, 0.52 diopter (S.D. = 0.9 diopter). Differences in mean cylindrical power were not significant between the normal and esotropic groups ($F = 1.80$; d.f. = 1; $P = .18$) or between normal and amblyopic groups ($F = 2.27$; d.f. = 1; $P = .13$).

Figure 5 shows the axis of the cylindrical error for the children with astigmatism. Of the 63 normal children with astigmatism, 45 (71%) had against-the-rule astigmatism. Astigmatism was present in 46 children with esotropia; of these, 22 (48%) had against-the-rule, 16 (35%) with-the-rule, and 8 (17%) oblique astigmatism. Seventeen patients with amblyopia had astigmatism; of these eight (41%) had against-the-rule, eight (47%) with-the-rule, and two (12%) oblique astigmatism.

Discussion

These cycloplegic results for normal eyes indicate that astigmatism occurs in a larger percentage of infants than adults, and there is a trend toward a decreasing incidence of astigmatism with increasing age. Thus, as recent noncycloplegic studies have concluded, astigmatism is characteristic of the developing human eye. We have no evidence that our group data reflect the course of refractions of individuals over the first three years. Howev-

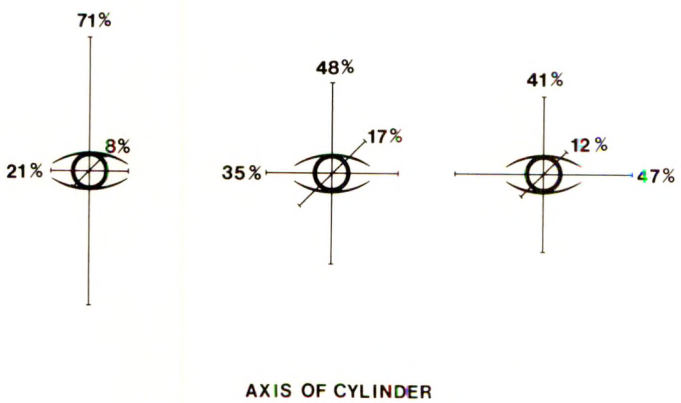


Fig. 5 (Fulton and associates. Axis of significant (≥1.0 diopter) cylindrical error in minus cylinder convention for normal children (left) and children with esotropia (middle) and amblyopia (right). See text for more detailed explanation.

er, in studies of infant visual function, group data and individual data gathered longitudinally have yielded similar results.[16]

There are differences between our cycloplegic results for normal children and the noncycloplegic results shown in the Table. The percentage of infants with astigmatism was smaller, the occurrence of severe (>2.0 diopters) degrees of astigmatism less, and the peak incidence of astigmatism later in our patients. Although an ophthalmologist may be less likely to see very young infants than those actively recruiting subjects for investigation,[4,5] there is no apparent reason why the selection of very young infants being examined by an ophthalmologist should be biased in regard to astigmatism. Also, Santonastaso's study, despite its shortcomings, suggests that discrepancies between cycloplegic and noncycloplegic results are not caused by population differences or variations among refractionists; his refractions were done on the same babies by the same examiner, and noncycloplegic retinoscopies revealed a greater percentage of infants with apparent astigmatism than cycloplegic retinoscopies did (Table). One can infer that there are differences between infantile astigmatism in the noncycloplegic state and that in which accommodation has been suspended pharmacologically. Sources of astigmatism are mainly the cornea and the lens.[17] In addition, Banks' hypothesis,[12] based on the disparity between the optical and visual axes of infants' eyes, predicts against-the-rule astigmatism. Among our normal infants, against-the-rule was more frequent than with-the-rule astigmatism (Table) and over the first three postnatal years against-the-rule astigmatism remained more frequent (Fig. 5). By the age of 4 years, against-the-rule astigmatism is rare.[14] Despite this, keratometry shows the frequency of corneal astigmatism increases between the ages of 2 and 6 years.[14]

The exact sources of infantile astigmatism remain to be determined. If pressure of the eyelids shaped the cornea, with-the-rule astigmatism would result. Therefore, we do not believe eyelid pressure and resulting corneal anisotropy alone account for the observed infantile astigmatism. Photokeratoscopic measurements now underway,[4] correlated with noncycloplegic and cycloplegic refractions of the same patients, will define sources of infantile astigmatism more exactly than is now possible.

In this study of refractive errors in children up to 3 years of age, we found no difference in the incidence of astigmatism between normal children and those with esotropia or normal children and those with amblyopia that cannot be accounted for by preselection. Also, spherical equivalents and anisometropia, whether based on spherical equivalent or meridian of the greatest refractive error,[18] do not identify the majority of children with esotropia and amblyopia in the population. As Ingram and associates have pointed out,[7,8] anisometropia and hyperopia are more common in normal infants and children than adults, but the distributions of spherical equivalents and anisometropia do not distinguish normal infants and children from those with esotropia and amblyopia. This limits the efficacy of cycloplegic refraction as a screening measure.

The straight-eyed child with amblyopia, a youngster with a severe, unilateral refractive error but no obvious strabismus for which ophthalmologic attention would be sought, is not represented in our population until the end of the third postnatal year. If screening refractions detected this vision-threatening situation early, treatment might be more successful.[19] Generally, a screening method, especially for infants and young children,

TABLE

INCIDENCE OF ASTIGMATISM IN THE FIRST POSTNATAL YEAR OF NORMAL INFANTS

Investigators and Technique	Infants Refracted		Cycloplegic Agent	Infants With Astigmatism	Peak Incidence		Axis	Patients With Severe Astigmatism*
	No.	Ages			Age	Astigmatism		
				Noncycloplegic				
Mohindra and associates,[4] near retinoscopy	276	birth-50 wks	None	45% c̄ ≥1.0 diopter	11-20 wks	50%	40% with-the-rule, 40% against-the-rule, 20% oblique	26.8%
Howland and associates,[5] photo-refraction	93	1 day-12 mos	None	49.6% c̄ ≥1.0 diopter (62.4% c̄ ≥0.75 diopter)	1-3 mos. mos. 6-8 mos.	60% ≥0.75 diopter ~50% ≥1 diopter	70% "horizontal or vertical"	11.8%
Santonastaso,[6] retinoscopy	80	birth-1 year	None	60% c̄ ≥1.0 diopter	10-20 wks	87%	NI†	29.6%
				Cycloplegic				
Santonastaso,[6] retinoscopy	80	birth-1 year	Atropine 1%, 1 drop 24 hrs before exam	50% c̄ ≥1.0 diopter	0-10 wks	73% 73%	NI†	12.5%
Ingram,[9] retinoscopy	1,648	1-year-olds	Atropine 1% ointment, twice daily for 4 days	13.2% c̄ ≥1.5 diopters	———	———	NI†	NI†
Ingram and Barr,[10,11] retinoscopy	148	1-year-olds	Atropine 1% ointment, twice daily for 4 days	14.9% c̄ ≥1.5 diopters	———	———	NI†	NI†
Ingram and Barr,[10,11] retinoscopy	614	1-year-olds	Cyclopentolate 1%, 30-40 min before exam	17.5% c̄ ≥1.5 diopters	———	———	NI†	NI†
Fulton and associates, retinoscopy	75	0-60 wks	Cyclopentolate 1%, 30-40 min before exam	19% 0-60 wks. 15% 50-60 wks c̄ 1.0 diopter	30-40 wks 40-50 wks.	25% 23.5%	82% against the-rule 18% with-the-rule	5.7%

*≥2.0 diopters.

†NI, designates not indicated

must be easy and fast. Conventional retinoscopy requires a skilled person. Photorefraction[5] may be relatively easy but requires some care and the equipment is not widely available. An alternative approach might be monocular vision testing with a behavioral method.[20-23]

Summary

We studied groups of normal infants and infants with amblyopia and esotropia to determine the incidence of infantile astigmatism. Under cycloplegia, 19% of normal infants had astigmatism; this was at least twice the incidence in adults, but less than one-half that found by noncycloplegic refractions of infants. During the first three postnatal years the incidence of astigmatism and distributions of spherical equivalents and anisometropia did not distinguish normal patients from most of those with esotropia and amblyopia.

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OCULAR ALIGNMENT AFTER CRANIOFACIAL RECONSTRUCTION

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The work of Tessier and associates[1-5] made the surgical reconstruction of patients with severe craniofacial abnormalities possible. Reconstructive surgery now can be done on young infants because of improved bone and cartilage grafting techniques. Abnormal ocular alignment and movement in patients with severe craniofacial abnormalities have been described previously,[6-13] but little information is available regarding the long-term effect of major craniofacial reconstruction on ocular alignment and maintenance of any pre-existing binocularity.

We compared the preoperative and postoperative ocular alignment in 160 children who had undergone reconstruction by two plastic surgeons at one hospital between 1972 and 1979. The prospective participants were examined by us. The diagnoses of the participating patients are summarized in Table 1.

Of the 160 patients undergoing craniofacial surgery, 120 were examined preoperatively and postoperatively by the ophthalmology department; special attention was given to alignment and adnexal features. Those patients not examined were predominantly classified as having craniofacial dysostosis; all were described by other physicians as having "straight eyes."

TABLE 1

DIAGNOSIS OF PATIENTS UNDERGOING SURGERY

Diagnosis	No. of Patients
Craniofacial dysostosis (Crouzon)	36
Isolated craniofacial dysostosis	33
Hypertelorism, pure	21
Hemifacial microsomia	15
Acrocephalosyndactyly (Apert)	13
Mandibulofacial dysostosis (Treacher Collins)	7
Trauma	6
Assorted (tumor, encephalocele, the Kleeblatschädel syndrome, and the like)	29
Total	160

Alignment was measured in all positions of gaze at 6 meters and 33 cm by using accommodation-controlling targets. A and V patterns were evaluated at 6 meters with the patient's head at approximately 25 degrees of upgaze and downgaze. In older patients, examination of sensory status was attempted with the Worth four-dot device and Titmus polarized stereotest. In the young, fusion was assumed if no shift was noted on alternate-cover testing. We evaluated only primary postiton alignment at distance fixation in this study.

RESULTS

Patients ranged from 2 weeks to 19 years of age, with a mean age of 7 years; 82 females and 78 males were included.

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TABLE 2

PREOPERATIVE AND POSTOPERATIVE PRIMARY POSITION ALIGNMENT

Position Alignment	No. of Patients
Straight pre- and postoperatively	71
Straight preoperatively, surgically induced strabismus	1
Preoperative strabismus, unchanged by surgery	39
Preoperative strabismus, changed by surgery	3
Preoperative third nerve palsy	1
Total	120

TABLE 3

PREOPERATIVE STRABISMUS

Diagnosis	No. of Patients	Percent
Craniofacial dysostosis	18/36	50
Isolated craniofacial dysostosis	2/33	6
Hypertelorism, pure	8/21	38
Hemifacial microsomia	4/15	27
Acrocephalosyndactyly	7/13	54
Mandibulofacial dysostosis	2/7	30

Postoperative follow-up ranged from six months to five years, with a mean of two years and three months. Most patients underwent only one major craniofacial reconstructive operation, but some required more. Some of the younger patients may be considered for further major surgery.

Preoperative and postoperative primary postion alignment at distance fixation is shown in Table 2. This postition of gaze was considered most representative and useful. Only one patient had a new surgically induced deviation.

The highest incidences of preoperative strabismus were in children with acrocephalosyndactyly (Apert) and craniofacial dysostosis (Crouzon) and the lowest in children with isolated craniofacial cysostosis (Table 3). Of the nine patients with surgically induced alteration in primary position alignment, three had a persistent alteration greater than 10 prism diopters.

CASE REPORTS

Case 1—The patient was first seen in 1973 at age 5 years because of a diagnosis of bifacial microsomia. Visual acuity was 6/6 (20/20) in both eyes. Lower eyelid lashes were absent with bilateral notching of the lower eyelids. Onlay rib grafts were placed inside, inferior and lateral to the orbits. Preoperatively, the eyes were straight to alternate-cover testing and the patient fused the Worth four-dot lights at both distance and near. Five years postoperatively, the patient manifested freely alternating constant exotropia of 15 prism diopters.

Case 2—The patient was first examined at age 10 years in 1972 because of a diagnosis of acrocephalosyndactyly. Visual acuity was 6/12 (20/40) in both eyes with Allen cards at distance; exophthalmos was severe. The eyes were straight in primary position but a right hypertropia in left gaze and left hypertropia in right gaze was noted and thought to be secondary to orbital deformities. A LeFort III osteotomy was performed in November 1972, and five years postoperatively a 20 prism diopter constant esotropia for distance and near, and limitation of upgaze were noted.

Case 3—The patient was first examined in 1975 at age 19 years for hypertelorism and median facial cleft. Visual acuity was R.E.: 6/6 (20/20) and L.E.: 6/12 (20/40) and the patient manifested a V pattern exotropia of 6 prism diopters at distance and 20 prism diopters at near, with overaction of inferior oblique muscles in both eyes. Two years after craniofacial reconstruction, a left esotropia of 50 prism diopters was noted as well as the overaction of both inferior oblique muscles. The possibility of a left sixth nerve palsy induced at surgery was considered. The patient was lost to follow-up.

DISCUSSION

Craniofacial surgeons have generally assumed that upper facial mobilization is likely to result in postoperative realignment of visual axis or disruption of any pre-existing binocularity, or both. Thus, correction of strabismus has been routinely postponed until major craniofacial reconstruction efforts have been completed. Most motility surgeons agree that early correction of congenital deviations

is more likely to result in attainment of binocularity. Therefore, it has been unsatisfactory to delay surgery in these children beyond the age of expected attainment of binocularity, and operating for cosmesis alone has been done. Our retrospective study was performed to ascertain the following: (1) if realignment of visual axes results from major craniofacial reconstruction, and (2) if such surgery creates a strabismus where none existed preoperatively.

Of 160 children undergoing surgery, 40 did not have preoperative ophthalmologic examination; however, photographs of all patients were consistent with straight eyes. Of the remaining 120, only nine manifested shift in primary position alignment caused by surgery. Only three had a shift greater than 10 prism diopters. In only one of these nine was a new strabismus created by craniofacial surgery.

The patients maintained preoperative fusional vergence amplitudes after surgery. All but one of the 72 patients with straight eyes preoperatively had straight eyes postoperatively. Additionally, as all patients entering surgery with strabismus had constant tropia, orbital manipulation did not appear to alter ocular alignment permanently in most patients. Possibly this was because of the subperiosteal approach to the orbit used by the plastic surgeons.

Thus, early surgical intervention can be performed based on the angle of strabismus before major craniofacial reconstructive effort, because the latter is unlikely to alter the strabismus. This is advantageous for the following reasons: (1) earlier surgery is more likely to result in attainment of binocularity, and (2) motility surgery after orbital manipulation tends to be technically less ideal, as hemorrhage is more profuse and the soft tissues more friable. Thus, all evidence favors aligning the visual axes at as early an age as possible in patients with craniofacial anomalies, preferably before major craniofacial reconstruction.

Summary

We undertook a retrospective study of 160 patients to ascertain if craniofacial reconstruction affected ocular alignment. In most patients pre-existing alignment and binocularity were unaffected by craniofacial surgery; thus, correction of any strabismus can be performed on a child without concern for disruption of muscle tissue by later craniofacial surgery.

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EPILEPTIFORM OCULAR MOVEMENTS WITH METHYLMALONIC ACIDURIA AND HOMOCYSTINURIA

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Recently a syndrome involving the hematologic and neurologic systems with anemia, intermittent lethargy, and variable dementia has been found to be associated with excretion of methylmalonic acid and homocystine in the urine.[1] The defect appears to be a genetically determined failure in the synthesis of cobalamin coenzymes.[2,3] Six cases (including the present case) have been reported previously.[2,4-7] One such patient was noted at age 5 years to have "frequent blinking of her eyes" which were thought to be minor convulsions and her electroencephalogram showed excess slow-wave activity with frequent discharges."[5] A second patient developed "fluttering" of her eyelids at age 3 years.[6] We studied this patient at age 7½ years, at which time the ocular movement disorder was the major symptom of the disease.

CASE REPORT

A 7½-year-old girl was referred to us from Australia with a diagnosis of methylmalonic aciduria and homocystinuria. She was the youngest of five siblings, one of whom died at age 5 years after spells of fever, lethargy, and coma similar to those of the proposita. No autopsy was performed. The present patient's initial symptoms, beginning at age 8 months, were lethargy and hypersomnia. She slept 14 to 20 hours at a time. "Fluttering" of the eyelids in response to bright lights became conspicuous at age 3 years. Mild hypotonia, ataxia, and some mental retardation were noted at this time. An especially severe episode occurred when, after an alleged attack of influenza, she became lethargic, temporarily lost control of her legs, and had "rolling" of her eyes. From this time the parents dated slow regression in the patient's mental state and continuation of severe blinking. Laboratory investigation showed low levels of methionine and increased total cobalamin (B_{12}), folate, and homocystine in the plasma and increased levels of methylmalonic acid and homocystine in the urine.

Considerable biochemical improvement occurred on treatment with intramuscularly injected hydroxocobalamin, pyridoxine taken orally, folic acid, and choline, and a low protein diet, but "fluttering of the lids" continued to be the parents' chief concern and their major reason for consultation with us. At the time of admission these movements were being treated unsuccessfully with carbamazepine. Except for mild mental retardation the patient was normal neurologically. Computed axial tomography showed slight dilatation of the lateral ventricles. The eyes were otherwise normal: the lenses were not dislocated; visual acuity, color perception (Gunkel chromograph), and dark adaptation were all normal.

The ocular motor abnormality consisted of stereotype blinking of the eyelids and simultaneous upward deviation of the eyes occurring as often as several times a minute and lasting only a few seconds (Figure). At other times they were absent for hours. We confirmed the mother's observation that they were especially frequent in bright lights. Wearing dark glasses was said to decrease the attacks to some extent. The patient was subjectively unaware of the ocular movements and was surprised to see them when confronted by subsequent replay of the videotape. During the few seconds of an attack the patient's responses were delayed. Thus, serial counting was either interrupted or slowed during the period of blinking, but she would carry out simple commands such as putting out her tongue. An electroencephalogram showed generalized 2½-second polyspike and wave discharges that were synchronous with the blinking and typical of the epileptiform discharge seen with absence (petit mal) seizures. Photic driving regularly precipitated the attacks. At other times the ocular movements were full without dissociation or nystagmus.

DISCUSSION

The epileptiform attacks in this patient were typical of absence (petit mal) sei-

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Figure (Cogan and associates). Photographs from a video-clip during a typical seizure characterized by blinking of the eyelids (left) and upward elevation of the eyes (right). The electrodes were applied to record the eyelid movements.

zures characterized by clonic blinking of the eyelids and simultaneous upward deviation of the eyes. This might be interpreted as epilepsy of the Bell's reflex. Absence seizures are generally idiopathic, beginning in childhood or early adolescence. Loss of normal responsiveness during an attack nearly always occurs, and associated phenomena such as mild clonic motion, increased or decreased postural tone, or automatisms are common. Clonus of the eyelids is the most common manifestation. Of 374 videotaped absence seizures analyzed by two observers, 178 had mild clonic motion; 87% of these movements involved the eyelids.[8] Although convulsions are a part of the seizure syndrome, and although 33% of patients with absence seizures will occasionally have a generalized tonic-clonic (grand mal) seizure, this patient had no generalized convulsions.

Patients with defective cobalamin metabolism leading to combined methylmalonicacidemia and homocystinuria have rarely been encountered. A recent analysis of the ability of lines of fibroblasts cultured from individual patients with this phenotype to complement one another biochemically after cellular fusion has led to the subdivision of such patients into two genetically distinct complementation groups (cobalamin C, and cobalamin D).[9,10] Whether these two groups represent lesions in entirely different genes (interallelic complementation), or in different loci within the same gene (intra-allelic complementation) remains to be established.[3,9,10] The present patient was shown to be a member of the cobalamin C group.[10,11] The three previously reported members of this group died at ages 7 weeks,[2] 7 years,[5] and 4 months[7] of illnesses that included a variety of neurologic and vascular abnormalities. The two identified members of the cobalamin D group, brothers aged 14 and 2 years, were less severely affected and are still alive, although the oldest is psychotic and retarded, with abnormal cerebellar and spinal cord function.[3] In only one of these patients has the particular pattern of epileptiform ocular and eyelid movements, noted in our patient, been observed[4]; such ocular movements have not been commented upon in reports of patients with other forms of congenital methylmalonicacidemia or homocystinuria. However, "wandering eye movements" and epileptiform discharges in the EEG have been noted in an infant with similar biochemical abnor-

malities resulting from vitamin B_{12} deficiency. This 6-month-old infant had been exclusively breast-fed by a strictly vegetarian mother.[12]

Summary

A 7½-year-old girl with a rare defect in cobalamin (vitamin B_{12}) metabolism ("cobalamin C" type) developed epileptiform ocular and eyelid movements as the major clinical manifestation of the disease. One of three other patients who have been described with this congenital syndrome was similarly noted to have "fluttering" of the eyelids interpreted as epileptic discharges. The metabolic abnormality produced a defect in synthesis of cobalamin coenzymes. It is characterized biochemically by the excretion of methylmalonic acid and homocystine in the urine.

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EFFECTS OF TOPICALLY APPLIED OCULAR DECONGESTANT AND ANTIHISTAMINE

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Ocular hay fever is characterized by itching and redness of the eye.[1] Topically applied allergens[2] and histamine[3] produce vasodilation and itching when instilled into the human eye. This histamine model of allergic ocular signs and symptoms has been developed for humans and used without evidence of systemic reaction or other adverse side effects. Agents that counteract the effect of histamine (antihistamines) and others that counteract blood vessel dilation (vasoconstrictors) are available for use in the eye.[4] The role and effectiveness of ocular antihistamines and antihistamine-vasoconstrictor combinations for itching, red eyes have not been well established. Corticosteroids are often effective in relieving allergic symptoms, but they are potent drugs that produce a number of serious ocular side effects, including cataract formation, glaucoma, and increased risk of infection.[5] No similar side effects have been reported for antihistamine-vasoconstrictor eyedrops.

We have determined the therapeutic usefulness of an antihistamine (antazoline phosphate) and a vasoconstrictor (naphazoline HCl), individually and in combination, in a histamine model of ocular itching and redness.

MATERIAL AND METHODS

Preparations—Histamine diphosphate was prepared in sterile distilled water to a concentration of 50 μg/ml. The vasoconstrictor (naphazoline 0.05%) and antihistamine (antazoline 0.5%) solutions were prepared under sterile conditions using an aqueous isotonic buffered solution preserved with phenylmercuric acetate. The buffer alone served as the placebo control.

Subjects—A total of 50 normal subjects participated in two independent comparative studies, each including 25 subjects. All subjects were in good health, had no ocular disease, and had refrained from the use of antihistamines and any systemic medication that might affect the test results. No eyedrops other than the test solutions were used throughout the test period.

Method of testing—Before testing, injection of the conjunctiva was evaluated by slit-lamp examination, as part of an external ocular examination conducted at each session for each subject, and graded on a scale of 0 to 2+ (0 = none, 1+ = mild redness, 2+ = gross redness). One eyedrop of the designated test solution was then placed in one eye and the placebo solution in the fellow eye of Study 1 subjects (Table 1). In Study 2, two additional comparisons of the individual test solutions with the combi-

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TABLE 1

STUDY 1 REGIMEN

Challenged Eye	Fellow Eye
Antazoline 0.5%	Placebo
Naphazoline 0.05%	Placebo
Antazoline 0.5%/ naphazoline 0.05%	Placebo
Placebo	Placebo

nation solution were added, and the placebo-placebo comparison was deleted (Table 2). After five minutes, each eye was challenged with 10 µl (500 ng) of the histamine solution. The eyes were then re-examined for redness, and in Study 2 were judged for the presence or absence of chemosis. Each subject indicated which eye itched more and quantified the intensity of itching on a scale of 0 to 3+ (0 = none, 1+ = mild itching, 2+ = moderate itching, 3+ = severe itching that required scratching). The testing sessions in Study 1 were held weekly for four consecutive weeks. In Study 2 five test sessions were held 48 hours apart.

A randomized treatment schedule was devised so that each subject received each of the possible combinations of drugs once. Randomization of the treatment schedule for successive visits was such that no subject received active ingredients in the same eye throughout the study nor did any subject have the same treatment sequence as that of another. With the exception of the placebo-placebo group, the same test ingredient was not placed in both eyes at any one session.

TABLE 2

STUDY 2 REGIMEN

Challenged Eye	Fellow Eye
Antazoline 0.5%	Placebo
Naphazoline, 0.05%	Placebo
Antazoline 0.5%/ naphazoline 0.05%	Placebo
Antazoline 0.5%	Antazoline 0.05%/ naphazoline 0.05%
Naphazoline, 0.05%	Antazoline 0.5%/ naphazoline 0.05%

To determine if any of the components had a local anesthetic effect, anesthesimeter measurements were performed. In Study 1, readings were taken after instillation of both test solutions and histamine; in Study 2, readings were taken before instillation of test solutions and after instillation of histamine.

Statistical comparisons—Before the differences in degrees of redness and itching could be evaluated, it was necessary to subtract the baseline scores for each eye from the corresponding testing session scores. Because injection measurements were categorized (ciliary, conjunctival, and episcleral vessels), it was necessary to total the three resulting scores. A maximum redness score could, therefore, be 6+ and a maximum itching score could be 3+ for any one eye.

McNemar's test for correlated proportions[6] was used to detect differences in the presence of itching and between drug and placebo. The Wilcoxon sign rank test[7] was used to detect differences in degree of redness and itching in eyes treated with various test solutions.

RESULTS

Pretreatment with antazoline (Study 1, $P < .025$; Study 2, $P < .05$) or a combination of antazoline/naphazoline (Study 1, $P < .005$; Study 2, $P < .025$) significantly prevented itching in the histamine-challenged eye (Figure). Both studies also demonstrated that naphazoline (Study 1, $P < .005$; Study 2, $P < .001$) or a combination of antazoline/naphazoline (Study 1, $P < .001$; Study 2, $P < .001$) significantly prevented redness in the histamine-

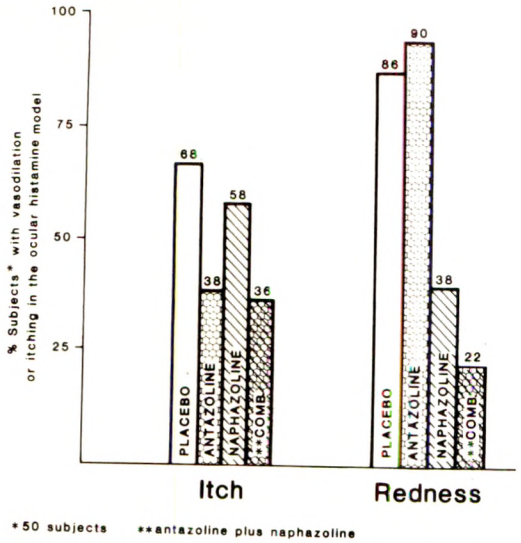


Figure (Abelson, Allansmith, and Friedlaender). Inhibition of itching and redness in the histamine model of ocular allergy. Note that the antihistamine (antazoline) prevents itching and that the vasoconstrictor (naphazoline) inhibits redness.

challenged eye. Conversely, in the placebo-controlled comparisons, naphazoline did not inhibit itching (Study 1, P = not significant; Study 2, P = not significant), and antazoline did not arrest redness (Study 1, P = not significant; Study 2, P = not significant).

When the combination solution was compared with antazoline, the combination-treated eye showed a significant decrease in the redness produced by histamine ($P < .001$), but there was no difference in the presence or degree of itching ($\chi^2 = 0$, not significant).

Comparison of naphazoline with the combination solution also showed differences in the effects of the test solutions. With respect to the presence and degree of itching produced by histamine after treatment with both test solutions, the results did not show the combination to be significantly more effective than naphazoline alone ($Z = 1.47$, $P = .14$, Wilcoxon sign rank test, two-tailed).

When the combination test solution was compared to naphazoline, with respect to the presence and degree of redness produced by histamine, a significant difference marked the combination-treated eyes ($\chi^2 = 5$, $P < .05$, McNemar's test; $Z = 2.20$, $P = .03$, Wilcoxon sign rank test).

Chemosis was found in most eyes in Study 2 after pretreatment with the test solutions followed by challenge with histamine. Results indicated that the number of eyes with chemosis was equal for antazoline and placebo (21/25) and approximately equal for naphazoline and the combination product (15/25 and 14/25, respectively).

No pain or redness was caused by the antazoline in this clinical model. Some of the subjects who received antazoline treatment, alone or in combination, reported a brief stinging sensation. No decrease in corneal sensitivity was found in any eye.

Discussion

Results of two independent studies show that the pretreatment of a human eye with antazoline 0.5% or the combination of antazoline 0.5% and naphazoline 0.05% is an effective method of preventing itching in the histamine model of acute ocular allergy. Pretreatment with naphazoline, alone or in combination with antazoline, is an effective method of vasoconstriction in the histamine model.

Study 2 showed that antazoline alone and antazoline in combination with naphazoline were equally effective in arresting itching, whereas the combination solution was significantly more effective in preventing redness.

The combination of antazoline/naphazoline was significantly better in preventing redness than was naphazoline alone. The increased whitening of the eye caused by the combination solution may be the result of the presence of antihistamine in the solution and its effect upon

the conjunctival, ciliary, and episcleral vessels. The combination solution could not be shown to be more effective than antazoline alone in arresting itching. The sample size of 50 is believed to be the controlling factor; if the sample were theoretically doubled and the degree of itching produced in these 100 subjects were considered, the difference would be significant ($Z = 2.23$, $P = .02$, Wilcoxon sign rank test).

Summary

In two independent studies including 25 subjects each, naphazoline caused significant whitening (but did not prevent itching) in the histamine-induced red, itchy eye. Antazoline inhibited itching (but not redness) to a significant degree in the same model. The combination of naphazoline and antazoline produced significant whitening and inhibition of itching in all eyes challenged by histamine. The combination of the two drugs was more effective than either component alone in preventing redness. The antihistamine and combination of antihistamine/vasoconstrictor were equally effective in arresting itching.

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HEMANGIOMA OF THE BONY ORBIT

ALVIN H. BRACKUP, M.D., F.A.C.S., MELVIN L. HALLER, M.D., AND MICHAEL M. DANBER, M.D.

New York, New York

Hemangiomas of the bone constitute less than 1% of all bony tumors. A primary hemangioma in the bones of the skull occurs even less frequently. To the best of our knowledge there has been only one other case of hemangioma of the zygoma reported.[1]

CASE REPORT

A 46-year-old woman came here in October 1977 because of a painful "lump" under her right eye of two months' duration. She had no history of trauma or sinusitis. She was in good health and was using no medication.

On examination her best corrected visual acuity was 6/7.5 (20/25) in each eye. There was no proptosis or diplopia and extraocular movements were full and equal in each eye. Intraocular pressure was 18 mm Hg in each eye. Ophthalmoscopic examination showed a nevus temporal to the macula in the left eye. Visual fields by Goldmann perimetry were full in each eye. Palpation showed a 1-cm hard bony mass at the temporal end of the right orbital rim that was not moveable and extended onto the anterior wall of the antrum below and the floor of the orbit above. It was slightly tender to the touch. There were no pulsations present nor was there any discoloration of the skin. The skin was freely moveable over the tumor.

Routine laboratory studies, chest X-ray, and electrocardiogram were normal. Physical examination disclosed no other tumors.

X-rays including hypocycloidal polytomography were first reported as showing the anterior inferior rim of the orbit to be markedly elevated. The osseous structure was described as vesicular in nature consisting of coarsened trabecular bone such as might be seen in fibrous osteoma (Fig. 1). On re-evaluation later, they were consistent with the honeycomb appearance of hemangioma (Fig. 2). On tangential views there was the "sun-ray" appearance produced by the radiating bony trabeculae (Fig. 3).

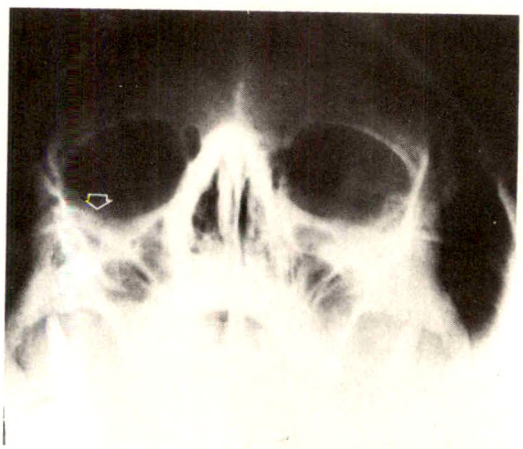


Fig. 1 (Brackup, Haller, and Danber). Coarsened trabecular bone (arrow), lower right orbital rim.

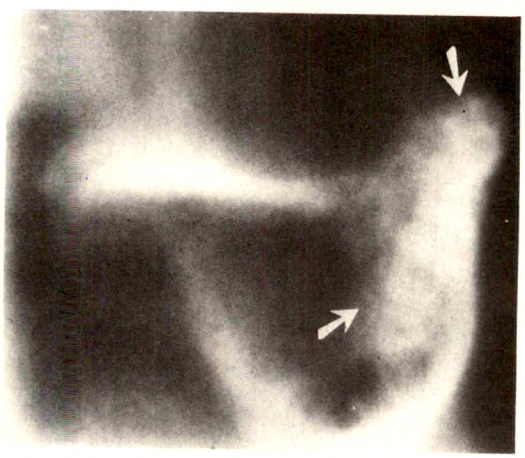


Fig. 2 (Brackup, Haller, and Danber). Honeycomb rarefaction of bone on polytomographic cut-sagittal view (arrows).

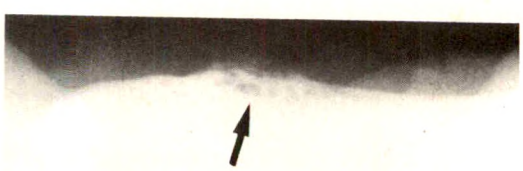


Fig. 3 (Brackup, Haller, and Danber). "Sun-ray" appearance on tangential view (arrow).

From the Department of Ophthalmology, Albert Einstein College of Medicine, New York, New York (Drs. Brackup and Haller); and the Department of Pathology, New York Medical College, New York, New York (Dr. Danber).

Reprint requests to Alvin H. Brackup, M.D., 30-74 36th St., Long Island City, NY 11103.

A technetium 99m scan showed increased nucleotide concentration in the right infraorbital area (Fig. 4) consistent with increased osteogenic activity.

At surgery, using the same approach as for the repair of an orbital floor fracture, we found an elevated, cystic, friable lesion on the orbital rim (Fig. 5). This was excised flush with the normal bony contour by sharp dissection with suction periosteal elevator. There was marked bleeding that could only be controlled finally with bone wax. Postoperatively, healing was uneventful with no further pain in the area.

The pathology report on microscopic examination showed bony trabeculae containing dilated vascular spaces with red blood cells. The vascular spaces were lined with flattened endothelial cells and were surrounded by loose fibrous tissue. The findings were consistent with the diagnosis of hemangioma (Fig. 6). An examination of the slides and X-rays by the Armed Forces Institute of Pathology confirmed the diagnosis of cavernous hemangioma of the bony orbit.

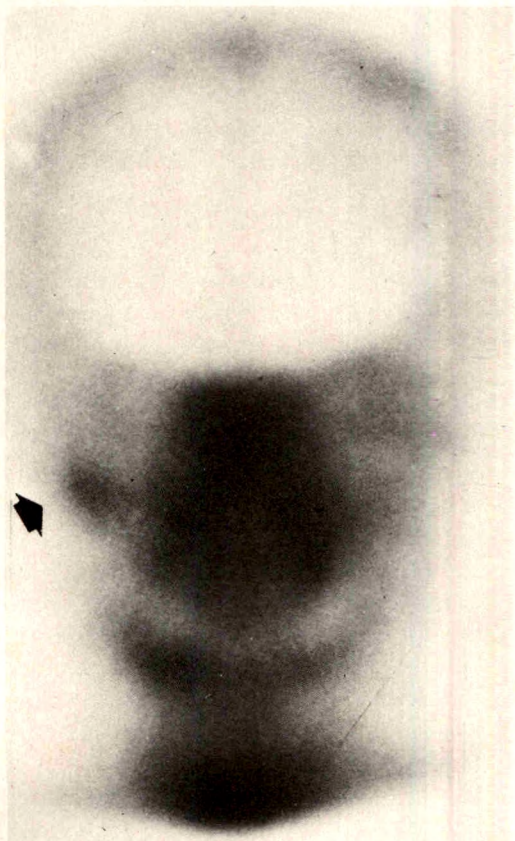


Fig. 4 (Brackup, Haller, and Danber). Technetium 99m scan showing increased uptake in area of tumor (arrow).

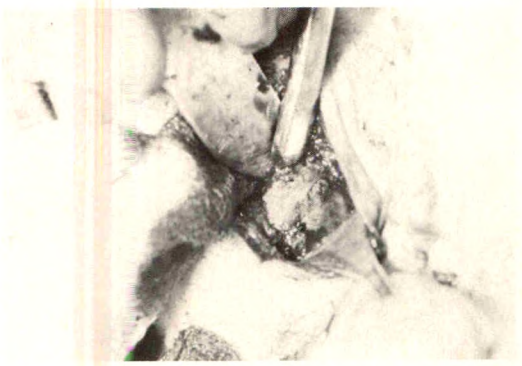


Fig. 5 (Brackup, Haller, and Danber). Elevated cystic friable lesion before surgical removal.

Discussion

Benign hemangiomas of the skull are most frequently found in patients between 31 and 40 years of age. They occur rarely before age 30 years. They occur three times as frequently in women as in men. Most of the tumors are located in the parietal and frontal bones with only two having been reported in the bones of the orbit.[2] Of the two, only one was reported in the zygoma.

The cause is unknown; however, the usual cause is believed to be either congenital in origin or related to previous trauma. It is difficult to diagnose a primary hemangioma purely by clinical methods and we must relay on X-ray studies and biopsy.

The X-ray findings are characteristic. There is a rounded area of rarefaction that has a diagnostic honeycomb appearance. Rarely, there may be evidence of osteosclerotic reaction around it, but this is usually absent. The spaces between the bony trabeculae may vary in size. On tangential views there is the characteristic sun-ray appearance produced by radiating bony trabeculae. These are arranged in a radial manner originating from a common center.

At surgery we usually find a hard, blue-domed lump on normal bone that



Fig. 6 (Brackup, Haller, and Danber). Microscopic sections showing dilated vascular spaces with flattened endothelial cells in bony trabeculae (×500).

can be removed. Deep red blood cysts are apparent while the tumor is in place but these collapse when the bone is removed, leaving crater-like depressions from which dark venous blood oozes.

Histologically, after decalcification, the tumor may be either capillary or cavernous in type. Nearly all hemangiomas of the skull, as in our case, are cavernous. They consist of large blood-filled lacunae embedded in connective tissue that are lined by large flat endothelial cells. Their walls do not contain the usual muscular or elastic tissue components characteristic of normal blood vessels. Both osteoblasts and osteoclasts are present.[3]

Primary hemangiomas of the skull are always benign. Treatment consists of excision with restoration of the defect created by either bone grafts or alloplastic replacements. Irradiation is seldom necessary and is only used where it is impossible to remove the entire tumor. Our patient is asymptomatic at present and we will only consider complete excision if she becomes symptomatic again.

Differential diagnosis should include cholesteatoma, fibrous dysplasia of bone, osteoma, osteogenic sarcoma, and aneurysmal bone cyst of the orbit.[4] The X-ray appearance is the most helpful in diagnosis preoperatively; however, the history is helpful in distinguishing cavernous hemangiomas from aneurysmal bone cysts because the latter usually occur in adolescents.

Summary

A 46-year-old woman had a hemangioma of the zygoma, which was excised

flush with the normal bony contour. To the best of our knowledge, this is the second such case reported.

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OPHTHALMIC MINIATURE

"The retinal nerves were switching from cones to rods and a faint area of light was forming to my left, higher than where I stood, and as I watched it I could feel the gooseflesh reaction along the arms . . . "

Adam Hall
The Sinkiang Executive
New York, Doubleday, 1978

NOTES, CASES AND INSTRUMENTS

A 20-GAUGE INTRAVITREAL CRYOPROBE

HERBERT S. WOLDOFF, M.D.
Phoenix, Arizona

The vitreous probe designed for use with the Frigitronics nitrous oxide cryosurgical system has been, up to now, 18 gauge (1.25 mm). This diameter is adequate for open-sky removal of a dislocated nucleous or intravitreal foreign body. This diameter of probe tip is also excellent for intracapsular cataract extraction.

Vitrectomy systems with a 20-gauge (0.89 mm) outside diameter are commonly used for trans pars plana vitrectomy. Use of the larger 18-gauge intravitreal probe during closed vitrectomy with these systems has necessitated enlargement of the sclerotomy site for endocryocoagulation. Subsequent sclerotomy leakage or entrance wound complications are a problem.

An intravitreal probe* (Figure) with a 20-gauge outside diameter and a shaft length of 35 mm is now available. It features low-pressure freeze and defrost cycles and can be gas-sterilized or autoclaved. The rate of freezing is controlled by varying the pressure of nitrous oxide (controlled on the console) from 400 psi to 600 psi. The probe design is such that only the probe tip freezes; no polyethylene sleeve for its shaft is necessary when the pressure is correctly adjusted for the various load conditions.

I have used the Frigitronics 20-gauge intravitreal probe for endocryocoagulation during vitrectomy. It has been used successfully to close retinal breaks by surrounding the tear with endocryopexy. The end point is determined by observing retinal freezing. Watch for retinal edema around the break after treatment. Be careful not to overtreat too heavily or hemorrhage from the choroid may occur.

The 20-gauge intravitreal cryoprobe also provides the surgeon with an excellent tool for selective controlled retinal destruction during trans pars plana vitrectomy. I plan to study its efficacy for retinal ablation at the time of vitrectomy. The probe also can be used, without the sleeve, for intracapsular cataract extraction.

SUMMARY

A 20-gauge intravitreal cryoprobe designed for endocryopexy during trans pars plana vitrectomy is now available. The probe may also be used for intracapsular cataract extraction and open-sky intravitreal cryopexy.



Figure (Woldoff). New 20-gauge intravitreal cryoprobe.

From the Rockefeller and Abbie Prentice Eye Institute, St. Luke's Hospital Medical Center, Phoenix, Arizona.

Reprints requests to Herbert S. Woldoff, M.D., Arizona Retina Associates, Ltd., 421 N. 18th Street, Suite 112, Phoenix, AZ 85006.

*This instrument is available from Frigitronics, Inc., Shelton, CT 06484.

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MEETINGS, CONFERENCES, SYMPOSIA

EDITED BY THOMAS CHALKLEY, M.D.

AMERICAN OPHTHALMOLOGICAL SOCIETY 1980 MEETING

The American Ophthalmological Society held its 116th annual meeting at Kamuela, Hawaii, May 21-26, 1980, under the presidency of John W. Henderson of Ann Arbor. Some 100 members and guests enjoyed a stimulating scientific program, took advantage of the golf, tennis, and water diversions at the Mauna Kea Beach Hotel, and met with friends old and new.

Eleven associate members were elected in 1979. Most attended the meeting and presented or discussed a paper. They included Daniel M. Albert, Boston; Robert C. Drews, Clayton, Missouri; William B. Glew, Washington, D.C.; Froncie A. Gutman, Cleveland; Malcolm N. Luxenberg, Augusta, Georgia; Manuel N. Miranda, San Juan; J. Donald Morin, Toronto; Frank M. Polack, Gainesville, Florida; Irving P. Pollack, Baltimore; Stephen J. Ryan, Los Angeles; and David S. Walton, Boston. Jerome P. Perlman was also elected posthumously in 1979.

The Howe Medal, the highest award of the American Ophthalmological Society, was given to Frederick Blodi in recognition of his clinical, scientific, and editorial contributions to ophthalmology. Edward W. D. Norton, a previous recipient, followed the traditional pattern of not revealing the name of the recipient until after he had described his schooling, background, and contributions to ophthalmology. Early in his discussion, though, it became evident that only Fred Blodi could have provided such a unique background. Dr. Blodi was particularly touching in his response, thanking his parents and his teachers for his education and recalling the warm welcome he received when he came to America after World War II.

William F. Hughes was elected president to succeed John W. Henderson. Robert Hollenhorst of Rochester, Minnesota, was named vice president to succeed Dr. Hughes. Thomas Kearns, of Rochester, Minnesota, follows Dr. Hollenhorst as secretary-treasurer; Stanley Truhlsen assumes full-time editorial responsibilities for the Transactions. Larry Calkins was elected to the Council, and Taylor Asbury succeeds A. Edward Maumenee as chairman of the Council. Nineteen associate members were elected.

The next meeting will be held May 24-27, 1981, at the Homestead in Hot Springs, Virginia.

FRANK W. NEWELL

INTERNATIONAL COUNCIL OF OPHTHALMOLOGY

The International Council of Ophthalmology met in London April 19, 1980, during the Centenary of the Ophthalmological Society of the United Kingdom.

Those who attended the meeting included the following: Jules François, president; A. E. Maumenee, vice president; E. Streiff, treasurer; A. Deutman, secretary; Barrie Jones (London), E. Grom (Caracas), J. Diallo (Dakar), J. Hudson (London), T. Krwawicz (Lublin), M. Luntz (New York), E. Malbran (Buenos Aires), F. Newell (Chicago), M. M. El Din Said (Alexandria), H. Saraux (Paris), and W. Straub (Marburg).

Dr. Maumenee gave a report on the organization of the XXIV International Congress of Ophthalmology in San Francisco in 1982, which will be held in association with the American Academy of Ophthalmology. The main reports for the Congress will be as follows:

A. Advances in Surgical Techniques: (1) cornea (C. Barraquer, Bogota), (2) cataract

(J. Charleux, Lyon), (3) vitreous (R. Machemer, Durham), (4) glaucoma and biological aspects of ocular reactions in surgery (S. Mishima, Tokyo).

B. Advances in Diagnostic Techniques and Genetics: (1) electrophysiology (H. Henkes, Rotterdam), (2) ultrasonography (K. Ossoinig, Iowa City), (3) automation of perimetry and modern methods of subjective examination of the eye (F. Fankhauser, Bern), (4) modern analytic genetics and the eye (I. Maumenee, Baltimore), and (5) multifactorial heredity in ophthalmology (J. François, Ghent).

Professor Deutman reported on the relations of the Council with the World Health Organization (WHO). Our Council will be a liaison between the ophthalmological societies, the International Agency for Prevention of Blindness, and WHO, and will communicate to the affiliated societies the most important information.

Professor Barrie Jones reported on the International Agency for Prevention of Blindness and Professor Miller on the International Study Committee on Teaching and Continuing Education in Ophthalmology.

The Council received an extensive report on the standardization of the electrophysiological examinations in ophthalmology from H. Van der Tweel (Amsterdam), chairman of a committee composed of Professor Tazawa, Dr. Hellner, Dr. Van Lith, Dr. Carr, and Dr. Lawwill.

The Council is in favor of colored labels for eyedrops (or ointments): red for mydriatics, green for miotics, yellow for anesthetics, blue for antibiotics, and brown for corticosteroids.

The Council edited "Perimetric Standards," which is available from JUNK bv, P.O. Box 13713, NL-2501 ES The Hague, The Netherlands.

Two new societies, the Jordanian Ophthalmological Society and the Ophthalmological Society of Singapore, joined the International Federation.

The Council accepted with gratitude the donation of a Duke-Elder Gold Medal from Lady Duke-Elder.

The next meeting will be held in Acapulco, Mexico, in May 1981.

JULES FRANCOIS

NEW WILLS EYE HOSPITAL DEDICATION

The new Wills Eye Hospital was dedicated in Philadelphia April 23, 1980, in an outdoor ceremony. Ophthalmologist-in-Chief Thomas D. Duane acted as Master of Ceremonies for the event. William S. Tasman, Co-Director of the Retina Service and author of a recently published history book about Wills Eye Hospital, spoke on the hospital's history. He also presented an honorary certificate to Theodore C. Wills, a great-great-nephew of James Wills, Jr., the Philadelphia Quaker merchant whose original $108,000 bequest led to the founding of the institution in 1832. Drs. Duane and Tasman were joined by city officials, members of the clergy, and Hospital Executive Director David S. Joseph, who also spoke.

The dedication was timed to occur the day before Wills Eye Hospital's Annual Clinical Conference, which attracts nearly 1,000 participants from all parts of the nation. Many, including a number of Wills ex-residents, were able to attend the ceremony and the reception and take advantage of the tours that followed.

The oldest and largest hospital in the country devoted solely to eye care, Wills was first established as a 70-bed, three-story institution on Logan Square in Philadelphia. In 1932 the Hospital relocated to a 120-bed, seven-story facility at 16th and Spring Garden Streets where it remained until the new building at 9th

and Walnut Streets opened this past March. The new 120-bed hospital, an award-winning architectural design, features eight operating rooms equipped with ceiling-mounted operating room microscopes and video cameras and greatly expanded outpatient areas. The new hospital provides 207,000 square feet of space in eight stories adjacent to the Thomas Jefferson Medical College complex. The top three floors, each providing 40 beds, are for inpatient care.

Basic research, including clinical and experimental pathology laboratories, occupies most of the fourth floor. Education, nursing, and business offices and a superb library complete the area.

On the second and third floors are the subspecialty, outpatient clinics including Retina (which includes Uveitis), Oncology, Cornea (and External Disease), Glaucoma, Pediatric Ophthalmology, Neuro-Ophthalmology, and Oculo-Plastics. We have also planned for the resident on-call suites, the administrative offices, Board room, and space for sundry other activities (including retinal photography, ultrasound, neurophysiology recording and the like) in strategic places.

The first floor houses the main entrances for ambulatory patients. A doctor's lounge, audiovisual laboratories, an auditorium, and a strategically placed receiving and truck loading platform are designed to avoid conflict with one another on this floor. The main entry area will be a pleasant environment for patients and their families and for employees. The General Ophthalmology Service and the Emergency Room constitute the main features of the professional space. The Pharmacy, Women's Committee Gift Shop, and commercial outlets (including a bank and opticians) likewise add to the ambience of this floor. The basement houses the kitchen, dining rooms, employee locker rooms, lounges and maintenance quarters.

THOMAS D. DUANE

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THE MANAGEMENT OF CHOROIDAL MELANOMAS

The management of malignant melanoma of the choroid has recently become a topic of considerable controversy. Several years ago most ophthalmologists believed that enucleation was warranted without delay for any eye containing a mass suspected of being a choroidal melanoma.[1] A number of more recent observations have led to a reassessment of the advisability of enucleation as a treatment for choroidal melanomas.

One observation that prompted reassessment of early enucleation was that many eyes removed because of a suspected choroidal melanoma were shown on subsequent pathologic studies to contain a lesion that simulated a melonoma (pseudomelanoma) rather than a true melanoma.[2,3] Another development that has led to reassessment of early enucleation was a recent reappraisal of Callender's classification of choroidal melanomas in which many small spindle-cell melanocytic tumors were downgraded from a malignant category to a benign one.[4] As a result, an ophthalmologist who several years ago enucleated an eye for a small choroidal melanoma diagnosed histopathologically as malignant may now be advised retrospectively that he actually removed a useful eye containing a benign tumor.

A third development that has brought about reassessment of the advisability of enucleation for small melanomas is the finding on follow-up studies of untreated small melanocytic choroidal tumors that most lesions of this type are biologically dormant, have little tendency to grow, and virtually no tendency to metastasize.[5,6] A fourth observation that has prompted reassessment of early enucleation is that removal of an eye containing a malignant melanoma may not improve the patient's prognosis for life and may actually hasten his demise from disseminated metastases.[7,8]

A recent article by Manschot and van Peperzeel[9] disregarded the observations set forth above and asserted the advisability of early enucleation of any eye containing a presumed choroidal melano-

ma. Their article has further clouded the choroidal melanoma management controversy and requires critical comment.

Manschot and van Peperzeel used data that are largely theoretical, based on studies of highly malignant nonocular tumors, to draw conclusions regarding the clinical behavior of small choroidal melanomas. They assumed that all melanocytic choroidal lesions greater than 5 mm in diameter are steadily growing tumors which, if not treated, will lead eventually to metastasis and death. Our clinical data suggest that this assumption is incorrect.

Between 1974 and 1979 almost 500 patients with a uveal melanoma were examined and managed by the Oncology Service of Wills Eye Hospital. Approximately 90% of the 175 patients with a small melanoma (between 5 × 5 × 2 mm and 10 × 10 × 3 mm) and about 30% of the 125 patients with a medium-sized melanoma (between 10 × 10 × 3 mm and 15 × 15 × 5 mm) have been followed up with serial fundus photographs and other ancillary studies without enucleation or other forms of therapy. Preliminary data from these patients suggest that only 10 to 15% of small melanomas and approximately 50% of medium-sized melanomas can be documented to grow. The remaining patients with small or medium-sized melanomas appear to have biologically dormant lesions that grow slowly if at all and pose no serious threat to the patient's general health.

These observations suggest that early enucleation of eyes containing a small melanocytic choroidal tumor, as advocated by Manschot and van Peperzeel, will result in unnecessary enucleation in at least 80% of cases. This seems irrational for patients who often have excellent visual acuity in the involved eye. Our preliminary data also suggest that patients with small melanomas, for whom Manschot and van Peperzeel recommend enucleation, rarely if ever, develop metastases even when no treatment is used. If enucleation were not performed, these patients would continue to lead healthy lives as binocular rather than as monocular individuals. Our clinical observations suggest that one should not assume that a pigmented lesion will grow or produce metastasis just because it is there. Most of these lesions probably develop or acquire pigmentation at puberty and most remain permanently stable.

On the basis of their theoretical material Manschot and van Peperzeel concluded that the only acceptable method of management is "early enucleation of all choroidal melanomas, but especially of small melanomas, in order to prevent the initial or later dissemination of tumor cells" and that "all other treatments, such as photocoagulation, radiation and excision of choroidal melanoma, and certainly also the attitude of observation, will be harmful to the patient because dissemination of tumor cells is promoted, either actively or passively." Our clinical data also suggest that this assumption is incorrect.

In recent years we have routinely used methods other than enucleation to manage almost all patients with small and medium-sized choroidal melanomas that show evidence of growth.[11] Since 1974, we have managed 35 patients with photocoagulation of their melanoma. There have been no tumor-related deaths in this group and none of these patients currently have evidence of metastatic disease. Since 1975, 90 patients have been managed with episcleral cobalt plaque radiotherapy. Two of these patients have died of disseminated metastasis; one of them had clinical and laboratory evidence of hepatic metastasis before placement of the plaque, and both of them had a large melanoma (greater than 15 × 15 × 5 mm) at the time of treatment. Since 1974, ten patients have been treated with eye wall

resection for choroidal melanoma. None of them have developed metastasis despite a significant number of epithelioid cells having been demonstrated pathologically in some of the resected tumors.

To date, there have been no tumor-related deaths in our series of patients with small and medium-sized melanocytic choroidal tumors observed without treatment or treated with methods other than enucleation. This compares favorably with reported mortality among similar patients managed by enucleation.[10] Data from the Oncology Research Laboratory at Wills Eye Hospital suggest that the immunologic antimelanoma defense mechanisms in patients with a choroidal melanoma may be enhanced by treatment such as photocoagulation or irradiation but not by enucleation.[12] We currently believe and recommend that most patients with small choroidal melanomas should be managed by observation and serial photographs only and that most patients with medium-sized choroidal melanomas should be managed by observation or methods other than enucleation, particularly if the patient is older, or if the involved eye is the only eye that has useful vision.

Our duty as ophthalmic clinicians is also to salvage vision, particularly if this can be achieved without endangering the patients' systemic health. The value of conservative treatment rather than early enucleation in such cases is supported by our preliminary clinical data.

JERRY A. SHIELDS
AND
JAMES J. AUGSBURGER

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OBITUARY

PETER CLEMONS KRONFELD
1899-1980

Peter C. Kronfeld, professor emeritus of ophthalmology at the University of Illinois Medical School, died January 1, 1980, at the age of 80. It was characteristic that his quiet reserved manner, coupled with his enthusiasm and productivity, concealed the seriousness of his long-standing illness from most of his countless friends and admirers.

It is difficult to put into perspective those characteristics that most distinguished this remarkably talented person. Born in Vienna, Austria, he retained his continental charm and mannerisms, as

well as his love of the mountains. Often he would recall his childhood memories of Sunday picnics with his family in the mountains near his home. His musical ability was also a product of his early education, culminating in his playing viola in the orchestra of the Vienna State Opera.

His education was interrupted by compulsory service in the Austrian army, but in 1923 he obtained an M.D. degree from the University of Vienna. Although his initial choice for specialization was physiology, he was convinced by Professor Mueller to enter ophthalmology. He completed his residency and then joined the staff of the first eye clinic at the University of Vienna.

During the great invasion of Austria by American ophthalmologists in search of postgraduate education, Dr. Kronfeld was able to communicate in excellent English. His scientific acumen and ability prompted E. V. L. Brown in 1928 to offer him a full-time position at the University of Chicago where he stayed until 1933.

To his good fortune it was necessary for him to return to Vienna in 1929 to complete an unfinished project, and during his stay he became engaged to Christl whom he had met before leaving for the United States. Christl and P. C. K., as Peter was fondly referred to by his close associates, were married in 1930. They were life-long and inseparable partners, she as talented and charming as he. They had two children, P. Paul, an anesthiologist, and Eleanor (Mrs. R. J. Andrews).

Invited to become chairman of the Department of Ophthalmology at the Peking Union Medical College in 1933, P. C. K. was able to resume his interests in the biochemistry of the aspirated aqueous. For recreation in China horseback riding and racing were added to his other athletic accomplishments. When the Japanese invaded China the Kronfelds returned to Chicago at the invitation of Harry Gradle, where Dr. Kronfeld became the Director of Education at the Illinois Eye and Ear Infirmary.



Peter C. Kronfeld, M.D.

In 1959, P. C. K. became chairman of the Department of Ophthalmology at the University of Illinois Medical School.

I think of Dr. Kronfeld primarily as a magnificent clinician who was devoted to the care and healing of the ill. His compassion and sensitivity to patients were boundless, and he treated the indigent, the helpless, and the successful all with the same care, devotion, and politeness. Time was of no concern; if necessary he worked seven days a week, often until the early hours of the morning. His commitment to his patients was reciprocated by their total devotion and love, which continued long after his private practice was curtailed because of the demands of the department and of the university.

Were it not for his enormous energy and determination his patient load would have been overwhelming, but in addition Dr. Kronfeld was a masterful teacher. In his early days at the infirmary his 11 A.M. conferences, where he discussed the difficult cases of the day, were the most inspiring part of the residency. By example or by gentle persuasion we were directed toward the proper care and treatment of the patient as a whole human being. For all students occasionally there is a teacher with a blend of the proper ingredients to inspire and encourage excellence in their colleagues. P. C. K. was one of the best.

Another part of this blend was the research capability which occupied the remainder of his academic time. Over 130 scientific papers, principally dealing with glaucoma, seven chapters and books, and innumerable medals and honors attest to Dr. Kronfeld's success in this area. His dramatic presentations of this work with which we are all so familiar, his characteristic facial, vocal, and postural expressions were the polished result of a highly skilled and broadly experienced intellectual.

After retirement from the university Peter and Christl chose to live in Tuscon, Arizona, surrounded by the mountains and deserts they had grown to love. Within this tranquil setting they were able to enjoy their mutual interests, traveling frequently to visit their children, to meetings, to concerts, or to visit the Navajo Indian reservations where Dr. Kronfeld was a consultant to the Indian Health Service. There was also time to review the German literature for THE AMERICAN JOURNAL OF OPHTHALMOLOGY, and to contemplate the many unanswered questions that remained.

During this somewhat relaxed period the compassion, sensitivity, and warmth that motivated Dr. Kronfeld were readily apparent. P. C. K.'s company, counsel, and wisdom will be sorely missed, but they will never be forgotten by those of us fortunate enough to know him.

JOSEPH HAAS

CORRESPONDENCE

Letters to the Editor must be typed double-spaced on 8½ × 11-inch bond paper, with 1½-inch margins on all four sides, and limited in length to two manuscript pages.

Incidence of Accidents in Patients With Spectacle Correction vs Contact Lens Correction

Editor

It is my hypothesis that patients with spectacle-corrected aphakia have a higher incidence of accidents (fractures of the hip, limb, skull, and the like) than patients with contact lens correction, or pseudophakia. Recently Oliver Dabezies, M.D., demonstrated on film the disability associated with spectacle-corrected aphakia. This lucid demonstration confirmed the hypothesis that I have considered for some time.

A well-controlled, collaborative study in a center where a large volume of orthopedic and ophthalmic surgery is performed might confirm the hypothesis, and lend credence to the concept of improved quality of life in the patient with pseudophakia or wearing a contact lens.

DON J. JACKSON, M.D.
Clarksville, Tennessee

Symblepharon Lysis With a Thermometer

Editor:

Symblepharon lysis is an important and necessary therapeutic maneuver for a variety of diseases, most notably acid and alkali burns, Stevens-Johnson syndrome,

and occasionally epidemic keratoconjunctivitis. Any disease that creates a pseudomembrane or true membrane with the potential for symblepharon formation should be considered for regular symblepharon lysis. In the case of an acute alkali burn, this should be done on a daily or, perhaps, twice daily basis. This procedure should be done with a small glass rod. However, it is often difficult to obtain a glass rod of the appropriate size and a substitute must be found. A readily available instrument in most hospitals is the standard mercury thermometer. This is commonly used in hospitals, easily sterilized by chemical means, and functions well as a glass rod. There are two cautions with this instrument, however.

1. Whatever solution is used to sterilize the instrument should be carefully rinsed off with sterile water before using it as a device for lysis.

2. Any glass rod is potentially fragile and the thermometer likewise can be broken with too vigorous an effort.

IVAN R. SCHWAB, M.D.
AND
ROBERT L. STAMPER, M.D.
San Francisco, California

Pupillary Dilation With Single Eyedrop Mydriatic Combinations

Editor:

I read with great interest the article, "Pupillary dilation with single eyedrop mydriatic combinations" (Am. J. Ophthalmol. 89:553, 1980), by L. Apt and A. Henrick.

For some years, in private practice my three associates and I had used routinely primarily tropicamide (Mydriacyl) and phenylephrine (Neo-Synephrine) for pupillary dilation. Because of repeated problems with inadequate dilation and iris spasms in attempted indirect ophthalmoscopy, we recently switched to cyclopentolate (Cyclogyl) and phenylephrine in order to give a longer and more complete cycloplegia. This has made examination easier, and the dilation more satisfactory. I find it surprising that the authors were able to obtain adequate dilation and cycloplegia for indirect ophthalmoscopy in patients with 0.5% or even 1.0% tropicamide and phenylephrine in single drop form.

Although many patients wonder why their pupils stay dilated so long since we have instituted the cyclopentolate and phenylephrine regimen, I think that the advantages in examination, both in children and adults, far outweigh this disadvantage.

R. LARRY BRENNER, M.D.
Pasadena, Texas

Reply

Editor:

Dr. Brenner finds that phenylephrine (Neo-Synephrine HCl) and tropicamide (Mydriacyl) do not give adequate and maintained pupillary dilatation on attempted indirect ophthalmoscopy, and thus he now uses phenylephrine and cyclopentolate (Cyclogyl) even though mydriasis and impaired accommodation last longer. This clinical experience is at variance with the clinical experience of others as well as ourselves, and with published pharmacologic studies. Although intense light stimulation, as in indirect ophthalmoscopy, may cause constriction of a pupil that has been dilated with only one instillation of phenylephrine, the concomitant use of a cycloplegic eyedrop (including tropicamide) prevents this response to intense illumination.

We obtained wide and sustained pupillary dilatation with one drop of either one of the three mydriatic combination solutions tested: tropicamide 0.5% with phenylephrine 2.5%, tropicamide 1% with phenylephrine 2.5%, and cyclopentolate 0.5% with phenylephrine 2.5%.

Certain factors are of utmost importance in obtaining excellent and sustained mydriasis. Perhaps our comments on these factors will explain Dr. Brenner's incomplete response with tropicamide. First, drugs combined in solution achieve higher intraocular concentrations after instillation of a single eyedrop than when instilled as drops of separate solutions within five minutes because of dilution occurring with multiple drops. Second, prior instillation of a drop of proparacaine 0.5% (Ophthaine, Ophthetic, Alcaine) improves absorption of the administered mydriatics. We advise waiting a minimum of one to two minutes after instilling proparacaine before instilling the mydriatic combination solution. Also we advise gently wiping the closed eyelids to remove the excess proparacaine solution that may be retained in the cul-de-sac before instilling the mydriatic solution, to avoid dilution or overflow. Instillation of the drops by the "pouch method" introduced by Frederick Fraunfelder, and described in detail in our article, assures retention in the cul-de-sac of a maximum amount of fluid and increases the contact time between the solution and the cornea. We cannot overemphasize the importance of the proper method of eyedrop instillation in obtaining wide and sustained pupillary dilation. If the ophthalmologist does not instill the eyedrops himself, he should explicitly teach the proper method to his office personnel.

We believe that Dr. Brenner also can achieve excellent and sustained mydriasis by administering the phenylephrine-tropicamide combination solution in the manner we described. The use of tropicamide rather than cyclopentolate in combination with phenylephrine permits accommodation and mydriasis to return to normal faster.

LEONARD APT, M.D.
AND
ANDREW HENRICK, M.D.
Los Angeles, California

Increased Incidence of Choroidal Malignant Melanoma and the Use of Inappropriate Statistical Methods

Editor:

The report of a cluster of five choroidal melanomas among the present or former employees of a chemical plant by Dr. Albert and his colleagues, "Increased incidence of choroidal malignant, melanoma occurring in a single population of chemical workers" (Am. J. Ophthalmol. 89:323, 1980), requires that a decision, with far-reaching ethical and scientific consequences, be made that this was a chance aggregation, or that the cases resulted from the action of an unrecognized occupational carcinogen. In the absence of any way of predicting for a specific human tumor the carcinogenicity of chemical compound, a necessary element in this decision is an estimate of the probability that this cluster of cases occurred by change.

The authors address the problem, and state that "We have determined that this represents a statistically significantly greater than expected occurrence of choroidal melanomas in the plant white male population." They derived expected numbers of cases from two standard sets of incidence data and obtained estimates of the probability for a plant of this appropriate size of 1/39 and 1/77. Their method was based on the assumption of a single trial of the data; that the particular plant had been specified in advance of knowledge of the actual occurrence of choroidal melanomas among its workers. Such a situation might have occurred if they had been led to the particular plant in response to animal studies or consideration of the actual chemicals handled. Indeed, the identification of the plant rested on an indefinite number of occasions where people looked through plant records, found nothing of note, and took no further steps. Each such negative trial adds to the probability fraction. Thus the

authors' finding of a single plant in the United States with this number of cases when the single trial expectation is as stated is not surprising.

The statistical way out of this difficulty of covert multiple testing is to take the complete set of cases for a large population, and test the randomness of their distribution by standard methods. This has been done for endemic disease, for example leukemia, but can clearly miss small point sources that provide only a small proportion of the total cases. Nevertheless it is mandatory to deal with the effect of multiple trials, whether overt or covert, in statistical analyses where the positive findings have been arrived at by other means than a systematic survey.

The alternative is to seek for more data. A continuation of the occurrence of cases in the plant would clinch the matter, as would discovery of similar groups of cases in comparable plants. Without confirmatory data, or the conduct of appropriate statistical tests, the proposition that the current findings are of statistical significance is an error.

JOHN A. H. LEE, M.D.
Seattle, Washington

Reply

Editor:

Dr. Lee makes the point that out of all the industrial plants in the United States, it is likely that at least one of them might fortuitously have had five cases of choroidal malignant melanoma and that our study may have been but a description of such a fortuitously occurring cluster, identified not as a result of a systematic survey whose hypotheses had been formulated a priori, but rather as a result of multiple overt and covert examinations of different data bases by different investigators. The implication would be that we have studied, after its discovery, the cluster of choroidal malignant melanoma cases that could have been expected to have occurred by chance alone. Dr. Lee additionally notes that the alternative to dealing with the effect of these multiple overt and covert data examinations, on the calculation of the probability fraction upon which is based the inference that a statistically significant excess number of choroidal malignant melanomas has occurred, is " to seek more data. A continuation of the occurrence of cases in the plant would clinch the matter . . . " Although our account of the chronology of events that led to our data collection may not have made it sufficiently clear, this is precisely what we have observed and what we have attempted to describe.

Our efforts began with the identification in late 1976 of two cases of choroidal malignant melanoma from a list of purported ocular tumors occurring at the plant. We submitted to the National Institute for Occupational Safety and Health a contract proposal, dated January 27, 1977 (quoted from our proposal), to undertake "a screening examination of the Belle chemical workers" to address the question, "Is there an increased incidence of malignant melanomas of the choroid, ciliary body or iris in this population?" About this same time, we reported the two cases in a letter to the editor of the New England Journal of Medicine, the implicit statement being that we would hope that additional cases of choroidal malignant melanoma discovered at other chemical plants might be reported if the medical community were made aware of our observations. This resulted in a letter, dated April 7, 1977, from physicians with the National Institutes of Health reporting to us Case 3. Later in 1977, review of company medical records uncovered Case 4. Case 5 was discovered in April 1978 during our ophthalmologic survey. We believe that these last three cases, discovered after our initiation of

plans to study this plant further, are in the nature of "a continuation of occurrence of cases" suggested by Dr. Lee to "clinch the matter."

We noted in our article our reasons for believing our observed and calculated expected number of choroidal malignant melanoma cases to be conservative estimates of the actual occurrence of choroidal malignant melanomas and its expectation in the plant population. Although there is no statistical way to settle the point to everyone's satisfaction, we believe that the true level of probability is closer to our stated level than Dr. Lee's letter would suggest. We agree with Dr. Lee that the discovery of similar cases in comparable plants would add greatly to support the conclusions we have drawn from our data.

DANIEL M. ALBERT, M.D.
Boston, Massachusetts
ALEXANDER BLAIR SMITH, M.D.
AND
SANFORD S. LEFFINGWELL, M.D.
Cincinnati, Ohio

BOOK REVIEWS

A Singular View, revised. By Frank B. Brady. Oradell, New Jersey, Medical Economics, Co., 1979. Hardcover, 129 pages, foreword, publisher's notes, 22 black and white figures. $15.50

Most of us enjoy sight with both eyes and give little thought to the complexities of everyday activities encountered by those who have to make a sudden transition to monocular vision. This manual for the newly one-eyed was written by an aviation engineer who had lost one eye during a bizarre accident caused by a duck crashing through the windshield of a plane in which he was co-pilot. In a breezy and engaging style, the author tells of the adjustment problems that plagued him after the accident. The book provides numerous helpful hints and suggestions of how to cope with the loss of stereopsis and restriction of the field of vision. The teaching of how to use clues of monocular depth perception reflects the aviation and engineering background of the writer. There are also useful chapters on driving, driver and pilot licensing standards in different states, and how to take care of a prosthesis, the socket, and the remaining good eye.

This warm, personal, and often humorous essay by someone who has mastered a difficult situation and resumed an active life in which flying plays a major part is recommended to all patients who have recently lost one eye. Ophthalmologists who read this book will be in a better position to advise their patients on how to cope with a bewildering and initially frightening handicap.

GUNTER K. VON NOORDEN

Congenital and Acquired Color Vision Defects. By Joel Pokorny, Vivianne G. Smith, Guy Verriest, and A. J. L. G. Pinckers. New York, Grune and Stratton, 1979. Hardcover, 409 pages, preface, references, index, 129 black and white figures. $33.50

Forewords to scholarly works, as a rule, are laudatory. The foreword to this work, by Professor W. D. Wright, is no exception. In this case, however, the foreword could have doubled as an unbiased review. This book is destined to serve as a key reference in its area and, to a lesser extent, as a text as well. It is a splendid addition to the published clinical and visual science material. The first two chapters summarize radiometric and photometric measures; the third reviews "physiological and theoretical bases of

normal color vision," thereby providing a framework with which to regard the later chapters. Chapters 4 through 6 introduce terminology and methodology for classifying, screening, diagnosing, and investigating both congenital and acquired color vision defects. The seventh and eighth chapters are massive and would themselves constitute an exhaustive source book, using the terminology, methods and viewpoints of the earlier chapters, dealing with first congenital and then acquired color vision defects and maladies. The book concludes with a small chapter on the "vocational and practical implications of defective color vision."

The structure within each chapter is well organized, making for easy reference. The organization naturally differs from chapter to chapter and somewhat from author to author, but the material is presented throughout so that the reader can easily pinpoint the particular pieces of information for which he is looking. A thorough index makes this task especially easy. The book contains a wealth of information reasonably unbiased toward one side or the other of unresolved issues, and is of great value to both clinicians and researchers. Chapter 8 presents an exhaustive treatment of pathologies and other insults known to affect color vision. The acquired defects are classified using Verriest's terminology and the results of the standard color tests, for example, Farnsworth-Munsell 100-hue, anomaloscope and pseudoisochromatic plates, as well as some laboratory techniques are summarized. Chapter 5, on clinical testing methods, provides an excellent, extremely practical review of more color vision tests than any one person would ever use (unless perhaps he is multilingual). Both the clinician with his own private practice, who possesses perhaps one or two color vision tests, and the ophthalmologist-researcher with a wide variety of techniques and referred patients at his disposal will be able to use the book with profit. Because the list of references is so complete and data from various studies are often combined in one table or graph, the book is a must for any investigator of color vision deficiencies.

One flaw noted by Professor Wright in the foreword and acknowledged by Professors Pokorny and Smith is the traditional description of tritan-type defects as blue-yellow defects, even though the loss of blue sensitivity does not lead to confusion of blue with yellow. We would be hard pressed to find any other significant faults. Perhaps acquired defects of central origin are not given enough coverage, but that is more a reflection of the field than the book. All in all, this is an absolutely marvelous and welcome work.

ALVIN EISNER
AND
JAY M. ENOCH

Disorders of the Peripheral Fundus. By William Tasman and Jerry A. Shields. Philadelphia, Lippincott/Harper, 1980. Hardcover, 227 pages, foreword, preface, index, 188 black and white figures, 3 color figures. $30

Although this text is entitled "Disorders of the Peripheral Fundus," many entities are discussed that primarily involve the posterior pole but sometimes also involve the periphery. The nine well-illustrated chapters cover the following topics: anatomy of the peripheral fundus; methods of examinations; hereditary disorders; development abnormalities; inflammatory diseases; vascular abnormalities; tumors; degenerative condition retinal and choroidal detachment.

Chapter 1 on the anatomy of the peripheral fundus, the sections on persistent hyperplastic primary vitreous in Chapter 4, and the sections on retro-

lental fibroplasia in Chapter 6 are extremely well written. However, many other areas are discussed briefly, presumably because the authors assume that the reader already has some knowledge of the clinical characteristics of the entity being discussed.

Although the book is best directed toward residents in ophthalmology because it provides them with a good introduction to retinal diseases, it also contains a great deal of useful information for the general ophthalmologist and retinal specialist.

MILTON BONIUK

SYMPOSIA

Behçet's Syndrome. Edited by T. Lehner and C. G. Barnes. New York, Academic Press, 1979. Hardcover, 323 pages, preface, index, 88 tables, 100 black and white figures. $38

T. LEHNER AND C. G. BARNES: **Criteria for diagnosis and classification of Behçet's syndrome**

T. LEHNER AND J. R. BATCHELOR: **Classification and immunogenetic basis of Behçet's syndrome**

R. J. LEVINSKY, R. PAGANELLI, AND T. LEHNER: **Immune complexes and their characterization in Behçet's syndrome and recurrent oral ulcers**

E. J. BURTON KEE, T. LEHNER, AND J. F. MOWBRAY: **Antigens in circulating immune complexes of Behçet's syndrome**

Y. H. ABDULLA AND T. LEHNER: **The effect of immune complexes on chemotaxis in Behçet's syndrome and recurrent oral ulcers**

J. M. A. WILTON AND T. LEHNER: **Defective polymorphonuclear leukocyte phagocytosis in patients with Behcet's syndrome and recurrent oral ulcers**

C. M. SCULLY, T. LEHNER, AND R. HARFITT: **Serum, salivary and lacrimal immunoglobulins in Behçet's syndrome and recurrent oral ulcers**

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B. E. D. COOKE: **Oral ulceration in Behçet's syndrome**

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P. GOW, T. LEHNER, AND G. S. PANAYI: **Joint manifestations in Behçet's syndrome and in recurrent oral ulcers**

R. A. C. HUGHES AND T. LEHNER: **Neurological aspects of Behçet's syndrome**

D. GWYN WILLIAMS AND T. LEHNER: **Renal manifestations of Behçet's syndrome**

M. H. LESSOF, D. B. JEFFERYS, T. LEHNER, M. MATTOCK, AND M. D. SAUNDERS: **Corticosteroids and azathioprine. Their use in Behçet's syndrome**

A. M. DENMAN, P. HOLLINGWORTH, A. D. B. WEBSTER, B. K. PELTON, AND E. L. RHODES: **Failure of transfer factor in the treatment of Behçet's syndrome. A double-blind trial**

T. LEHNER AND J. M. A. WILTON: **The therapeutic and immunological effects of Levamisole in recurrent oral ulcers and Behçet's syndrome**

Symposium on Medical and Surgical Diseases of the Cornea. Transactions of the New Orleans Academy of Ophthalmology. St. Louis C. V. Mosby Co., 1980. Hardcover, 641 pages, 603 black and white illustrations, preface, index. $72.50

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DAVID PATON: **The principal problems of penetrating keratoplasty graft failure and graft astigmatism**

RICHARD C. TROUTMAN AND RONALD N. GASTER: **Combined keratoplasty techniques**

JORGE N. BUXTON: **Surgery in keratoconus, including thermokeratoplasty**

PERRY S. BINDER: **Eye banking and corneal preservation**

JORGE N. BUXTON: **Special procedures. Mooren's ulcer, conjunctival flaps, the triple procedure**

JORGE N. BUXTON: **Visual rehabilitation, including rotating autografts**

MAX FINE: **Modified techniques of keratoplasty in aphakia and pseudophakia**

RICHARD C. TROUTMAN AND RONALD N. GASTER: **Effects of disparate-sized graft and recipient opening**

DAVID PATON: **Lamellar keratoplasty**

RICHARD C. TROUTMAN, RONALD N. GASTER, AND CASIMIR SWINGER: **Refractive keratoplasty**

JOSE I. BARRAQUER: **Keratomileusis for the correction of aphakia**

PETER R. LAIBSON AND GEORGE W. WARING: **Corneal cystrophies**

DAN B. JONES: **Viral and chlamydial keratoconjunctivitis**

PETER R. LAIBSON: **Therapy of epithelial herpetic keratitis**

Correction

In the correspondence entitled, "Lysozyme content of tears" (Am. J. Ophthalmol. 89:459, 1980), the name of Mr. Ian A. Mackie, London, England, was inadvertantly dropped as a co-author with D. V. Seal, M.D.

ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

Albrecht von Graefes Archiv für Klinische und Experimentelle Ophthalmologie

HISTOLOGICAL FINDINGS IN RIEGER'S MESODERMAL DYSGENESIS OF THE IRIS. (German) Troeber, R., and Rochels, R. (Univ. Augenklinik, Langenbeckstr., Bundesrepublik Deutschland). Albrecht von Graefes Arch. Klin. Ophthalmol. 213:169, 1980.

The histologic examination of an enucleated eye with Rieger's anomaly showed microcornea, broad anterior synechia, hypoplasia and atrophy of the stroma of the iris as well as of the ciliary body. Excessive proliferation of the endothelium combined with the formation of thick glass membranes allow the assumption that Rieger's anomaly, essential progressive atrophy of the iris, and Chandler and Cogan-Reese syndromes are related in the etiopathogenesis and are only variations of the same proliferative endotheliopathy. (3 figures, 11 references)—David Shoch

IRIS FLUORESCEIN ANGIOGRAPHY AND IRVINE-GASS SYNDROME. (German) Bonnet, M., and Bievelez, B. (Clinique Ophthalmologique, Univer. B., France). Albrecht von Graefes Arch. Klin. Ophthalmol. 213:187, 1980.

Iris fluorescein angiography and fundus fluorescein angiography were performed three months to three years after cataract extraction in two series of aphakic eyes—32 eyes with vitreous adherence to the wound, and 32 eyes with no vitreous adherence to the wound. In these series there is a significant correlation between dye leakage on iris fluorescein angiography and vitreous adherence to the corneal wound since dye leakage was observed in 40.6% of eyes with vitreous adherence to the wound and only 31.1% of eyes with no vitreous adherence to the corneal wound.

On the other hand cystoid macular edema was observed in 100% of eyes with vitreous adherence to the wound associated with dye leakage on iris fluorescein angiography and in only 10% of eyes with vitreous adherence to the wound but no dye leakage on iris fluorescein angiography. (5 figures, 5 references)—Authors' abstract.

Archives of Neurology

PERIODIC ALTERNATING NYSTAGMUS IN PHENYTOIN INTOXICATION. Campbell, W. (Dept. Neurol., Wilford Hall U.S. Air Force Med. Ctr., Lackland Air Force Base, Tex.). Arch. Neurol. 37:178, 1980.

Periodic alternating nystagmus is an uncommon cyclic oculomotor phenomenon consisting of horizontal jerk nystagmus that waxes and wanes in amplitude and reverses direction (alternates) in a periodic set fashion of fairly constant timing in a particular patient. It generally implies structural brainstem disease especially at the craniocervical junction. The patient reported here was an alcoholic who was given a very large dose of phenytoin (Dilantin) in an attempt to prevent withdrawal symptoms. He was admitted to the hospital with numerous neurologic findings most of them ascribable to his alcoholism. However, in addition to the usual findings there was a classic periodic alternating nystagmus. This gradually subsided as the blood levels of phenytoin diminished and eventually cleared completely. The patient continued with his alcoholism and continued to have neurologic findings but the periodic alternating nystag-

mus never returned. There appears to be no previous report of drug-related periodic alternating nystagmus. (2 figures, 16 references)—David Shoch

Archives of Ophthalmology

ANAEROBIC AND AEROBIC BACTERIAL FLORA OF ACUTE CONJUNCTIVITIS IN CHILDREN. Brook, I. (Dept. Pediatr. Univ. California, Irvine, Calif.). Arch. Ophthalmol. 98:833, 1980.

Aerobic and anaerobic cultures and clinical data were obtained from 126 pediatric patients with acute conjunctivitis. Similar cultures were obtained from 66 persons who did not have a conjunctival inflammation. Anaerobes were isolated from 47 patients (37.3%). In 26 cases (20.6%), they were in mixed cultures with aerobes, and in 21 cases (16.7%), they were the only isolates. Aerobes alone were recovered in 72 patients (57.1%) No bacterial growth was noted in seven patients (5.6%). The organisms recovered from eyes with conjunctivitis in statistically significant numbers were *Staphylococcus aureus*, *Streptococcus pneumoniae*, *Haemophilus influenzae*, and anaerobic Gram-positive cocci. (2 tables, 20 references)—Author's abstract

EFFECTS OF OCULAR DECONGESTANTS. Abelson, M. B., Yamamoto, G. K., and Allansmith, M. R. (Dept. Ophthalmol., Harvard Med. School, Boston, Mass.). Arch. Ophthalmol. 98:856, 1980.

Eight widely used commercially available ocular decongestants were compared on the basis of vasoconstrictive effectiveness. The ability of these preparations to counteract histamine-induced erythema and to block its recurrence when rechallenged after one hour was evaluated in six human subjects in seven sessions. All vasoconstrictors tested were effective. No preparation was statistically different from 0.02% naphazoline hydrochloride in vasoconstrictive effect. In fact, this intermediate concentration of naphazoline (0.02%) was found to be significantly better than the other nonprescription decongestant preparations tested as a group of higher concentrations of naphazoline. (4 tables, 4 references)—Authors' abstract

BARING OF THE CIRCUMLINEAR VESSEL: AN EARLY SIGN OF OPTIC NERVE DAMAGE. Herschler, J., and Osher, R. H. (Bascom Palmer Eye Inst., Dept. Ophthalmol., Univ. Miami, Miami, Fla.). Arch. Ophthalmol. 98:865, 1980.

Baring of the circumlinear vessel is reported as an early sign of optic nerve damage. Evidence is presented to show that this finding is not present in the normal population and, unlike previously described signs of glaucomatous cupping, can be observed at a stage of glaucoma prior to the development of visual field loss. Analysis of a series of stereoscopic photographs from selected high-risk ocular hypertensive patients disclosed a 54% incidence of baring of the circumlinear vessel. Although initially thought to be pathognomonic for glaucomatous cupping, baring of the circumlinear vessel is seen rarely in acquired cupping from optic atrophy secondary to other causes. (12 figures, 2 tables, 17 references)—Authors' abstract

Bulletin of the New York Academy of Medicine

ANCILLARY TESTS FOR THE DIAGNOSIS OF RETINOBLASTOMA. Abramson, D. H., and Ellsworth, R. M. (Dept. Ophthalmol., Edward S. Harkness Eye Inst., New York, N. Y.). Bull. N. Y. Acad. Med. 56:221, 1980.

The diagnosis of retinoblastoma is usually made on clinical grounds from the appearance of the lesion in the eye. However there are additional tests that are useful and should always be used if the diagnosis is in question. Skull x-rays may reveal intraocular calcifications which have been demonstrated in ap-

proximately 75% of cases with retinoblastoma. Computerized axial tomography (CT scan) is noninvasive, painless and gives information about the eye, the orbit, the optic nerve and the brain. Small tumors can be detected in both eyes. Ultrasound is useful in ruling out small eyes with persistent hyperplastic primary vitreous and can confirm the presence of multiple tumors.

Further, there are chemical tests that are of use, particularly lactic acid dehydrogenase which is found in increased concentration in the aqueous humor of eyes with retinoblastoma. Finally a new immunoassay has been developed to rule out toxocariasis of the eye. This is the enzyme-linked immunosorbent assay (ELISA). At the moment this test is only available from the Communicable Disease Center in Atlanta, Georgia. (6 figures, 23 references)—David Shoch

Cancer

THE ROLE OF RADIATION THERAPY IN THE MANAGEMENT OF OCULAR RETICULUM CELL SARCOMA. Margolis, L., Fraser, R., Lichter, A., and Char, D. H. (Univ. California, San Francisco, Calif., and Johns Hopkins Hosp., Baltimore, Md.). Cancer 45:688, 1980.

This report concerns nine patients with ocular lymphomas six of whom had visual symptoms as the first manifestation of their disease. Eight of the nine patients developed intracranial lymphoma at some time during the course of the disease. Despite lymphoma work-up including bone marrow biopsies and lymphangiogram, only one patient was found to have documented systemic involvement. The diagnosis of ocular lymphoma was based on pathologic material from the eye in five cases or from central nervous system biopsy in four patients in association with tumor cell infiltrates in the retina and vitreous clouding. Radiation therapy to the eyes improved vision in ten of 13 eyes treated in eight patients. The usual dose was in the range of 3500 to 4500 rads given over four to five weeks. In addition, seven patients received central nervous system irradiation. This study confirms the literature statement that there is a frequent association of ocular lymphoma with intracranial lymphoma and that there is rare systemic dissemination. This disease process has previously been referred to as ocular reticulum cell sarcoma. (3 tables, 20 references)—Authors' abstract

Investigative Ophthalmology and Visual Science

SIMULTANEOUS STEREOSCOPIC FUNDUS CAMERA INCORPORATING A SINGLE OPTICAL AXIS. Donaldson, D. D., Prescott, R., and Kennedy, S. (Howe Lab., Boston, Mass.). Invest. Ophthalmol. Visual Sci. 19:289, 1980.

The method of stereoscopic fundus photography, where both photographs are taken simultaneously, yields the most consistent and reproducible record of depths in the fundus. However, all systems devised to date have had undesirable photographic aberrations. For the most part, this has resulted because the optical axis of each image is not the same as that of the objective lens. A single-axis system has now been devised to minimize distortions in the stereoscopic photographs, which therefore makes stereophotogrammetry relatively simple. This design also has other advantages such as simplified alignment procedures, the use of Barlow lenses for multiple magnifications, and various paired apertures for obtaining photographs especially adapted to unusual situations. (7 figures, 12 references)—Authors' abstract

Journal of the American Medical Association

POSTPARTUM *Candida* ENDOPHTHALMITIS. Cantrill, H. L., Rodman, W. P., Ransay, R. C., and Knobloch, W. H. (Dept. Ophthalmol., Univ. Minnesota, Minneapolis, Minn.). J.A.M.A. 243:1163, 1980.

Endogenous *Candida* endophthalmitis has been reported after the use of intravenous catheters and particularly following the use of immunosuppressive drugs. Generally there is an impaired host defense as might occur with malignant neoplasms, cirrhosis, and diabetes; another possible cause is here described and this is in the postpartum patient. Two such patients are described, one of whom had a known history of *Candida* vaginitis. In the second patient no *Candida* could be cultured from the vagina, blood or urine but cultures of the vitreous aspirate grew abundant *C. albicans*. *Candida* endophthalmitis must be included in the differential diagnosis in the postpartum patient with visual symptoms. (7 figures, 13 references)—David Shoch

Klinische Monatsbläetter für Augenheilkunde

JUVENILE MACULA DEGENERATION. (German) François, J. (Univ. Augenklinik Gent/Belgien). Klin. Monatsbl. Augenheilkd. 175:715, 1979.

Juvenile macular degeneration can be broken down into various subgroups and the diagnosis made easier by considering these groupings. 1) Primarily neuro-epithelial disease: Stargardt's disease; dominant juvenile degeneration; central pigmentary retinopathy; progressive cone dystrophy and cystoid macular edema. 2) Primarily pigment epithelium: vitelliform degeneration of the macula, fundus flavimaculatus, Sjögren's pigmentary dystrophy; butterfly shaped dystrophy and grouped pigmentation of the macular region. 3) Bruch's membrane: hyaline dystrophy or drusen of the lamina vitrea and dominant progressive foveal dystrophy. 4) Chorcid: central areolar atrophy of the choroid and Sorsby's pseudo-inflammatory dystrophy. All of these macular heredo-degenerations are bilateral and symmetrical. Proper diagnosis depends on ophthalmoscopy, fluorescein angiography, ERG, EOG and a genetic study of the affected families. (26 figures, 54 references)—David Shoch

DETACHABLE SUPPLEMENTARY LENSES ON THE SIDEPIECE OF CATARACT SPECTACLES FOR BETTER UTILIZATION OF THE TEMPORAL VISUAL FIELD. (German) Jaeger, W. (Univ. Augenklinik, Heidelberg). Klin. Monatsbl. Augenheilkd. 176:21, 1980.

Ordinary cataract glasses have many defects but two specific ones are a annular scotoma caused by the prismatic action of the edges of the strong convex lens and a temporal island of poor vision corresponding to those rays of light which enter the eye temporal to the spectacles. The author has devised accessory cataract lenses which fit onto the temples and allow correction of this temporal field. He states that this is particularly useful in driving but that it can be confusing for general use. Therefore, the lens has been designed with small magnets on them so that they can be easily removed. The aphake can wear his cataract glasses without the lateral lenses normally and afix them to the sidepiece of the spectacles when he drives. (7 figures, 2 references)—David Shoch

Mayo Clinic Proceedings

RESOLUTION OF VENOUS STASIS RETINOPATHY AFTER CAROTID ARTERY BYPASS SURGERY. Kearns, T. P., Younge, B. R., and Piepgras, D. G. (Dept. Ophthalmol., Mayo Clinic, Rochester, Minn.). Mayo Clin. Proc. 55:342, 1980.

Venous stasis retinopathy was a term first used to describe the retinal changes associated with severe stenosis or occlusion of the internal carotid artery. Generally this retinopathy has been seen to resolve after endarterectomy. Another mechanism that apparently will resolve venous stasis retinopathy is a bypass of the carotid artery. The authors report a patient with carotid occlusive disease in whom the anterior branch of the right superficial temporal artery was anastomosed to a cortical branch of the right middle cerebral artery. Within a week there was significant resolution of the venous stasis

retinopathy. This resolution reflects the increased perfusion to the retina and to the cerebral hemisphere. (5 figures, 5 references)—David Shoch

Ophthalmology

ACTIVITY AND DIABETIC VITREOUS HEMORRHAGES. Anderson, B. (Retina Ser., Duke Eye Ctr., Durham, N. C.). Ophthalmol. 87:173, 1980.

To ascertain the relationship between activity and vitreous hemorrhage, the author questioned 72 consecutive diabetic patients presenting with 95 episodes of vitreous hemorrhage. The activity most commonly associated with symptoms of hemorrhage was sleep (36%) followed closely by sitting or lying (26%). Only one of six hemorrhages was associated with activity that could be considered more strenuous than walking. Limiting the activity of diabetic patients does not seem an effective method of preventing vitreous hemorrhage. (2 tables, 1 reference)—Author's abstract

AUTOSOMAL DOMINANT OPTIC ATROPHY: A SPECTRUM OF DISABILITY. Hoyt, C. S. (Dept. Ophthalmol., Univ. California, San Francisco, Calif.). Ophthalmol. 87:245, 1980.

Autosomal dominant optic atrophy is an abiotrophy with an insidious onset in the first decade of life. The clinical features of 31 individuals in six pedigrees are detailed in this study. These data suggest that there is considerable intrafamilial and interfamilial expression of dysfunction. Moreover, asymmetry of the visual loss is not unusual. An unexpected result of this study is the previously unreported frequent association of a neural hearing loss with this disorder. (1 table, 31 references)—Author's abstract

CONJUNCTIVAL BIOPSY AS AN AID IN THE EVALUATION OF THE PATIENT WITH SUSPECTED SARCOIDOSIS. Nichols, C. W., Eagle, R. C., Yanoff, M., and Menocal, N. G. (Dept. Ophthalmol., Univ. Pennsylvania, Philadelphia, Pa.). Ophthalmol. 87:287, 1980.

Conjunctival biopsy is an underused but simple technique in the evaluation of the patient with sarcoidosis and occasionally other systemic diseases. In 55% of patients with biopsy-proven sarcoidosis from other sites, a blind conjunctival biopsy was positive. Bilateral conjunctival biopsies and the examination of multiple sections of each biopsy were essential to obtain this high yield. There was no relationship between an anterior uveitis and a positive conjunctival biopsy. (1 table, 6 references)—Authors' abstract

Radiology

MENINGIOMATOUS CHANGES IN THE OPTIC CANAL: A POLYTOMOGRAPHIC STUDY. Strother, C. M., Hoyt, W. F., Appen, R. E., and Newton, T. H. (Depts. Radiol., Neurol., and Ophthalmol., Univ. Wisconsin Clinical Science Ctr., Madison, Wisc.). Radiology 135:109, 1980.

Eleven patients who had unilateral insidious compression of the optic nerve but exhibited no detectable abnormality on plain skull radiographs or non-tomographic views of the optic canal were studied. Paracanalicular meningioma was proved surgically in ten. Complex-motion tomography demonstrated characteristic alterations in the bone forming the optic canal in all cases. These changes consisted of canal enlargement, changes in contour, and an atypical radiographic appearance of the cortical bone, seen as (a) diffuse, spongy, non-homogeneous thickening of the canal margins; (b) multiple tiny saw-tooth-like exostoses projecting into the canal lumen; or (c) short, well-circumscribed areas of smooth bone thickening. Other techniques such as angiography and pneumoencephalography were not as helpful in the diagnosis as complex-motion tomography. (5 figures, 14 references)—Authors' abstract

Transactions of the American Ophthalmological Society

DETERMINATION OF 8-METHOXYPSORALEN IN SERUM, AQUEOUS, AND LENS:

RELATION TO LONG-WAVE ULTRAVIOLET PHOTOTOXICITY IN EXPERIMENTAL AND CLINICAL PHOTOCHEMOTHERAPY. (Thesis) Glew, W. B. (Washington Hosp. Ctr., Washington D. C.). Trans. Am. Ophthalmol. Soc. 77:464, 1979.

In recent years a most successful therapy for psoriasis has been developed; this is a combination of oral administration of 8-methoxypsoralen and the use of long-wave ultraviolet light to excite the psoralen molecules in the skin. Apparently these activated psoralen molecules interfere with the normal DNA in the skin cells and thus inhibit cell replication. Clearance of psoriatic skin lesions results from the reduction of epidermal cellular hyperreplication. However, it has been postulated that exactly the same mechanism can occur in the lens and serve as a potential mechanism for cataractogenesis. In order to determine this, levels of psoralen were determined in serum aqueous and lens and it was found that an enormous ultraviolet dosage was required to produce lens opacities in rabbits. There is a wide margin of safety present. The author also followed 100 eyes of 50 patients undergoing therapy for psoriasis for two to three years. There was some progression of cataracts already present in two patients but these seem to be unrelated to the therapy. However, the long-term actinic effects on the lens may still occur if there are residual levels of photoactive 8-methoxypsoralen in the lens. To prevent this excitation sunglasses that block all ultraviolet must be worn for the duration of the photosensitized state on each day of therapy. Prolonged levels of photoactive drug can be detected by serum bioassay. (15 figures, 6 tables, 93 references)—David Shoch

THE NATURAL COURSE OF ACTIVE CHOROIDAL LESIONS IN THE PRESUMED OCULAR HISTOPLASMOSIS SYNDROME. (Thesis) Gutman, F. A. (Cleveland Clinic, Cleveland, Ohio). Trans Am. Ophthalmol. Soc. 77:515, 1979.

Eighty-two patients and 106 eyes with the presumed ocular histoplasmosis syndrome have been followed for one year or longer. Untreated active choroidal lesions were divided into groupings based on the distance from the center of the capillary free zone and the relationship to the border of the capillary free zone. Twenty-three percent of untreated active choroidal lesions that were located inside the capillary free zone achieved 20/20 (6/6) to 20/40 (6/12) vision. Sixty-three percent of untreated active choroidal lesions that were located outside of the capillary free zone achieved a vision of 20/20 (6/6) to 20/40 (6/12). The active choroidal lesions with the best visual prognosis were 0.25 to 0.5 disk diameters, demonstrated no growth, and had little or no associated choroidal bleeding. Activation of choroidal lesions in the fellow eye at the site of preexisting atrophic scars was documented in 21% of the eyes. One fellow eye developed a de novo active choroidal lesion. Eighteen percent of the cases had structurally congested nerve heads characterized by slight elevation of the disk and no physiologic depression; 3% of the eyes had drusen of the optic nerve. A relationship may exist between peripapillary scarring in the presumed ocular histoplasmosis syndrome and nerve head changes (10 figures, 7 tables, 50 references)—Author's abstract.

AN EXPERIMENTAL APPROACH TO THE STUDY OF INTRAOCULAR *Toxocara canis*. (Thesis) Luxenberg, M. N. (Med. College of Georgia, Augusta, Ga.). Trans. Am. Ophthalmol. Soc. 77:542, 1979.

Six owl monkeys were infected either by nasogastric tube using embryonated *T. canis* eggs or by carotid or intravitreal injection of second stage larvae. Only minimal or no intraocular changes were seen after systemic infection but significant abnormalities such as retinal hemorrhages and venous dilation were noted after intravitreal infection. Motile larvae were observed in the lenses of three eyes and in the vitreous of five eyes and, probably a sixth, after intravitreal injection. The intensity and timing of the intraocular reaction seemed to correlate with the infecting dose and apparent disappearance of larvae from the eye. Pathologic confirmation of larvae in the lens was obtained in one eye. A marked inflammatory reaction occurred in eyes receiving intraocular infection but none was seen in eyes with only systemic infection. Various laboratory and serologic studies were performed, including the enzyme-linked im-

munosorbent assay (ELISA) test, which were used to evaluate systemic as well as intraocular responses to infection with *T. canis*. The two monkeys infected by nasogastric tube gave a positive ELISA response in the serum but intraocular fluids gave a negative response in all monkeys including those infected systemically and/or intraocularly. (14 figures, 3 tables, 90 references)—David Shoch

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EDITED BY THOMAS CHALKLEY, M.D.

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AMERICAN ACADEMY OF OPHTHALMOLOGY: ANNUAL MEETING SITES

The American Academy of Ophthalmology has scheduled annual meetings as follows:

1980	Chicago	Nov. 2–7
1981	Atlanta	Nov. 1–5
1982	San Francisco	Oct. 31–Nov. 6
1983	Chicago	Oct. 30–Nov. 3
1984	Atlanta	Nov. 11–15
1985	San Francisco	Sept. 30–Oct 4
1986	New Orleans	

ALBANY MEDICAL COLLEGE: SYMPOSIUM PRACTICAL APPROACH TO COMMON ORBITAL DISEASES

The Albany Medical College and Eastern New York Ophthalmology Society will hold a symposium Practical Approach to Common Orbital Diseases in Albany New York, Oct. 9, 1980. For further information, write Gregory B. Krohel, M.D., Dept. of Ophthalmology, Albany Medical College, Albany, NY 12208

STRABISMUS SYMPOSIUM

A Strabismus Symposium will be held in Amsterdam, Sept. 3 and 4, 1981. For further information, write Wilhelmina Gasthuis, Eye Dept., Eerste Helmersstraat 104 1054 EG Amsterdam, The Netherlands.

THE EYE FOUNDATION: THIRD ANNUAL INTERNATIONAL CATARACT SURGERY SYMPOSIUM

The Eye Foundation will present its Third Annual International Cataract Surgery Symposium Feb. 16–18, 1981, in Scottsdale, Arizona. For further information, write Ronald W. Barnet, M.D., The Eye Foundation, 421 N. 18th Street, Suite 105, Phoenix, AZ 85006.

QUEEN'S UNIVERSITY: CLINICAL DAY IN OCULAR MOTILITY

The Department of Ophthalmology of Queen's University will sponsor a Clinical Day in Ocular Motility, Oct. 31, 1980. For further information, write Dr. R. M. H. Pinkerton, Dept. of Ophthalmology, Queen's University, Kingston, Ontario Canada K7L3N6.

INTERNATIONAL EYE CONGRESS

The 1st Annual International Eye Congress, sponsored by the Metropolitan Eye & Ear Hospital, will be held in Atlanta, Georgia, April 24–26, 1981. The fee for practicing ophthalomogists will be $300 and $75 for residents and fellows. For further information, write Ms. Linda Albright, Congress Coordinator, c/o Metropolitan Eye & Ear Hospital, 3223 Howell Mill Road NW, Atlanta, GA 30327.

MIAMI OPHTHALMOLOGICAL SOCIETY: THIRD ANNUAL WINTER SEMINAR

The Third Annual Winter Seminar of the Miami Ophthalmological Society will be held in Aspen, Colorado, Jan. 11–18, 1981. For further information, write Dr. David J. Singer, 1160 Kane Concourse, Miami Beach FL 33154.

THE NEW JERSEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY: ANNUAL FALL SCIENTIFIC MEETING

The New Jersey Academy of Ophthalmology and Otolaryngology will hold its Annual Fall Scientific Meeting on Oct. 22, 1980, in West Orange, New Jersey. For further information, write Marshall S. Klein, New Jersey Academy of Ophthalmology and Otolaryngology, 15 South 9th Street, Newark, NJ 07107.

MERCY–BAPTIST EYE FOUNDATION: SEMINAR ON NEURO–OPHTHALMOLOGY FOR CLINICAL OPHTHALMOLOGISTS

Mercy Baptist Eye Foundation will sponsor a seminar on neuro–ophthalmology for clinical ophthalmologists held Oct. 5, 1980, in St. Louis, Missouri. For further information, write Eye Seminars, 621 S. New Ballas Road, Suite 343, St. Louis, MO 63141.

JACKSONVILLE SOCIETY OF OPHTHALMOLOGY: FALL CONFERENCE

The Jacksonville Society of Ophthalmology will hold its annual fall conference Sept. 27 and 28, 1980. Registration fee is $175. For further information, write Jacksonville Health Education Program, 580 W. 8th St., Jacksonville, FL 32209.

Second International Congress on Cataract Surgery

The Second International Congress on Cataract Surgery will be held on June 17–20, 1981, in Florence, Italy. For further information, write Professor Ivan Esente, M.D., Scientific Secretariat, Corso Italia, 2, Florence, Italy.

University of South Florida: Oculoplastic–Orbital Update

The Department of Ophthalmology at the University of South Florida will sponsor a course on Orbital and Oculoplastic Disorders on March 5–7, 1981, in Tampa, Florida. For more information, write James A. Rush, M.D., Department of Ophthalmology, University of South Florida College of Medicine, 12901 N. 30th Street, Tampa, FL 33612.

Long Island Jewish–Hillside Medical Center: Clinical Day on Corneal Disease

A clinical day on corneal disease will be sponsored by the Department of Ophthalmology, Long Island Jewish–Hillside Medical Center and Health Sciences Center, Dec. 3, 1980, in New Hyde Park, New York. The tuition is $60. For further information, write Continuing Education Coordiantor, Long Island Jewish–Hillside Medical Center, New Hyde Park, NY 11042.

Joint Commission on Allied Health Personnel in Ophthalmology: New Officers

The new officers of the Joint Commission on Allied Health Personnel are: President–Peter Y. Evans, Washington, D. C.; Vice–President, Bernard R. Blais, Washington, D. C.; Secretary, Robert L. Stamper, San Francisco; Treasurer, R. Linsy Farris, New York; Executive Vice–President, Robert Hugh Monahan, St. Paul, Minnesota; Immediate Past President, Thomas J. Kirby, Rochester, Minnesota. This is the federated agency representing the American Academy of Ophthalmology, the American Association of Ophthalmology, the American Medical Association, the American Ophthalmological Society, the Association of University Professors of Ophthalmology, the American Orthoptic Council, the Canadian Ophthalmological Society, the Contact Lens Association of Ophthalmologists, and the Society of Military Ophthalmologists.

Volunteers Needed

Over 150,000 Cambodian refugees are living in UN-sponsored camps inside Thailand. Ophthalmologists are urgently needed to help care for them. Transportation costs, living expenses, and surgical facilities are provided by the American Refugee Committee. If you are interested in volunteering your services for six weeks or longer, please write Karen Johnson, American Refugee Committee, 310 4th Ave. South, Suite 410, Minneapolis, MN 55415; telephone (612) 332-5365.

Personals

Alson E. Braley

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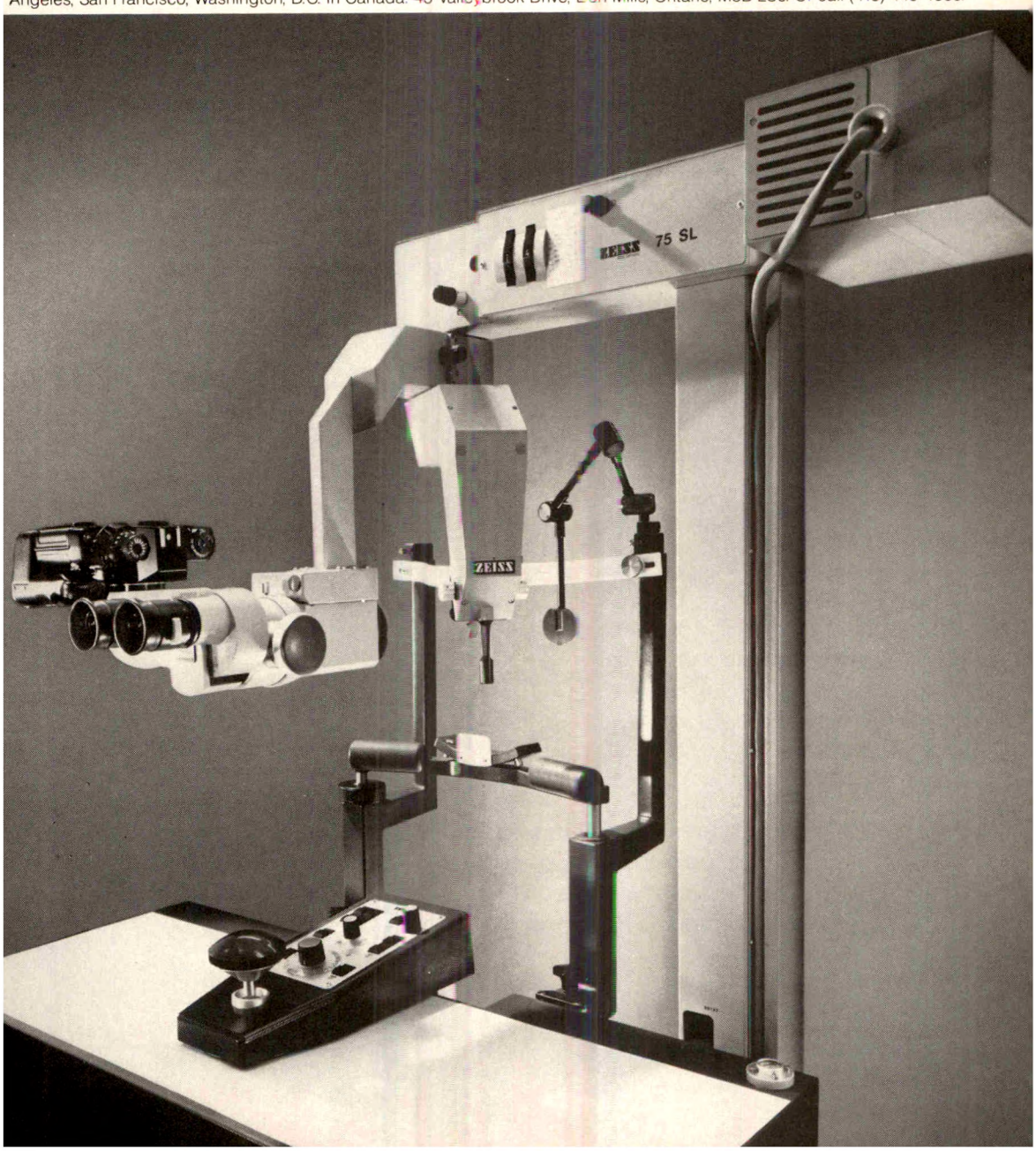
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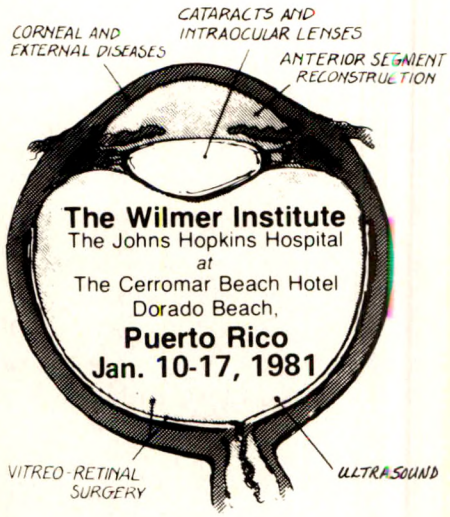
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Shearing and Sinskey Lenses



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Dates:
Friday, September 19, 1980
Friday, November 21, 1980
7:30 A.M. – 4:00 P.M.

Tuition:
$300.00
25% cancellation fee

Credit:
8 Hours, Category 1

Course Director:
Robert M. Sinskey, M.D.

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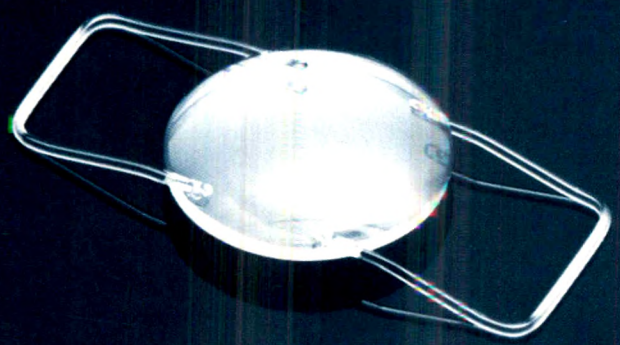
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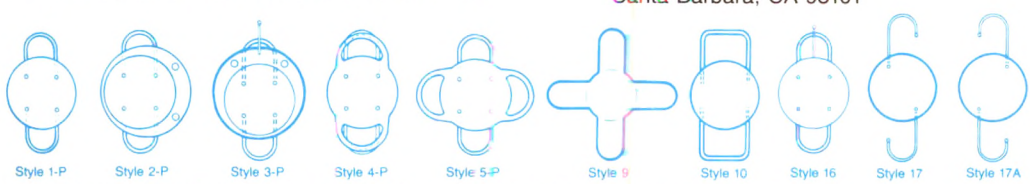
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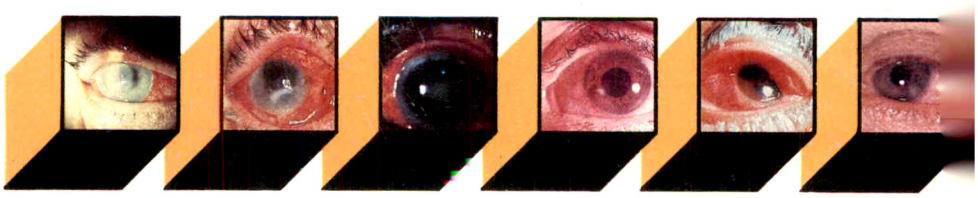
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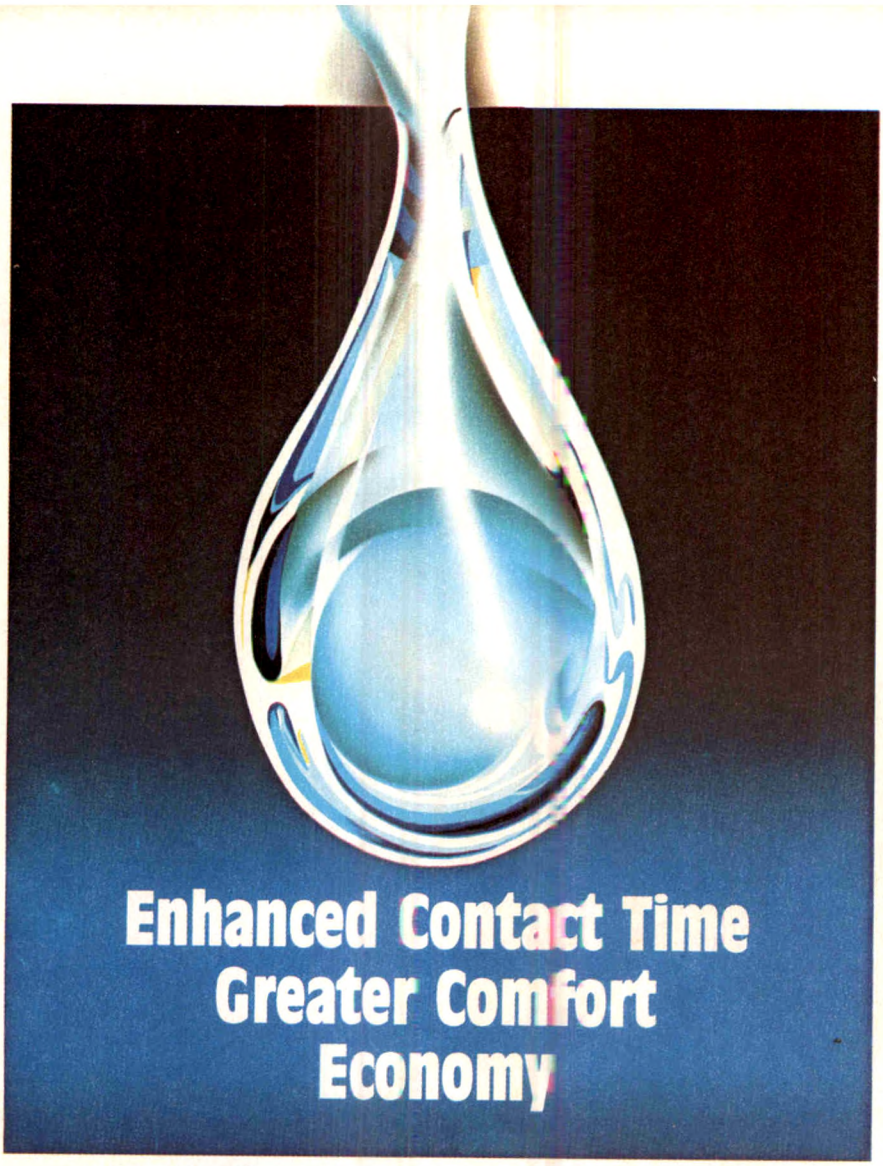
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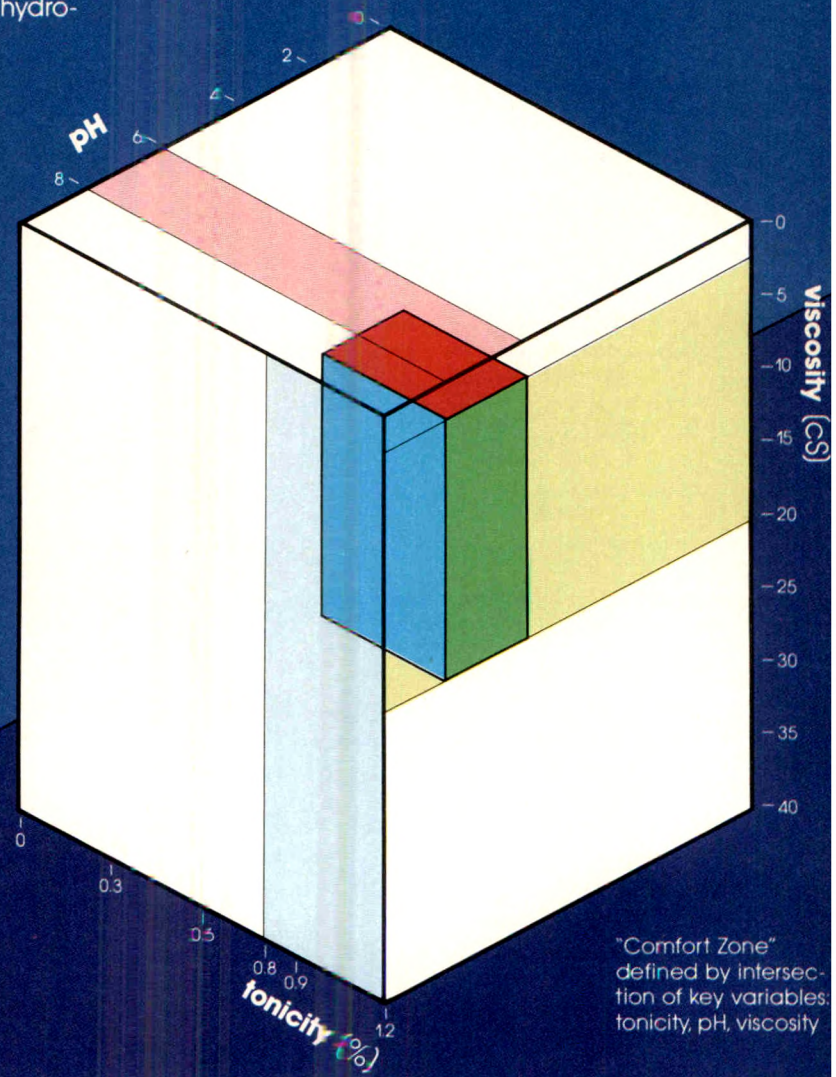
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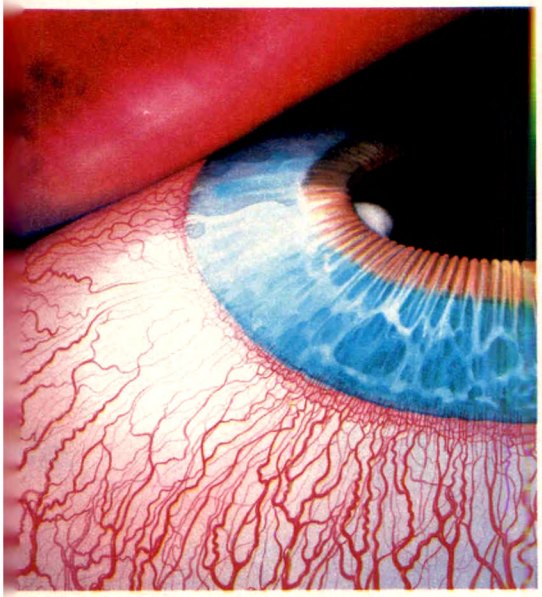
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The no.1 sulfa/steroid combination:

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Treats the cause and complaint of conjunctivitis.*

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Description: Suspension contains: sulfacetamide sodium 10.0%; prednisolone acetate* (microfine suspension) 0.2%; phenylephrine HCl 0.12%; Liquifilm (polyvinyl alcohol) 1.4%; with: phenylmercuric nitrate (0.004%); antipyrine (0.1%); polysorbate 80; edetate disodium, sodium phosphate dibasic, anhydrous; sodium phosphate monobasic; sodium thiosulfate; hydrochloric acid to adjust pH to 6.8; and purified water.
Ointment contains: sulfacetamide sodium 10.0%; prednisolone acetate* 0.2%; with: phenylmercuric acetate (0.0008%); mineral oil; white petrolatum; and nonionic lanolin derivatives. Licensed under patent 3,134,718.

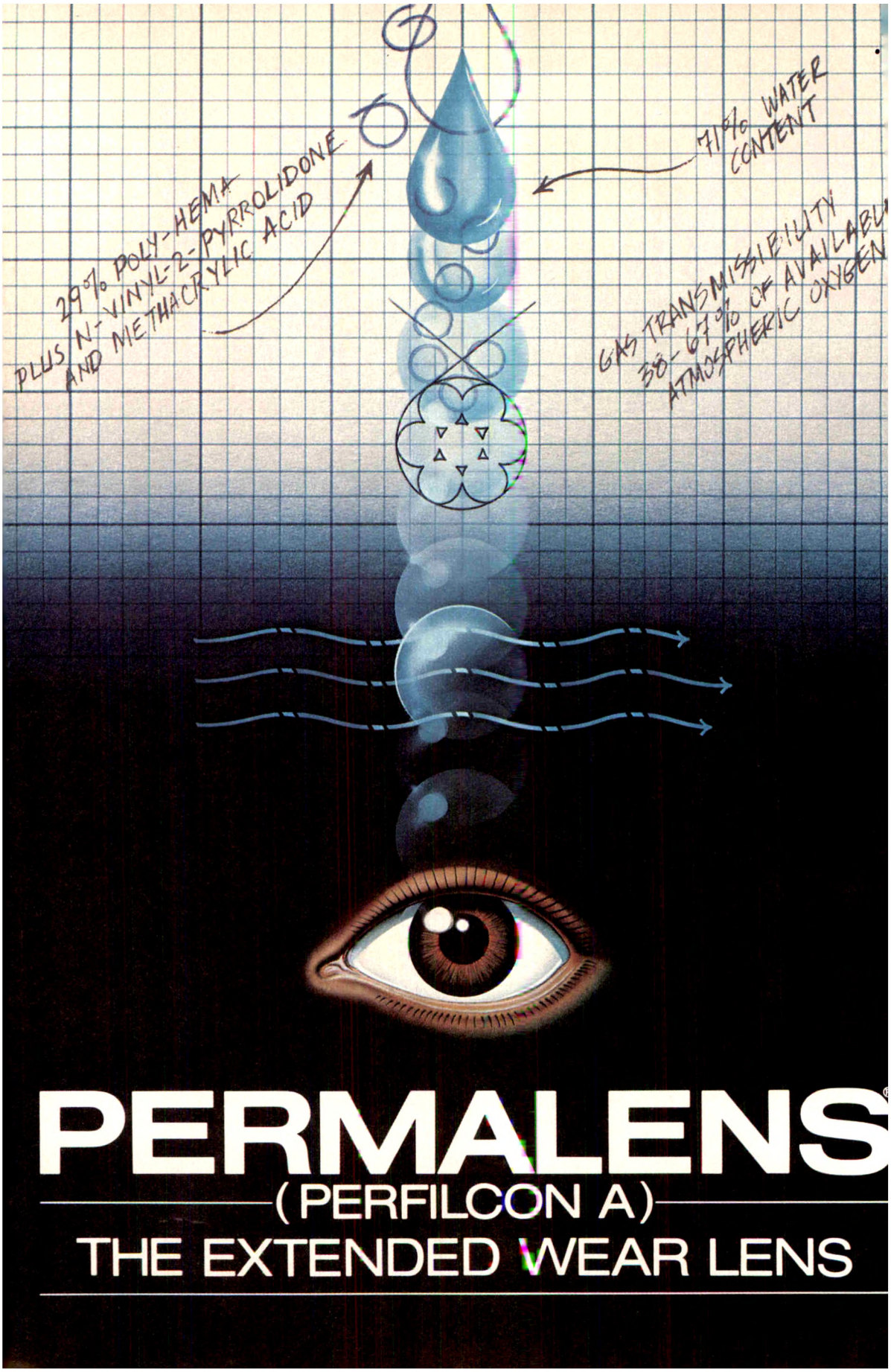
Indications: Based on a review of this drug by the National Academy of Sciences-National Research Council and/or other information, FDA has classified the indications as follows: "Possibly" effective: Nonpurulent blepharitis and blepharoconjunctivitis (seborrheal, staphylococcal, allergic); nonpurulent conjunctivitis (allergic and bacterial). Final classification of the less-than-effective indications requires further investigation.

Contraindications: Acute herpes simplex (dendritic keratitis) purulent untreated infections, vaccinia, varicella and most other viral diseases of the cornea and conjunctiva, ocular tuberculosis and fungal diseases of the eyes. **Warnings:** 1) In diseases due to microorganisms, infection may be masked, enhanced or activated by the steroid. 2) Extended use may cause increased intraocular pressure in susceptible individuals. It is advisable that the intraocular pressure be checked frequently. 3) In those diseases causing thinning of the cornea, perforation has been known to have occurred with the use of topical steroids. 4) Use with caution in patients with known or suspected sensitivity to sulfonamides—if sensitivity or other untoward reactions occur, discontinue medication. 5) BLEPHAMIDE ophthalmic suspension should be used with caution in the presence of narrow-angle glaucoma. 6) Reports in the literature indicate that posterior subcapsular lenticular opacities have been reported to occur after heavy or protracted use of topical ophthalmic corticosteroids.

Leibowitz HM, Kupferman A. Bioavailability and therapeutic effectiveness of topically administered corticosteroids. Trans Am Acad Ophthalmol Otolaryngol 1975;79(1): OP78-OP88.

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Permalens® Contact Lenses: not just extended wear of a lens, but a lens **designed specifically for extended wear.**

That's why Permalens® Contact Lenses are rapidly changing the field of vision correction. Now your aphakic patients can again experience near-normal vision from the moment they wake up in the morning. Without the bother of daily insertion and removal.

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This response is partly due to the Permalens® material, perfilcon A, which combines high water content, high oxygen permeability, and dimensional stability. These properties were designed into the lens to facilitate oxygen transfer directly through the lens, so the cornea can receive an oxygen supply adequate to maintain itself in a healthy state.

These properties also make Permalens® Contact Lenses more supple, and therefore comfortable on the patient's eyes. Patients have reported, in fact, they're the next best thing to wearing no lens at all.

Easy to fit, Permalens® Contact Lenses are available in a range of powers from +11.00 to +17.00 diopters. And with just four parameters, the lens fits most corneal sizes and curvatures.

Maintenance, too, is uncomplicated. Since Permalens® Contact Lenses usually need only be removed once a month, cleaning and disinfection are simplified. The preferred regimen is Preflex®/Flexsol®/Normol.® (Thermal regimens can also be used.) For re-hydration during lens wear, Clērz® or Adapettes® eye drops are available.

Patient comfort and convenience . . . clinically proven safety . . . easy fitting . . . simple maintenance. Good reasons why thousands of patients and practitioners alike have accepted Permalens® as **the** extended-wear lens.

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(perfilcon A)

Hydrophilic Contact Lens
For Extended Wear
Post-Cataract (Aphakic) Use

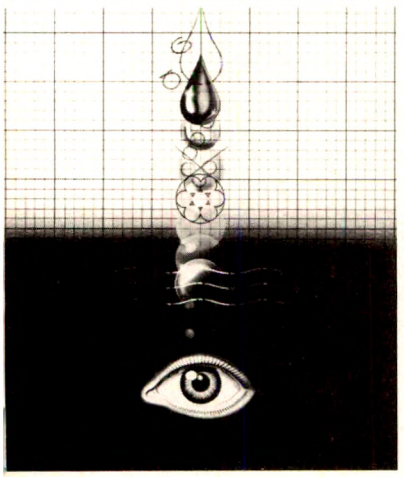
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Permalens®
(perfilcon A)

Hydrophilic Contact Lens For Extended Wear Post-Cataract (Aphakic) Use

DESCRIPTION

The **Permalens**® (perfilcon A) Hydrophilic Contact Lens is a hemispherical shell which covers the cornea and may cover a portion of the adjacent sclera. The lens material, perfilcon A, is a terpolymer of 2-hydroxyethyl methacrylate, N-vinyl-2-pyrrolidone, and methacrylic acid, with ethyleneglycol dimethacrylate as a crosslinking agent. The hydrated lens consists of 29% perfilcon A and 71% water by weight. The gas permeability of **Permalens**® Hydrophilic Contact Lenses, using the Fatt method,[1] has been determined to be: 42.0×10^{-11} $cm^2 \cdot ml\ O_2/sec \cdot ml \cdot mm\ Hg$ at 36°C.

ACTIONS

When placed on the human cornea, the hydrated **Permalens**® Hydrophilic Contact Lens acts as a refracting medium to compensate spherical ametropias.

INDICATIONS AND USAGE

Aphakia

Permalens® (perfilcon A) Hydrophilic Contact Lenses are indicated for extended-wear aphakic vision correction in persons with non-diseased eyes.

Note: Supplemental spectacles may be required, in some conditions, to provide near vision or to compensate for uncorrected refractive astigmatism.

CONTRAINDICATIONS

Permalens® Hydrophilic Contact Lenses are contraindicated by the presence of any of the following conditions:

1. Acute and subacute inflammations of the anterior segment of the eye.
2. Any eye disease which affects the cornea or conjunctiva.
3. Insufficiency of lacrimal secretion.
4. Corneal hypoesthesia.
5. Any systemic disease which may affect the eye or be exaggerated by wearing contact lenses.

WARNINGS

Abrasions and Infections

If a lens becomes less comfortable than it was when it was first placed on the wearer's cornea, the lens should be removed and inspected for the presence of a foreign body, lens coating, deposits, or lens damage. If examination of the patient's eye reveals any eye abrasion, ulceration, irritation or infection, or any other abnormal eye condition is observed concurrently with lens wear, the lens should be removed immediately and a physician consulted.

Patients should not be fitted with Permalens® (perfilcon A) Hydrophilic Contact Lenses during the post-operative period until, in the opinion of the surgeon, the eye has healed completely.

Medications and Eye Drops

Permalens® Hydrophilic Contact Lenses must be stored in an appropriate solution when off the eyes. The solution used is dependent on the system selected for disinfection. When the lenses are disinfected with the thermal disinfection system, they may be stored in PERMASOL™ Rinsing and Storage Solution (sterile buffered balanced salt solution preserved with edetate disodium 0.1% and thimerosal 0.001%) or BOILnSOAK® Sterile Saline Solution (sterile buffered isotonic solution containing boric acid, sodium borate, sodium chlorice 0.7%, preserved with Thimerosal (Lilly) 0.001% and edetate disodium 0.1%). When lenses are disinfected with the chemical disinfection system, they may be stored in FLEXSOL® Sterile Disinfection and Storage Solution (sterile buffered isotonic solution of sodium chloride, sodium borate, boric acid, polyvinylpyrrolidone, polyoxyethylene and polyoxypropylene, preserved with Thimerosal (Lilly) 0.001%, chlorhexidine 0.005% and edetate disodium 0.1%).

Only the above solutions and the following may be used with **Permalens**® Hydrophilic Contact Lenses:

1. PREFLEX® Sterile Cleaning Solution (sterile solution of a nonionic cleaner preserved with Thimerosal (Lilly) 0.004% and edetate disodium 0.2%).
2. NORMOL® Sterile Rinsing Solution (sterile buffered isotonic aqueous solution of sodium chloride, sodium borate and boric acid, preserved with Thimerosal (Lilly) 0.001%, edetate disodium 0.1% and chlorhexidine 0.005%).
3. CLERZ® Lubricating and Rewetting Eye Drops (sterile solution preserved with edetate disodium 0.1% and Thimerosal (Lilly) 0.001%).
4. ADAPETTES® Sterile Lubricating Solution (buffered isotonic aqueous solution containing Adsorbobase® (polyvinylpyrrolidone with other water-soluble polymers), Thimerosal (Lilly) 0.004%, and edetate disodium 0.1%).

No conventional (hard) contact lens solutions should be used. Only those solutions specifically recommended by your eye-care practitioner should be used during wear.

WEARING RESTRICTIONS

Permalens® (perfilcon A) Hydrophilic Contact Lenses should not be worn while swimming or in the presence of noxious or irritating vapors.

Lens-Care Regimen

Patients must adhere to the recommended lens-care regimen of **Permalens**® Hydrophilic Contact Lenses. Failure to follow this procedure may result in the development of serious ocular infections which might result in corneal ulcers.

PRECAUTIONS

Storage

Permalens® Hydrophilic Contact Lenses must be stored in the appropriate storage solution: PERMASOL™ Rinsing and Storage Solution, BOILnSOAK® Sterile Saline Solution, or FLEXSOL® Sterile Disinfection and Storage Solution, depending on disinfecting methods used. If left exposed to air, the lenses will dehydrate, become brittle and break readily. If a lens dehydrates, it should be soaked in one of the following until it returns to a hydrated (soft) pliable state:

1. PERMASOL™ Rinsing and Storage Solution
2. BOILnSOAK® Sterile Saline Solution
3. FLEXSOL® Sterile Disinfection and Storage Solution

PERMA-case™ Contact Lens Storage and Carrying Case should be used for storing lenses.

Hygiene

Hands must be washed and rinsed thoroughly and dried before handling the lenses. Cosmetics, lotions, soaps and creams must not come in contact with the lenses since eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until the spray has settled.

Cleaning and Disinfecting

Prior to wearing, **Permalens**® (perfilcon A) Hydrophilic Contact Lenses must be both cleaned and disinfected.

Cleaning: Monthly removal for cleaning is recommended. However, at the discretion of the eye-care practitioner, the lenses may be removed at a frequency adjusted to the needs of each patient. Each time the lenses are removed from the wearer's eyes, both surfaces of the lenses must be cleaned using several drops of PREFLEX® Sterile Cleaning Solution. Between regular cleaning periods, CLERZ® Lubricating and Rewetting Eye Drops or ADAPETTES® Sterile Lubricating Solution may be used to maintain lens hydration while being worn.

Routine Thermal Disinfection Method: Permalens® Hydrophilic Contact Lenses may be effectively disinfected using a low-heat disinfecting unit approved for use with hydrophilic contact lenses. After cleaning with PREFLEX® Sterile Cleaning Solution, rinse lenses with PERMASOL™ Rinsing and Storage Solution or BOILnSOAK® Sterile Saline Solution, and disinfect in a RINCON® low-heat Thermal Disinfection Unit or other approved unit. The lens storage containers must be emptied and filled with fresh PERMASOL™ Rinsing and Storage Solution or BOILnSOAK® Sterile Saline Solution just prior to disinfecting the lenses.

Alternate Thermal Disinfection Method: If a thermal unit is not available, the following disinfecting system can be used temporarily. After cleaning the lenses according to instructions, fill each chamber of the PERMA-case™ with PERMASOL™ or BOILnSOAK®. Bring a pan of water to a rolling boil. Remove the pan from heat. Place the sealed carrying case into hot water and allow to cool. When the water is cool, lenses have been disinfected.

NOTE: USE OF THE RINCON® THERMAL DISINFECTION UNIT SHOULD BE RESUMED AS SOON AS POSSIBLE.

Chemical Disinfection Method: Disinfection with PREFLEX® Sterile Cleaning Solution, FLEXSOL® Sterile Disinfection and Storage Solution and NORMOL® Sterile Rinsing Solution has been shown to be an effective disinfection system. Following each removal, **Permalens**® Hydrophilic Contact Lenses must be cleaned with PREFLEX® Sterile Cleaning Solution and rinsed with NORMOL® Sterile Rinsing Solution. Each time the lens is stored, the PERMA-case™ Contact Lens Storage and Carrying Case must be emptied and refilled with fresh FLEXSOL® Sterile Disinfection and Storage Solution. Fresh FLEXSOL® Sterile Disinfection and Storage Solution must also be used when disinfecting a lens.

WARNING: FLEXSOL® STERILE DISINFECTION AND STORAGE SOLUTION SHOULD *NEVER* BE USED WITH

LENS EJECTION

Occasionally, a lens may become partially dehydrated during sleep. This may result in the lens being dislodged from the lid action or rubbing the eyelids. Dislodged or routinely rehydrated lenses should be thoroughly rehydrated in one of the following solutions:

1. PERMASOL™ Rinsing and Storage Solution
2. NORMOL® Sterile Rinsing Solution
3. BOILnSOAK® Sterile Saline Solution

After the lens returns to a soft, supple state it should be cleaned and disinfected prior to replacement on the eye.

ADVERSE REACTIONS

Serious corneal damage may result from wearing lenses which have been soaked in conventional hard contact lens solutions. Eye irritation may occur within a short time after putting on lenses stored in a solution of improper pH or tonicity. Removal of the lens will relieve the irritation.

Excessive tearing or redness, unusual eye secretions, reduced visual acuity, and photophobia are not normal; if these symptoms occur, the patient should be examined by an eye-care practitioner to determine the cause.

DOSAGE AND ADMINISTRATION

Fitting

Conventional methods of fitting contact lenses do not apply to **Permalens**® (perfilcon A) Hydrophilic Contact Lenses. For a detailed description of the fitting technique, refer to the **Permalens**® Hydrophilic Contact Lens Professional Fitting Guide, copies of which are available from Cooper Laboratories, Inc., N. Whisman Road, Mountain View, California 94043.

WEARING SCHEDULE

Permalens® Hydrophilic Contact Lenses are indicated for extended-wear (24-hour) use, without a gradual daily increase in wearing time required. Cleaning is recommended at monthly intervals. However, at the discretion of the eye-care practitioner, the lenses may be removed at a frequency adjusted to the needs of each patient.

Lens Care and Handling

Care must be taken on the initial visit to ensure that the patient is supplied with all necessary accessory products and fully understands all care and handling instructions for the lenses. Regular post-fitting visits are necessary to assure patient health and compliance with instructions.

HOW SUPPLIED

Each lens is supplied sterile in a sealed glass vial containing a buffered isotonic salt solution. The vial is marked with the dioptric power, base curve, diameter, lot number, and expiration date of the lens.

Accessories

Patient Care Kit	Cat. #80
Wearer's Manual	Cat. #80
PERMA-case™ Contact Lens Storage and Carrying Case	Cat. #80
FLEXSOL® Sterile Disinfection and Storage Solution*	Cat. #90
NORMOL® Sterile Rinsing Solution*	Cat. #90
PREFLEX® Sterile Cleaning Solution*	Cat. #90
CLERZ® Lubricating and Rewetting Eye Drops	Cat. #80
PERMASOL™ Rinsing and Storage Solution	Cat. #80
RINCON® Thermal Disinfection Unit**	Cat. #90
BOILnSOAK® Sterile Saline Solution*	Cat. #90
ADAPETTES® Sterile Lubricating Solution	Cat. #90

CAUTION: Federal Law Prohibits Dispensing Without Prescription

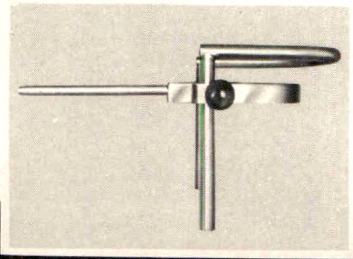
[1] Fatt I, Morris JA: A survey of gas-permeable contact lenses. *The Optician*: 27-36 (November 4, 1977).

*A product of Burton, Parsons & Company, Inc.
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6



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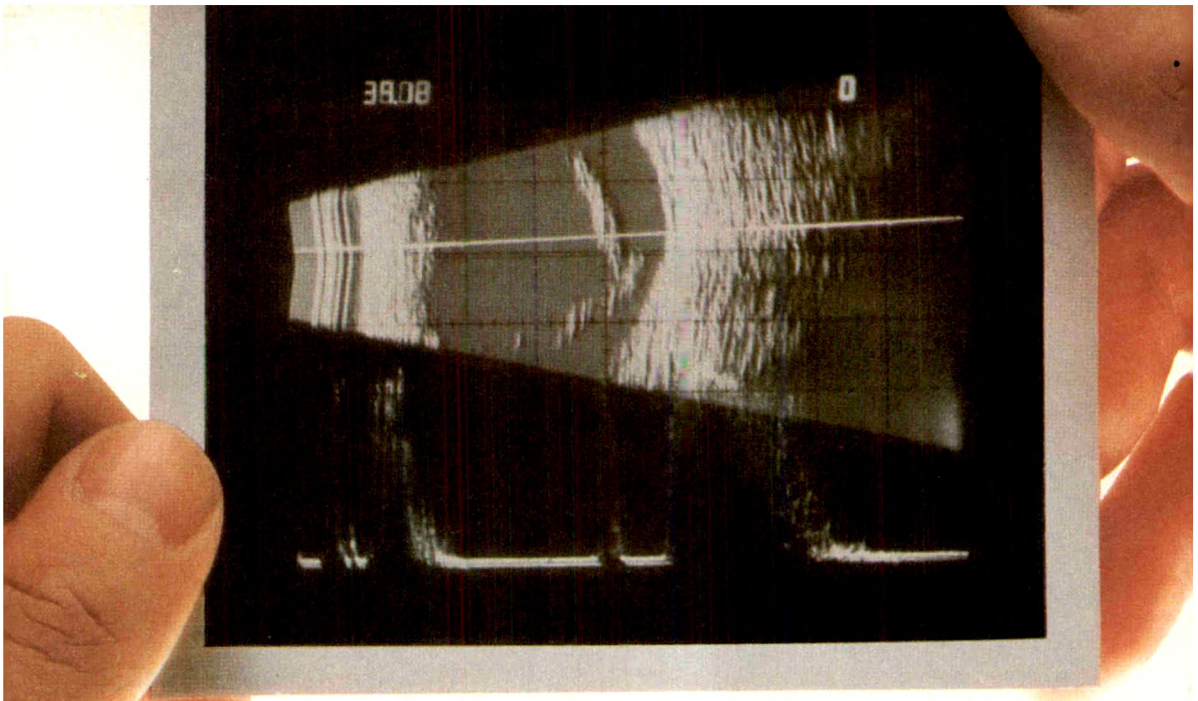
FOR MORE INFORMATION . . .on the Gibraltar head rest call or write Surgical Engineering Co., Inc., 4605 Linwood Drive, Birmingham, Alabama 35222, (205)595-4447. On ordering give the make and model number of your operating table. (Gibraltar combination wrist and head rest . . . $685.00 each.)



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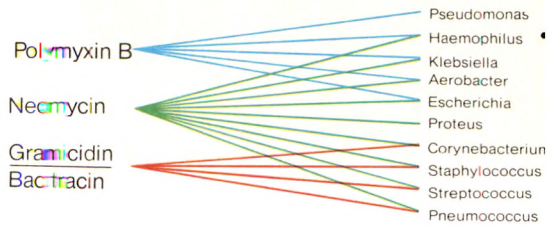


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when eyes need to be open and vision clear, eye drops may be the more appropriate choice

solution will not cause blurring



NEOSPORIN®

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NEOSPORIN®
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Each cc contains: Aerosporin® brand Polymyxin B Sulfate 5,000 Units; neomycin sulfate 2.5 mg (equivalent to 1.75 mg neomycin base); gramicidin 0.025 mg. Vehicle contains alcohol 0.5%, thimerosal (preservative) 0.001% and the inactive ingredients propylene glycol, polyoxyethylene polyoxypropylene compound, sodium chloride and purified water.

NEOSPORIN®
Ophthalmic Ointment Sterile (Polymyxin B–Bacitracin–Neomycin)

Each gram contains: Aerosporin® brand Polymyxin B Sulfate 5,000 Units; zinc bacitracin 400 Units; neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base); special white petrolatum qs.

Brief Disclosure below applies to the solution and ointment.

INDICATIONS: For the short-term treatment of superficial external ocular infections caused by organisms susceptible to one or more of the antibiotics contained therein.

CONTRAINDICATIONS: Contraindicated in those persons who have shown sensitivity to any of the components.

WARNINGS: Prolonged use may result in overgrowth of nonsusceptible organisms. Ophthalmic Ointment may retard corneal healing.

PRECAUTIONS: Culture and susceptibility testing should be performed during treatment.

Allergic cross-reactions may occur which could prevent the use of any or all of the following antibiotics for the treatment of future infections: kanamycin, paromomycin, streptomycin, and possibly gentamicin.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Complete literature available on request from Professional Services Dept. PML.

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Research Triangle Park
North Carolina 27709

CONTACT LENS ASSOCIATION OF OPHTHALMOLOGISTS, INC.

Scientific Meeting

Sunday, November 2, 1980

7:00 p.m. - 9:00 p.m.

Room D, Hilton Hotel, Chicago, Illinois

PROGRAM

Richard H. Keates, M.D., Chairman

7:00 pm—SYMPOSIUM: Controversial Questions in Contact Lenses.
Panel: H. Dwight Cavanagh, M.D., Paul R. Honan, Jr., M.D., Perry S. Binder, M.D., R. Linsy Farris, M.D., Oliver H. Dabezies, Jr., M.D.

7:30 pm-8:15 pm Contact and Intraocular Lens Presentations

7:30 pm—Richard A. Saunders, M.D.: Empirical Fitting of Hard Contact Lenses in Infants and Young Children. *Discussant:* Perry Rosenthal, M.D.

7:41 pm—Louis A. Wilson, M.D.: Thimerosal Hypersensitivity: A Cause of Ocular Redness and Irritation amongst Patients Wearing Soft Contact Lenses. *Discussant:* Rudolf M. Frankin, M.D.

7:52 pm—A. Robert Bellows, M.D.: Endophthalmitis in Aphakic Patients with Inadvertent Filtering Blebs Wearing Contact Lenses. *Discussant:* Frederick H. Theodore, M.D.

8:04 pm—J. V. Greiner, M.D.: Effect of Contact Lens Wear on the Conjunctival Mucus Systems. *Discussant:* Roswell Pfister, M.D.

8:15 pm—SYMPOSIUM: Controversial Questions in Implants and Refractive Surgery.
Panel: Richard P. Kratz, M.D., Henry M. Clayman, M.D., Jerald L. Tennant, M.D., Kenneth J. Hoffer, M.D., Stephen A. Obstbaum, M.D.

No Registration Fee

The Profession is Cordially Invited

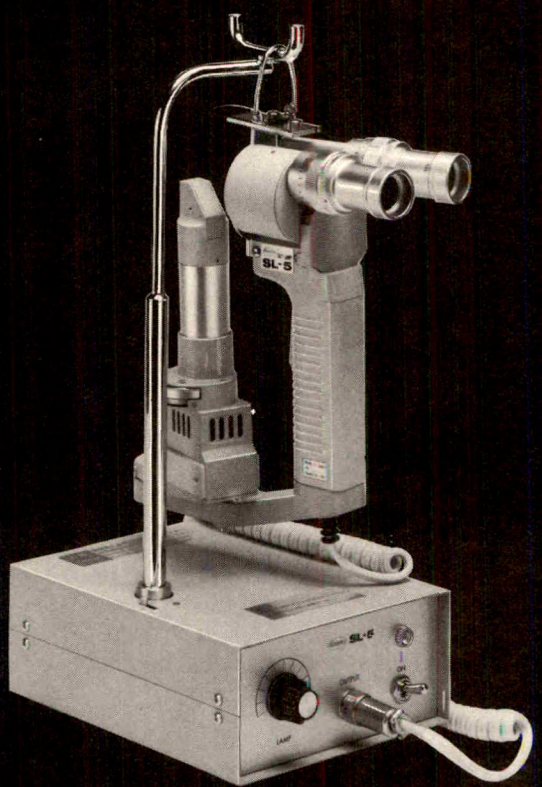
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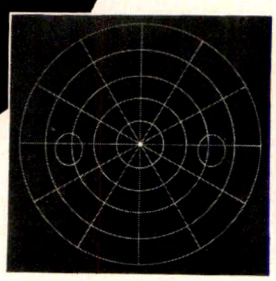
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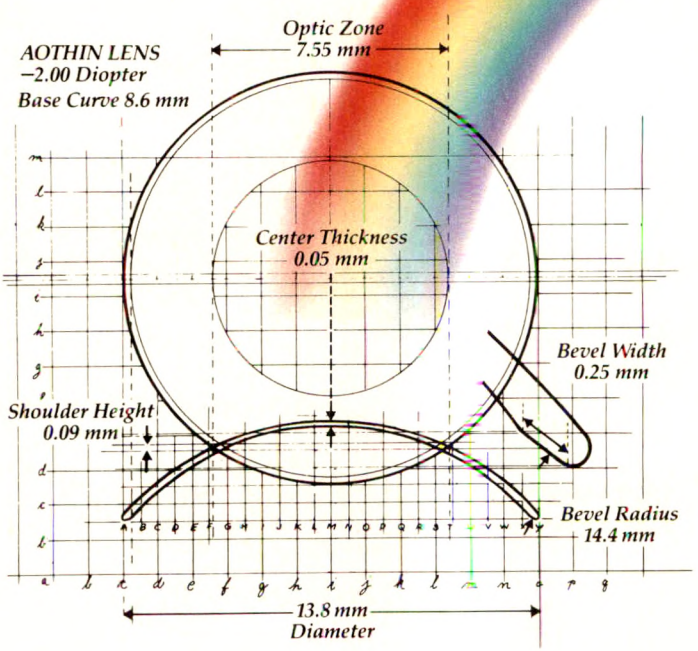
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THE THIN SOFT LENS WITH A CONSISTENTLY THIN CARRIER

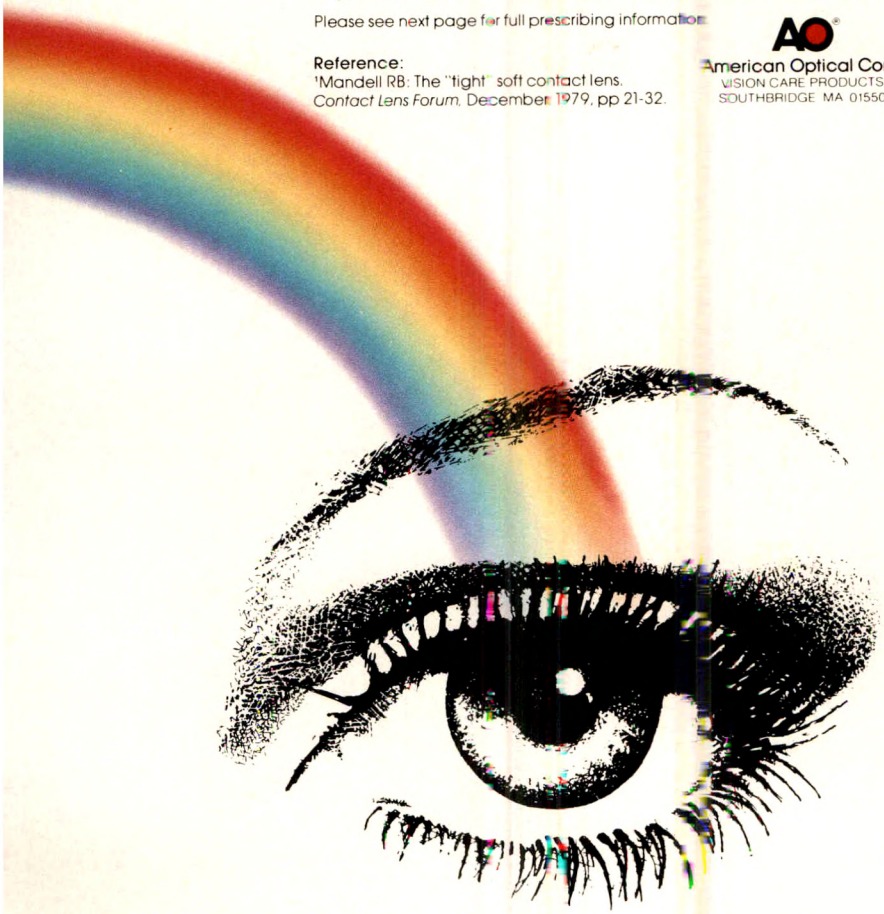
With some other thin soft lenses, portions of the lens periphery are just as thick as with a standard soft lens. Yet overall, the thickness of a soft lens may be the most frequent cause of corneal edema.[1] So we've developed a thin soft lens with a carrier that's consistently thin throughout, even in high minus powers, for consistently good oxygen exchange.

Of course, the new AOTHIN lens offers you all you've come to expect from an American Optical lens. Excellent visual acuity, durability and ease of handling. With a simple set of lens parameters: one diameter (13.8 mm), three base curves (8.3, 8.6 and 9.0 mm) and a minus range of -0.25D to -7.75D in .25 diopter steps. For further details, please contact your American Optical representative or American Optical Soft Contact Lens Distributor TODAY!

Please see next page for full prescribing information.

Reference:
[1]Mandell RB: The "tight" soft contact lens. *Contact Lens Forum*, December 1979, pp 21-32.

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TO BRING MORE OXYGEN TO THE CORNEA

NEW AOTHIN™ (TETRAFILCON A)

SOFT CONTACT LENS

AOTHIN™ (tetrafilcon A) Soft Contact Lens

DESCRIPTION: AOTHIN™ (tetrafilcon A) Soft Contact Lens is a hemispherical shell which covers the cornea and may cover a portion of the adjacent sclera. The lens material, tetrafilcon A, is a hydrophilic random terpolymer of 2-hydroxyethyl methacrylate, N-vinyl-2-pyrrolidone and methylmethacrylate. The polymer is a three-dimensional network of terpolymer chains joined by divinylbenzene cross-links. It consists of 57.5% tetrafilcon A and 42.5% water by weight when fully hydrated in 0.9% sodium chloride solution buffered with monobasic and dibasic sodium phosphates.

ACTIONS: In its hydrated state, the AOTHIN™ Soft Contact Lens is soft and pliable. When dry, the lens becomes hard and brittle. These states are completely reversible and a lens which has been permitted to dry out will recover all of its hydrated properties when placed in normal saline for a period of two hours. When placed on the human cornea, the hydrated lens acts as a refracting medium to compensate spherical ametropias. The material has a refractive index of 1.43 and the lens has a visible light transmittance greater than 97%.

INDICATIONS: AOTHIN™ Soft Contact Lenses are indicated for the correction of vision in persons with non-diseased eyes who have spherical ametropias, corneal astigmatism of 2.50 diopters or less and/or refractive astigmatism of 2.00 diopters or less.

CONTRAINDICATIONS: AOTHIN™ Soft Contact Lenses are contraindicated in the presence of any of the following conditions: (1) Acute and subacute inflammation of the anterior segment of the eyes. (2) Any eye disease which affects the cornea and conjunctiva. (3) Insufficiency of lacrimal secretion. (4) Corneal hypoesthesia. (5) Any systemic disease which may affect the eye or be exaggerated by wearing contact lenses.

WARNINGS: Medications and Eye Drops: AOTHIN™ Lenses must be stored in an appropriate solution when off the eyes; the type of solution being dependent on the system used for disinfection. When the lenses are disinfected with the thermal disinfection system, they may be stored *only* in LENSRINS® or BOIL n SOAK® Solution (sterile, buffered preserved saline solution). When lenses are disinfected with the chemical disinfection system, they may be stored *only* in FLEXSOL® Solution (sterile, isotonic buffered solution containing Adsorbobase — a water soluble polymeric vehicle — and preserved with thimerosal 0.001%, chlorhexidine 0.005% and edetate disodium 0.1%).

No ophthalmic solutions or medications, including hard contact lens solution, can be used by AOTHIN™ Lens wearers prior to or while the lenses are in place on the eyes. Also, no solutions, including hard contact lens solutions, other than LENSRINS® or BOIL n SOAK® Solution or FLEXSOL® Solution may be used on AOTHIN™ Lenses when they are off the eyes.

Since liquids and vapors may be absorbed by AOTHIN™ Lenses, they should not be placed in the mouth for wetting, nor wiped with a cloth or tissue.

Abrasions and Infections: If a lens becomes less comfortable than it was when first placed on the wearer's cornea, the lens should be removed immediately and the wearer's eye and the lens examined for the possible presence of a foreign body. If any eye abrasion, ulceration, irritation or infection is present, or any abnormal eye condition is observed concurrently with lens wear, the lens should be removed immediately and a physician consulted.

Wearing Restrictions: AOTHIN™ Soft Contact Lenses should not be worn while swimming, sleeping, or in the presence of irritating fumes or vapors.

Visual Blurring: If visual blurring occurs, the lens must be removed until the condition subsides.

Lens Sanitation: Patients must adhere to the recommended daily sanitary care procedures for AOTHIN™ Soft Contact Lenses. Failure to follow this procedure may result in the development of serious ocular infections.

PRECAUTIONS: Storage: AOTHIN™ Lenses may be stored *only* in the appropriate storage solution: LENSRINS® or BOIL n SOAK® Solution or FLEXSOL® Solution depending on disinfecting methods used. If left exposed to air, the lenses will dehydrate, become brittle and break readily. If a lens dehydrates, it should be soaked in either LENSRINS® BOIL n SOAK® or FLEXSOL® Solution until it returns to a soft, supple state.

Cleaning and Disinfecting: AOTHIN™ Lenses must be both cleaned and disinfected daily. Separate procedures and products are needed to clean and to disinfect. Two methods of disinfection, thermal or chemical, have been shown to be equally effective. The choice of disinfection system should be made in consultation with your eye care practitioner.

Cleaning: Daily cleaning is necessary to remove mucus and other deposits which may have accumulated on the lens surface. Each time the lenses are removed from the wearer's eyes, both surfaces of the lenses must be cleaned using several drops of PREFLEX® Cleaning Solution (sterile solution of a nonionic cleaner preserved with thimerosal 0.004% and edetate disodium 0.2%). Lenses must be cleaned before they are disinfected, as deposits on the lenses tend to harden and become more difficult to remove after the lenses are disinfected.

Disinfecting: AOTHIN™ Soft Contact Lenses may be disinfected with either a thermal or chemical regimen. One method or the other must be selected, but not both. THE USER MUST NOT ALTERNATE BETWEEN METHODS.

Thermal Disinfection Method: AOTHIN™ Lenses may be effectively disinfected using a disinfecting unit approved for use with tetrafilcon A hydrophilic contact lenses. After cleaning with PREFLEX® Cleaning Solution, rinse lenses with LENSRINS® or BOIL n SOAK® solution and disinfect in an approved thermal disinfection unit. The lens storage containers must be emptied and filled with fresh LENSRINS® or BOIL n SOAK® solution just prior to disinfecting the lenses.

Alternate Thermal Disinfection Method: If a thermal unit is not available, the following disinfection system can be used temporarily. After cleaning the lenses according to instructions, fill the lens storage container with fresh LENSRINS® or BOIL n SOAK® solution. Bring a pan of water to a rolling boil. Remove the pan from heat. Place the tightly sealed lens storage container into the hot water and allow to cool. When the water is cool, lenses have been disinfected.

NOTE: Use of an approved thermal disinfection unit should be resumed as soon as possible.

Chemical Disinfection: Disinfection with PREFLEX® Cleaning Solution, FLEXSOL® Solution and NORMOL® Rinsing Solution (sterile isotonic saline solution preserved with thimerosal 0.001% and chlorhexidine 0.005%) has also been shown to be an effective disinfection system for daily care of AOTHIN™ (tetrafilcon A) Soft Contact Lenses.

AOTHIN™ Lenses must be cleaned and rinsed daily (or after wearing) with PREFLEX® Cleaning Solution and NORMOL® Rinsing Solution. The FLEXKIT Lens Case must be emptied and refilled with fresh FLEXSOL® Solution each time the lens is stored. Fresh FLEXSOL® Solution must be used daily for storage and disinfection.

WARNING: DO NOT MIX OR ALTERNATE THE DISINFECTION AND STORAGE SYSTEMS. FLEXSOL® SOLUTION SHOULD NOT BE USED WITH HEAT.

Hygiene: Before handling the lenses, hands must be washed, rinsed thoroughly and dried with a lint-free towel. Cosmetics, lotions, soaps, oil and hand creams must not come in contact with the lenses since eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until the spray has settled.

Fluorescein: Never use fluorescein while the patient is wearing the lenses because the lenses will become discolored. Whenever fluorescein is used, flush the eyes with LENSRINS® or BOIL n SOAK® and wait at least one hour before replacing the lenses. Too early replacement may allow the lenses to absorb residual fluorescein.

ADVERSE REACTIONS: Serious corneal damage may result from wearing lenses which may have soaked in hard contact lens solutions. Eye irritation may occur within a short time after putting on a hypotonic lens. Removal of the lens will relieve the irritation.

Very rarely a lens may adhere to an eye as a result of a patient sleeping with the lens on, as a result of wearing a hypotonic lens. If a lens adheres for any reason, the patient may be instructed to apply a few drops of LENSRINS® or BOIL n SOAK® Solution (if using a thermal disinfection regimen) or ADAPETTES® Cleaning and Rewetting Solution (sterile, isotonic, buffered solution containing Adsorbobase and preserved with thimerosal 0.004% and edetate disodium 0.1%) (if using a chemical disinfection regimen), and wait until the lens moves freely before removing it.

Clinical studies indicate that corneal edema as manifested by symptoms such as rainbows or halos around light or visual blurring may occur if lenses are worn continuously for too long a time. Removal of the lenses and a rest period of at least one hour generally relieve these symptoms. If symptoms do not subside promptly, professional consultation should be obtained.

Excessive tearing, unusual eye secretions and photophobia are not normal. If these symptoms occur, the patient should be examined to determine their cause.

DOSAGE AND ADMINISTRATION: Fitting: Conventional methods of fitting contact lenses do not apply to AOTHIN™ Soft Contact Lenses. For a detailed description of the fitting technique, refer to the Fitting Guide for AOTHIN™ (tetrafilcon A) Soft Contact Lenses, copies of which are available from: American Optical Corp., Vision Care Products, Southbridge, MA 01550.

Wearing Schedule: There may be a tendency for the patient to overwear the lenses initially. Therefore, the importance of adhering to the following initial daily wearing schedule should be stressed to the patient.

Day	Wear Time *(Hours)*	Rest Period *(Hours)*	Wear Time *(Hours)*
1	4	2	4
2	4	2	4
3	5	2	5
4	6	2	5
5	7	2	5
6	7	1	6
7	8	1	7
8	8	1	8
9	9	1	8
10-14	10	1	balance of
15	all waking hours		waking hours*

*lenses should never be worn 24 hours a day.

LENS CARE AND HANDLING: Care must be taken on the initial visit to assure that the patient is supplied with an approved disinfection system and fully understands all care and handling instructions for the lenses. As with any contact lens, regular recall visits are necessary to assure patient health and compliance with instructions.

HOW SUPPLIED: Each lens is supplied sterile in a glass vial containing 0.9% sodium chloride solution buffered with monobasic and dibasic sodium phosphates. The glass vial is marked with the Base Curve, Diameter, Dioptric Power, and Lot Number.

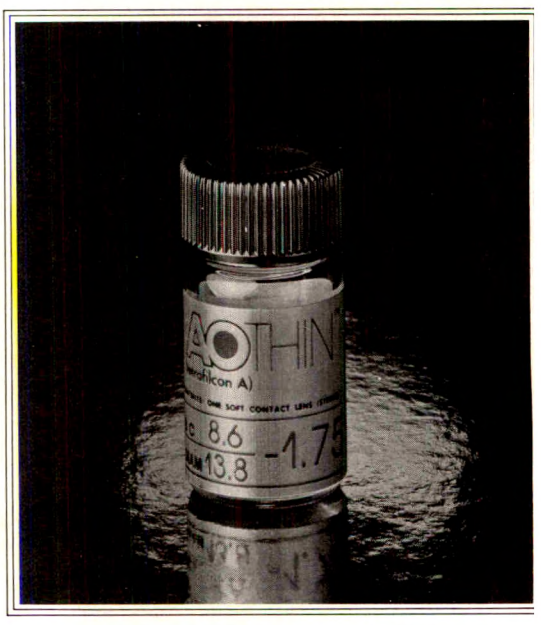
Approved thermal and chemical disinfection systems and care kits are available from a variety of manufacturers. Instructions on the use of these systems are contained in the AOTHIN™ Patient Instruction Booklet.

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Epithelial Keratitis Caused by Herpes Simplex Virus

VIROPTIC®

(Trifluridine)

OPHTHALMIC SOLUTION, 1%

STERILE

95% of Patients Responded

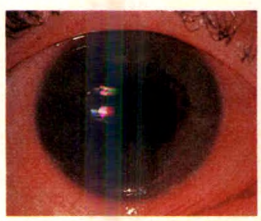
During controlled clinical trials 95% of patients (92 of 97) responded to VIROPTIC therapy. Dendritic and geographic ulcers were re-epithelialized in a mean time of 6 and 7 days, respectively.

Significantly More Convenient

- Less frequent applications
- No middle of the night dosage
- None of the blurring and inconvenience of ointments

5 to 9 Drops Daily

One drop every 2 hours while awake until herpetic lesion has completely re-epithelialized, then one drop every 4 hours for 7 days post-re-epithelialization.



0 days



5 days after VIROPTIC therapy

Effective in 138 of 150 patients Unresponsive or Intolerant to Idoxuridine or Vidarabine

In studies of patients unresponsive or intolerant to idoxuridine or vidarabine, VIROPTIC demonstrated 92% effectiveness. Dendritic and geographic ulcers were re-epithelialized in a mean time of 6 and 12 days, respectively.

Please see following page for brief summary of prescribing information.

The First Antiviral Product From ONE HUNDRED YEARS WELLCOME **Burroughs Wellcome Co. Research**

UNIVERSITY COLLEGE OF MEDICINE LIBRARY

VIROPTIC®
(Trifluridine)
OPHTHALMIC SOLUTION, 1%
STERILE

DESCRIPTION: VIROPTIC® is the brand name for trifluridine (also known as trifluorothymidine, F_3TdR,F_3T), an antiviral drug for topical treatment of epithelial keratitis caused by Herpes simplex virus. The chemical name of trifluridine is 5-trifluoromethyl-2′-deoxyuridine.

VIROPTIC sterile ophthalmic solution contains 1% trifluridine in an aqueous solution with acetic acid and sodium acetate (buffers), sodium chloride, and thimerosal 0.001% (added as a preservative).

CLINICAL PHARMACOLOGY: Trifluridine is a fluorinated pyrimidine nucleoside with *in vitro* and *in vivo* activity against Herpes simplex virus, types 1 and 2 and vacciniavirus. Some strains of Adenovirus are also inhibited *in vitro*.

Intraocular penetration of trifluridine occurs after topical instillation of VIROPTIC into human eyes. Decreased corneal integrity or stromal or uveal inflammation may enhance the penetration of trifluridine into the aqueous humor. Unlike the results of ocular penetration of trifluridine *in vitro*, 5-carboxy-2′-deoxyuridine was not found in detectable concentrations within the aqueous humor of the human eye.

INDICATIONS AND USAGE: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% is indicated for the treatment of primary keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus, types 1 and 2. VIROPTIC is also effective in the treatment of epithelial keratitis that has not responded clinically to the topical administration of idoxuridine or when ocular toxicity or hypersensitivity to idoxuridine has occurred. In a smaller number of patients found to be resistant to topical vidarabine, VIROPTIC was also effective.

The clinical efficacy of VIROPTIC in the treatment of stromal keratitis and uveitis due to Herpes simplex or ophthalmic infections caused by vacciniavirus and Adenovirus has not been established by well-controlled clinical trials. VIROPTIC has not been shown to be effective in the prophylaxis of Herpes simplex virus keratoconjunctivitis and epithelial keratitis by well-controlled clinical trials. VIROPTIC is not effective against bacterial, fungal or chlamydial infections of the cornea or nonviral trophic lesions.

During controlled multicenter clinical trials, 92 of 97 (95%) patients (78 of 81 with dendritic and 14 of 16 with geographic ulcers) responded to VIROPTIC therapy as evidenced by complete corneal re-epithelialization within the 14 day therapy period. In these controlled studies, 56 of 75 (75%) patients (49 of 58 with dendritic and 7 of 17 with geographic ulcers) responded to idoxuridine therapy. The mean time to corneal re-epithelialization for dendritic ulcers (6 days) and geographic ulcers (7 days) was similar for both therapies. In other clinical studies, VIROPTIC was evaluated in the treatment of Herpes simplex virus keratitis in patients who were unresponsive or intolerant to the topical administration of idoxuridine or vidarabine. VIROPTIC was effective in 138 of 150 (92%) patients (109 of 114 with dendritic and 29 of 36 with geographic ulcers) as evidenced by corneal re-epithelialization. The mean time to corneal re-epithelialization was 6 days for patients with dendritic ulcers and 12 days for patients with geographic ulcers.

CONTRAINDICATIONS: VIROPTIC brand Trifluridine Ophthalmic Solution, 1%, is contraindicated for patients who develop hypersensitivity reactions or chemical intolerance to trifluridine.

WARNINGS: The recommended dosage and frequency of administration should not be exceeded (see Dosage and Administration).

PRECAUTIONS:

General: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% should be prescribed only for patients who have a clinical diagnosis of herpetic keratitis.

VIROPTIC may cause mild local irritation of the conjunctiva and cornea when instilled but these effects are usually transient.

Although documented *in vitro* viral resistance to trifluridine has not been reported following multiple exposure to VIROPTIC, the possibility exists of viral resistance development.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Mutagenic Potential. Trifluridine has been shown to exert mutagenic, DNA damaging and cell transforming activities in various standard *in vitro* test systems, and clastogenic activity in *Vicia faba* cells.

Although the significance of these test results is not clear or fully understood, there exists the possibility that mutagenic agents may cause genetic damage in humans.

Oncogenic Potential. The oncogenic potential of trifluridine is unknown at this time. The oncogenic potential of trifluridine in rodents is being evaluated.

Pregnancy: The drug should not be prescribed for pregnant women unless the potential benefits outweigh the potential risks.

Nursing Mothers: It is unlikely that trifluridine is excreted in human milk after ophthalmic instillation of VIROPTIC because of the relatively small dosage (≤5.0 mg/day), its dilution in body fluids and its extremely short half-life (approximately 12 minutes). The drug should not be prescribed for nursing mothers unless the potential benefits outweigh the potential risks.

ADVERSE REACTIONS: The most frequent adverse reactions reported during controlled clinical trials were mild, transient burning or stinging upon instillation (4.6%) and palpebral edema (2.8%). Other adverse reactions in decreasing order of reported frequency were superficial punctate keratopathy, epithelial keratopathy, hypersensitivity reaction, stromal edema, irritation, keratitis sicca, hyperemia, and increased intraocular pressure.

OVERDOSAGE: Overdosage by ocular instillation is unlikely because any excess solution should be quickly expelled from the conjunctival sac.

Acute overdosage by accidental oral ingestion of VIROPTIC has not occurred. However, should such ingestion occur, the 75 mg dosage of trifluridine in a 7.5 ml bottle of VIROPTIC is not likely to produce adverse effects. Single intravenous doses of 15-30 mg/kg/day in children and adults with neoplastic disease produce reversible bone marrow depression as the only potentially serious toxic effect and only after 3-5 courses of therapy. The acute oral LD_{50} in the mouse and rat was 4379 mg/kg or higher.

DOSAGE AND ADMINISTRATION: Instill one drop of VIROPTIC Ophthalmic Solution, 1% onto the cornea of the affected eye every two hours while awake for a maximum daily dosage of nine drops until the corneal ulcer has completely re-epithelialized. Following re-epithelialization, treatment for an additional seven days of one drop every four hours while awake for a minimum daily dosage of five drops is recommended.

If there are no signs of improvement after seven days of therapy or complete re-epithelialization has not occurred after 14 days of therapy, other forms of therapy should be considered. Continuous adminsitration of VIROPTIC for periods exceeding 21 days should be avoided because of potential ocular toxicity.

HOW SUPPLIED: VIROPTIC Ophthalmic Solution, 1% is supplied as a sterile ophthalmic solution in a plastic Drop-Dose® dispenser bottle of 7.5 mL.

Burroughs Wellcome Co.
Research Triangle Park
North Carolina 27709

MID WINTER NATIONAL MEETING

SAHARA HOTEL, LAS VEGAS, NEVADA

JANUARY 29 - FEBRUARY 1, 1981

(ending 12 noon, Sunday)



CONTACT LENS ASSOCIATION OF OPHTHALMOLOGISTS, INC.

CONRAD BERENS LECTURER
John A. Dyer, M.D.
Corneal Contact Lens in Perspective—1948-1981

DISTINGUISHED VISITING LECTURER
Thomas F. Spring, M.D.
Contact Lenses, Endothelium and Keratoconus

PRACTICE SEMINAR
(9:00 a.m. - 12:00 noon Thursday)

Scientific Program begins at
2:00 p.m. Thursday

Thursday Afternoon (January 29)

Symposium: EXTENDED WEAR OF CONTACT LENSES IN APHAKIA ..... Jack Hartstein, M.D., *Moderator*
Patient Selection ..... Joseph A. Baldone, M.D.
Cooper Lenses ..... Richard G. Lembach, M.D.
Hydrocurve Lenses ..... Perry S. Binder, M.D.
Sauflon Lenses ..... Anthony B. Nesburn, M.D.
Silicone Lenses ..... Robert T. Blackhurst, M.D.
CSI Lenses ..... H. Dwight Cavanagh, M.D.
Complications of Extended Wear Lenses ..... G. Peter Halberg, M.D.
Panel Discussion

THE USE OF EXTENDED WEAR LENSES IN MYOPIA ..... Louis A. Wilson, M.D.
Symposium: THE USE OF EXTENDED WEAR LENSES FOR THERAPEUTIC PURPOSES ..... Richard H. Keates, M.D., *Moderator*
Cooper Lenses ..... Richard G. Lembach, M.D.
Bausch & Lomb Lenses ..... John Brodrick, M.D.
Sauflon Lenses ..... Bruce Bodner, M.D.
PRACTICAL WORKSHOPS ..... *Topics to be announced*

Friday Morning (January 30)

Symposium: TORIC SOFT LENSES ..... Barry A. Maltzman, M.D., *Moderator*
Lens Availability and Design ..... Fitting Techniques and Wear Expectations (Dura-Soft—Hydrocurve)
Fitting Techniques and Wearing Expectations (Hydromore–Miracon–Hycron)...Pitfalls to Avoid in Fitting Toric Lenses
Panel Discussion - Questions and Answers. ***Participants:*** Herschell H. Boyd, M.D., Paul R. Honan, Jr., M.D., Bernard J. Slatt, M.D., Harold A. Stein, M.D.
Symposium: HYGIENIC CARE OF SOFT LENSES ..... Ellis Gruber, M.D., *Moderator*
OPHTHALMIC DEVICES: A Review of Three Years of FDA Involvement ..... Max Talbott, Ph.D.
Symposium: CONTACT LENSES IN INFANTS AND CHILDREN ..... Harold P. Koller, M.D., *Moderator*
Indications and Available Materials for Contact Lenses in Infants and Children ..... Judith E. Gurland, M.D.
Pathophysiology of Contact Lens Therapy in Infants and Children ..... I. Matthew Rabinowicz, M.D.
Contact Lens Fitting Techniques Unique to the Pediatric Population ..... Harold P. Koller, M.D.
Indications and Complications of Intraocular Lenses vs. Contact Lenses in Infants and Children ..... Jerry W. Maida, M.D.
Results of Extended and Daily Wear Contact Lenses in Infants and Children ..... Harold P. Koller, M.D.
DISTINGUISHED VISITING LECTURER ..... **Thomas F. Spring, M.D.**

Spouse Luncheon at Don the Beachcomber Compliments of CLAO

Friday Afternoon (January 30)

CONTACT LENS COURSES - Section I ..... R. Linsy Farris, M.D., *Director, Course Program*
1. Current Concepts in the Correction of Myopia—Extended Wear Lens, Orthokeratology and Refractive Corneal Surgery ..... Perry S. Binder, M.D.
2. Management of Corneal & Contact Lens Problems ..... G. Peter Halberg, M.D., Robert A. D'Amico, M.D.
3. How to Select the Best Soft Lens for Your Patient ..... Ellis Gruber, M.D.

CLAO MID-WINTER MEETING

4. Astigmatism Correction with Hard & Soft Contact Lenses ..... Paul R. Honan, M.D., Herschell H. Boyd, M.D.
5. Specialized Techniques of Lens Fitting ..... Joseph W. Soper
6. Hard Lens Modification—Basic Evaluation Techniques ..... Marvin A. Garland, M.D.
7. Fitting of CAB and Silicone Lenses for Daily and Extended Wear .... Jay I. Lippman, M.D., Dan Sigband, M.D.
8. Contact Lens Fitting in Infants & Children ..... Harold P. Koller, M.D.
9. Introduction to Refractive Keratoplasty ..... Miles H. Friedlander, M.D., Herbert E. Kaufman, M.D.
10. Tearing Problems and Contact Lenses ..... David Lamberts, M.D., Frank J. Holly, Ph.D.
11. Extended Wear of Contact Lenses ..... Rao N. Gullapalli, M.D.
12. Contact Lens Equipment and Solutions ..... Ronald H. Akashi, M.D.

INTRAOCULAR LENS COURSES—Section I ..... Herve M. Byron, M.D., *Director, Course Program*

13. Intracapsular Extractions with Implants—I ..... Stephen Obstbaum, M.D., Marvin Kwitko, M.D.
14. Extracapsular Extractions with Implants—I ..... William Harris, M.D., Robert F. Azar, M.D.
15. Techniques for the Advance Implant Surgeon—I ..... Richard P. Kratz, M.D., Charles Kelman, M.D.
16. Special Instrumentation & Therapeutic Agents ..... Buol K. Heslin, M.D., David J. McIntyre, M.D.

CONTACT LENS COURSES—Section II ..... R. Linsy Farris, M.D., *Director, Course Program*

1. Extended Contact Lens Wear ..... Anthony B. Nesburn, M.D.
2. Update on Therapeutic Contact Lenses ..... Edward L. Shaw, M.D.
3. Keratoconus ..... Frank B. Hoefle, M.D.
4. Soft Lens Fitting Technique ..... Joseph W. Soper
5. Bifocal & Multifocal Contact Lenses for the Presbyope and Aphake ..... Frank J. Weinstock, M.D., Abraham Schlossman, M.D.
6. The Beginning Training & Utilization of a Contact Lens Technician ..... Robert J. Crossen, M.D.
7. Starting from Scratch—Contact Lens Fitting ..... Paul R. Honan, M.D., Herschell H. Boyd, M.D.
8. Hard Lens Modification—Advanced Techniques ..... Marvin A. Garland, M.D., Paul R. Honan, M.D.
9. Soft Toric Lenses ..... Barry A. Maltzman, M.D.
10. Fitting Contact Lenses by Piggy Back Technique ..... Joseph A. Baldone, M.D.
11. Contact Lens Fitting in Difficult Cases ..... Donald J. Doughman, M.D.
12. Allergic Problems & Contact Lens Wear ..... Mark B. Abelson, M.D.

INTRAOCULAR LENS COURSES—Section II ..... Herve M. Byron, M.D., *Director, Course Program*

13. Intracapsular Extraction with Implants—II ..... Stephen Obstbaum, M.D., Marvin Kwitko, M.D.
14. Extracapsular Extraction with Implants—II ..... William Harris, M.D., Robert F. Azar, M.D.
15. Techniques for the Advanced Implant Surgeon—II ..... Richard Kratz, M.D., Charles Kelman, M.D.
16. Keratorefractive Surgery ..... James V. Aquavella, M.D., Dennis D. Shepherd, M.D.

Wine and Cheese Tasting Party Compliments of Barnes-Hind Pharmaceuticals, Inc.

Saturday Morning (January 31)

Symposium: INTERACTION OF THE CORNEAL EPITHELIUM, TEAR FILM, AND THE CONTACT LENS ..... James V. Aquavella, M.D., *Moderator*

Stimulation and Tear Flow ..... Jules L. Baum, M.D.
The Contact Lens Surface ..... Rao N. Gullapalli, M.D.
Immunological Aspects ..... Mark B. Abelson, M.D.
Tear Film ph ..... Michael A. Lemp, M.D.
Tear Film Tonicity ..... R. Linsy Farris, M.D.
Summary and Discussion ..... James V. Aquavella, M.D.

Symposium: PROTECTION OF ENDOTHELIUM DURING IOL SURGERY .. Herbert E. Kaufman, M.D., *Moderator*

OVERVIEW—Problem of Cell Loss During Surgery ..... Herbert E. Kaufman, M.D.
Quantitation of Cell Loss and Its Prevention by Altering Surgical Technique ..... William M. Bourne, M.D.
The Use of Viscose Agents to Minimize Surgical Cell Loss ..... David Miller, M.D.
Lens Coatings and Other Non-Viscose Type Approaches to the Prevention of Cell Loss . Herbert E. Kaufman, M.D.

ISSUES FACING OPHTHALMOLOGY ..... George Edward Garcia, M.D., *President American Association of Ophthalmology*

CONRAD BERENS LECTURER ..... **John A. Dyer, M.D.**
OPHTHALMIC TECHNICIANS COURSE ..... Topics to be announced

Saturday Afternoon (January 31)

THEODORE E. OBRIG MEMORIAL LECTURER ..... To Be Announced
Symposium: WHAT'S NEW AND INTERESTING IN LENS IMPLANTATION ..... Harold A. Stein, M.D., *Moderator*

Patient Selection—Extended Wear of IOL? ..... Harold A. Stein, M.D.
Instrumentation in Surgery ..... Aziz J. Anis, M.D., F.A.C.S.
Newer Intraocular Lens Designs ..... Ronald W. Barnet, M.D.
Sutures for Cataracts, Implant Surgery and Astigmatism Control ..... J. Elliott Blaydes, Jr., M.D.
Statistics on Implant Results ..... James V. Aquavella, M.D.
Some Mechanical Considerations ..... C. William Simcoe, M.D.
Preventing Complications with IOL's ..... Stanley C. Becker, M.D.
Managing Complications ..... Richard P. Kratz, M.D.

Symposium: IS EXTRACAPSULAR CATARACT SURGERY WORTH IT? .... C. William Simcoe, M.D., *Moderator*

Three panelists (Richard P. Kratz, M.D., Richard H. Keates, M.D., and K. Buol Heslin, M.D.) will each give a 10 minute

presentation of their personal experiences regarding: Prognosis for a Successful Repair of Retinal Detachment; Cystoid Macular Edema; Quality of Vision. A Panel Discussion and Questions from the Audience will follow.
MOVIES ..... Robert F. Azar, M.D., *Moderator*

International Party Sponsored by Alcon/BP

Sunday Morning (February 1)

SURGICAL CORRECTION OF REFRACTIVE ERRORS
Jack Hartstein, M.D., *Chairman*

Symposium: SURGICAL CORRECTION OF ASTIGMATISM ..... Richard P. Kratz, M.D., *Moderator*
Prevention: Cataract Wound Closure ..... Clifford Terry, M.D.
Comparison of Keratometers ..... Dennis Shepherd, M.D.
Correction: Reconstruction of Previous Incision ..... Thomas Cravy, M.D.
Corneal Tuck ..... Robert F. Azar, M.D.
Symposium: SURGICAL CORRECTION OF MYOPIA—PRO's AND CON's ..... Walter J. Stark, M.D., *Moderator*
History of Surgical Correction of Myopia and Early Clinical Results ..... Leeds Katzen, M.D.
Surgical Correction of Myopia—Studies in Laboratory Animals and Human Cadaver Eyes ..... Ronald Smith, M.D.
Complications of Radial Keratotomy ..... J. James Rowsey, M.D.
Update of Clinical Experience with Surgical Correction of Myopia ..... Leo D. Bores, M.D.
A Prospective, Multicenter, Clinical Trial of Radial Keratotomy for Myopia ..... George Waring, M.D.
Summary and Conclusions ..... Walter J. Stark, M.D.
Symposium: SURGICAL CORRECTION OF APHAKIA ..... Perry S. Binder, M.D., *Moderator*
Keratophakia and Hypermytropic Keratomileusis ..... Cass Swinger, M.D.
Hypermytropic Keratomileusis ..... Jorge Krumeich, M.D.
Keratophakia & Keratomileusis Hyperopia Using Precarved Frozen Material ..... Miles H. Friedlander, M.D.
Epikeratophakia ..... Herbert E. Kaufman, M.D.

Contact & Intraocular Lens Presentations—Thursday afternoon, Friday morning and Saturday morning

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An optional Practice Management Seminar will be conducted on Thursday, January 29, 1981 immediately preceding the scientific session. There is no registration fee for the Practice Management Seminar; the registration fee for the Scientific Session is the total fee for all sessions.

Registration for the Scientific Session will take place on Wednesday, January 28 from 12:00 noon to 5:00 p.m. and Thursday, January 29 from 7:30 a.m. to 5:30 p.m. Use the registration form below to pre-register and save time at the meeting.

Refund Policy. Full refund if cancellation is received by January 1, 1981. A $25.00 service charge per person for cancellations from January 2, 1981 to January 28, 1981. No refunds for cancellations received after January 29, 1981.

A special room rate has been arranged with the Sahara Hotel. For room reservations only, write to the Sahara Hotel, Las Vegas, Nevada 89114.

PRE-REGISTRATION FORM—CLAO MID-WINTER NATIONAL MEETING
January 29 - February 1, 1981 Sahara Hotel Las Vegas, Nevada

Name _____ M.D. _____ Other _____

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☐ Physician Member of CLAO—US$100.00 ☐ Physician Non-Member—US$250.00

☐ Resident Physician in Ophthalmology—US$50.00

☐ Technician* employed by Ophthalmologist (Physician Member of CLAO). Must be identified by letter by the employing ophthalmologist—US$75.00

☐ Technician* employed by Ophthalmologist (Non-Member of CLAO). Must be identified by letter by the employing ophthalmologist—US$175.00

☐ ALL OTHERS—US$275.00 ☐ ONE DAY REGISTRATION—US$50.00 Please specify day:

Please make check payable to: CLAO—Mid Winter Meeting and forward to R. Linsy Farris, M.D.
Treasurer—CLAO
2620 Jena Street
New Orleans, LA 70115

*Technicians. This fee includes the entire meeting (3-1/2 days), in addition to the Ophthalmic Assistants and Technicians Course.

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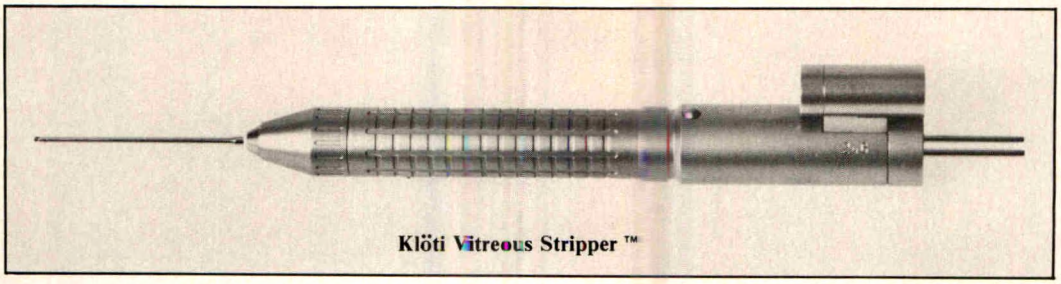
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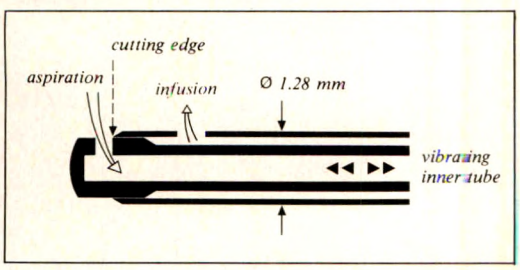
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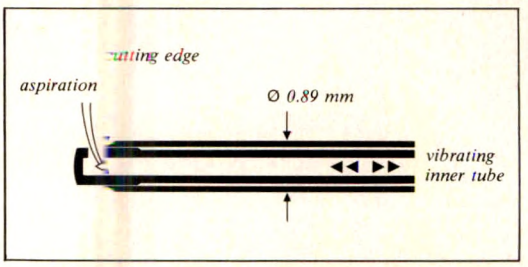
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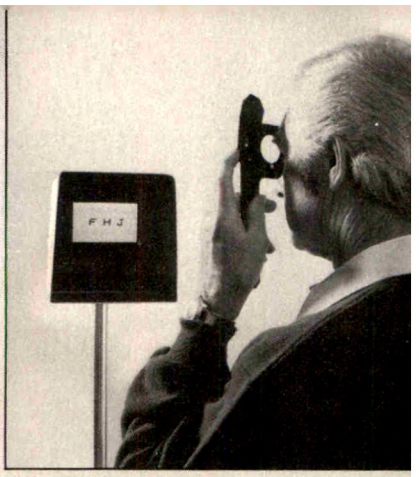


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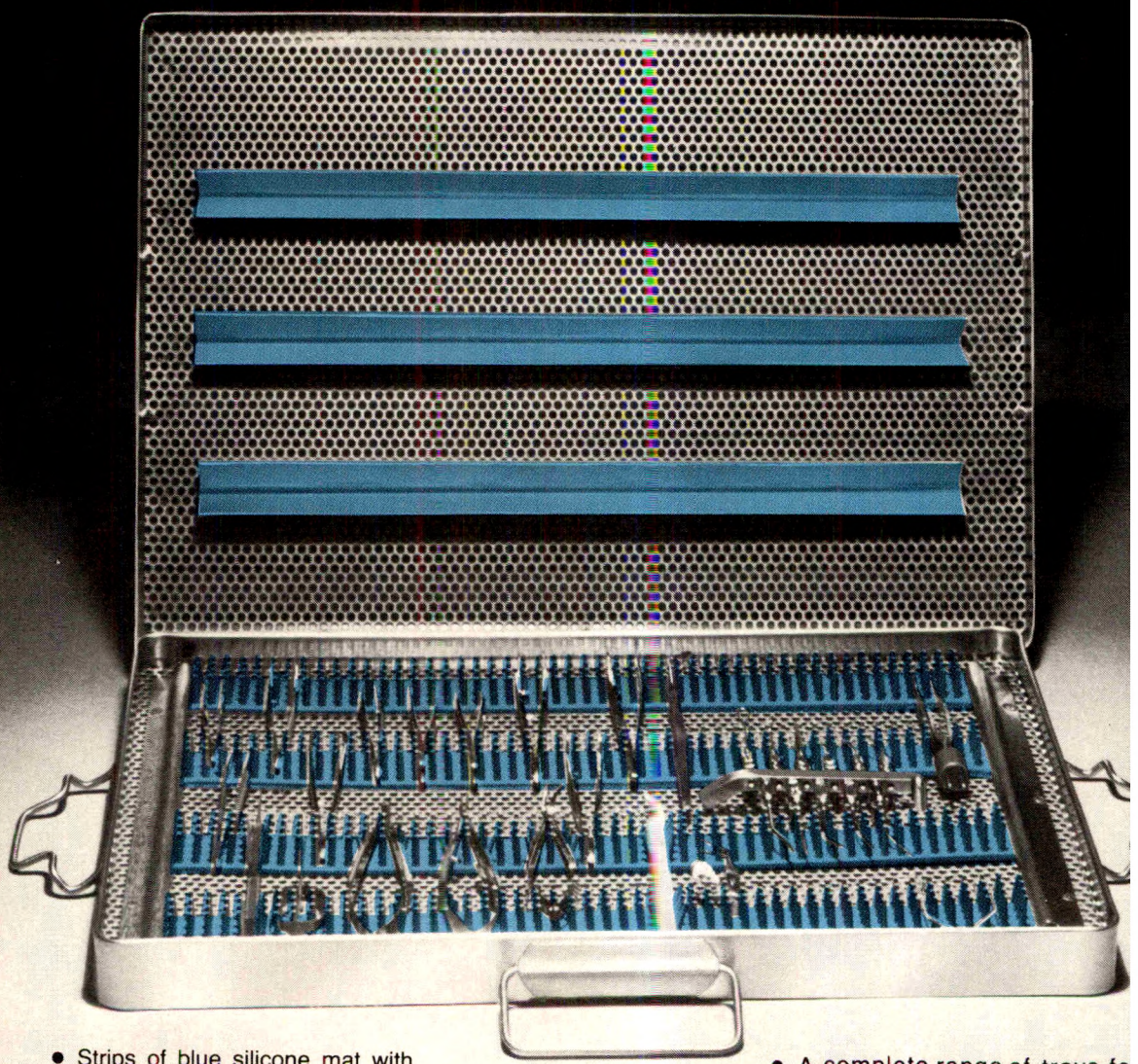
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ere's a winning strategy for your contact s practice...offer your patients the cellent centration and outstanding visual uity of Aquaflex (tetrafilcon A) lenses at a wer cost.

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I prefer to it from:
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In chronic open-angle glaucom

NEW 40-MONTH STUDY REPORTS LONG-TERM EFFICACY AND TOLERABILITY

Timoptic

(TIMOLOL MALEATE | MSD)
OPHTHALMIC SOLUTION

In an open, long-term maintenance study* of the effect of continuous therapy (up to 40 months) of chronic open-angle glaucoma with TIMOPTIC, 96% of the patients exhibited IOP values of ≤ 21 mm Hg at each measurement during treatment with TIMOPTIC...and the drug was generally well tolerated. (See graphs and data on facing page.)

A better quality of life for many patients

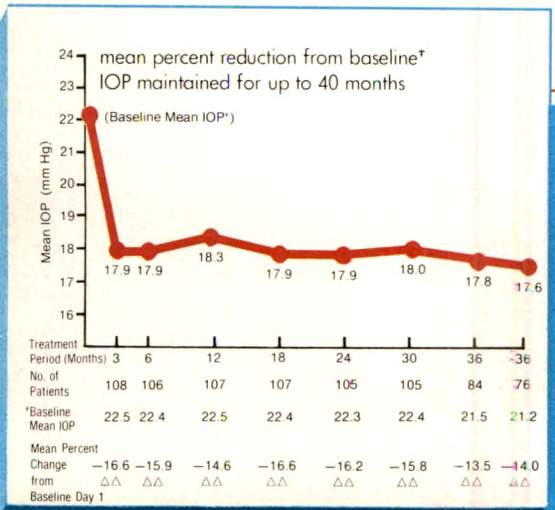
Patients on TIMOPTIC generally do not experience the following therapy-limiting side effects: night blindness/blurred vision/dimming of vision**

Patients on TIMOPTIC have significantly fewer and less severe side effects than those on pilocarpine or epinephrine, i.e., a much lower incidence of ocular discomfort—such as less burning, smarting, itching, browache, and headache.**

TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product. As with other topically applied ophthalmic drugs, this drug may be absorbed systemically. As with the use of other antiglaucoma drugs, diminished responsiveness to TIMOPTIC after prolonged therapy has been reported in some patients.

*Data on file, Merck Sharp & Dohme Research Laboratories. Additional information available on request.

**Based on controlled multiclinic studies. Data on file, Merck Sharp & Dohme Research Laboratories. Additional information available on request.

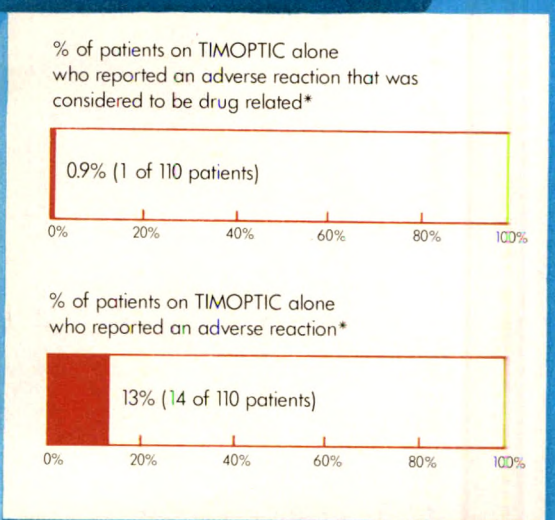


long-term efficacy of Timoptic® (Timolol Maleate) MSD

There was virtually no loss of therapeutic effect in patients treated for up to 40 months with TIMOPTIC alone, i.e., the IOP of almost all patients was lowered to 21 mm Hg or below throughout the study.*

†Of the 110 patients on TIMOPTIC alone, 91 were on previous antiglaucoma treatment and had a treated baseline mean IOP of 21.4 mm Hg. The 19 patients with no previous antiglaucoma treatment had an untreated baseline mean IOP of 27.5 mm Hg. Thus, the mean IOP at baseline for the combined group is 22.5 mm Hg.

ΔΔSignificant percent change from baseline, $p < 0.01$.



long-term tolerability of Timoptic® (Timolol Maleate) MSD

During the 40-month study,* 14 of 110 patients (13%) on TIMOPTIC alone reported an adverse reaction, but only one of these, an allergic reaction, was considered to be drug related.

Visual disturbances including refractive changes (due to withdrawal of miotic therapy in some cases) have been infrequently associated with therapy with TIMOPTIC. Rarely, aphakic cystoid macular edema has been reported, but a causal relationship to therapy with TIMOPTIC has not been established.

*Data on file, Merck Sharp & Dohme Research Laboratories. Additional information available on request.

For a brief summary of prescribing information, please see following page.

MSD
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NOW AVAILABLE
10-ml OCUMETER®

b.i.d.

Timoptic®

(TIMOLOL MALEATE | MSD)
OPHTHALMIC SOLUTION

a beta-adrenergic blocking agent in ophthalmic solution

a significant advance for the reduction of IOP in:

- chronic open-angle glaucoma
- aphakic glaucoma
- some patients with secondary glaucoma
- patients with elevated intraocular pressure at sufficient risk to require lowering of the ocular pressure
- some patients who respond inadequately to multiple antiglaucoma drug therapy

How to start patients on TIMOPTIC:
Usual starting dosage: one drop 0.25% TIMOPTIC in each eye twice a day.

How to transfer from current therapy to TIMOPTIC:
1. On the first day: continue with the agent(s) already being used and add one drop 0.25% TIMOPTIC in each eye b.i.d.

2. On the second day: discontinue the previously used agent(s) and continue with TIMOPTIC in each eye b.i.d.

If clinical response is not adequate:
Dosage may be increased (from the 0.25% solution) by changing to one drop 0.5% TIMOPTIC twice a day in each eye. Dosages above one drop of 0.5% TIMOPTIC twice a day generally have not been shown to produce further reduction of IOP. If the intraocular pressure is maintained at satisfactory levels, the dosage schedule may be changed to one drop once a day in each eye.

Because of diurnal variations in intraocular pressure, satisfactory response to the once-a-day dose is best determined by measuring the intraocular pressure at different times during the day.

In patients with a history of severe cardiac disease, pulse rates should be checked.

CONTRAINDICATIONS: TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product.
WARNINGS: As with other topically applied ophthalmic drugs, this drug may be absorbed systemically.
PRECAUTIONS: Should be used with caution in patients with known contraindications to systemic use of beta-adrenergic receptor blocking agents. These include bronchospastic disease; sinus bradycardia and greater than first-degree block; cardiogenic shock; cardiac failure; concomitant use with adrenergic-augmenting psychotropic drugs. In patients with a history of severe cardiac disease, pulse rates should be checked. Patients who are already receiving a beta-adrenergic blocking agent orally and who are given TIMOPTIC should be observed for a potential additive effect either on the intraocular pressure or on the known systemic effects of beta blockade.

Sufficient clinical data have not been obtained to show safety or efficacy in narrow-angle or angle-closure glaucoma.

As with the use of other antiglaucoma drugs, diminished responsiveness to TIMOPTIC after prolonged therapy has been reported in some patients. However, in one long-term study in which 96 patients have been followed for at least 3 years, no significant difference in mean intraocular pressure has been observed after initial stabilization.
Pregnancy: TIMOPTIC has not been studied in human pregnancy. The use of TIMOPTIC in pregnant women requires that the anticipated benefit be weighed against possible hazards.
Use in Children: Since clinical studies in children have not been done, TIMOPTIC is not recommended for use in children at this time.
Animal Studies: No adverse ocular effects were observed in rabbits and dogs administered TIMOPTIC topically in studies lasting one and two years respectively.

Lifetime studies with timolol have been completed in rats at oral doses of 25, 100, and 300 mg/kg/day and in mice at oral doses of 5, 50, and 500 mg/kg/day. In male and female rats and male mice at all dose levels and in female mice at dose levels of 5 and 50 mg/kg/day, timolol demonstrated no carcinogenic effect. There was a slight increase in the incidence of mammary adenocarcinomas in female mice that received 500 mg/kg/day (about 500 times the maximum recommended human oral dose on a mg/kg basis). Timolol caused dose-related elevations of serum prolactin in female mice at doses of 100 mg/kg or more, but only very slight transient elevations were found in male mice at doses of 500 mg/kg. Since numerous studies have demonstrated that drugs which cause elevations of serum prolactin are associated with mammary tumors in rodents, the mammary tumors in the female mice in the highest dosage group of this study were considered to have resulted from an increased serum prolactin. In humans, no such association between serum prolactin and mammary carcinoma has been established. Furthermore, in adult human female subjects who received oral dosages of up to 60 mg of timolol, the maximum recommended human oral dosage, there were no clinically meaningful changes in serum prolactin.
ADVERSE REACTIONS: TIMOPTIC Ophthalmic Solution is usually well tolerated. The following adverse reactions have been reported either in clinical trials of up to 3 years duration prior to release in 1978 or since the drug has been marketed:

Signs and symptoms of ocular irritation, including conjunctivitis, blepharitis, and keratitis, have been reported occasionally.

Hypersensitivity reactions, including localized and generalized rash, and urticaria have been reported rarely.

Visual disturbances including refractive changes (due to withdrawal of miotic therapy in some cases) have been infrequently associated with therapy with TIMOPTIC. Rarely, aphakic cystoid macular edema has been reported, but a causal relationship to therapy with TIMOPTIC has not been established.

Aggravation or precipitation of certain cardiovascular and pulmonary disorders has been reported, presumably related to effects of systemic beta blockade (see PRECAUTIONS). These include bradyarrhythmia, hypotension, syncope, and bronchospasm (predominantly in patients with pre-existing bronchospastic disease). In clinical trials, slight reduction of the resting heart rate in some patients (mean reduction 2.9 beats/minute, standard deviation 10.2) has been observed.

The following adverse effects have been reported rarely, and a causal relationship to therapy with TIMOPTIC has not been established: headache, anorexia, dyspepsia, nausea, dizziness, CNS effects (fatigue, confusion, depression, somnolence, and anxiety), palpitation, and hypertension.
HOW SUPPLIED: TIMOPTIC Ophthalmic Solution, 0.25% and TIMOPTIC Ophthalmic Solution, 0.5%. Both are available in 5-ml and 10-ml plastic OCUMETER® Ophthalmic Dispensers with a controlled drop tip.
980(DC)4E011)

MSD
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For more detailed information, consult your MSD representative and the full Prescribing Information.
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This precision hand held instrument permits easy reading of diastolic and systolic retinal artery pressure. The disc and arteries are seen clearly through a fundus contact lens in conjunction with a slit lamp. While viewing, the examiner applies gradual force to the lens until the end point is reached. Total force applied is shown in grams by the maximum indicator, with the pointer remaining at the end point until pressure is released.

The examiner can hold this easy-to-use instrument in one hand while performing the measurement, with no need for an assistant. Both patient and examiner may remain in one position for examination of both eyes.



Yablonski Fundus Lens
Ophthalmodynamometer

Features

- Two bi-directional index pointers — direct force indicator, and maximum force indicator which remains in place for measurement recording. Gauge is calibrated in grams.
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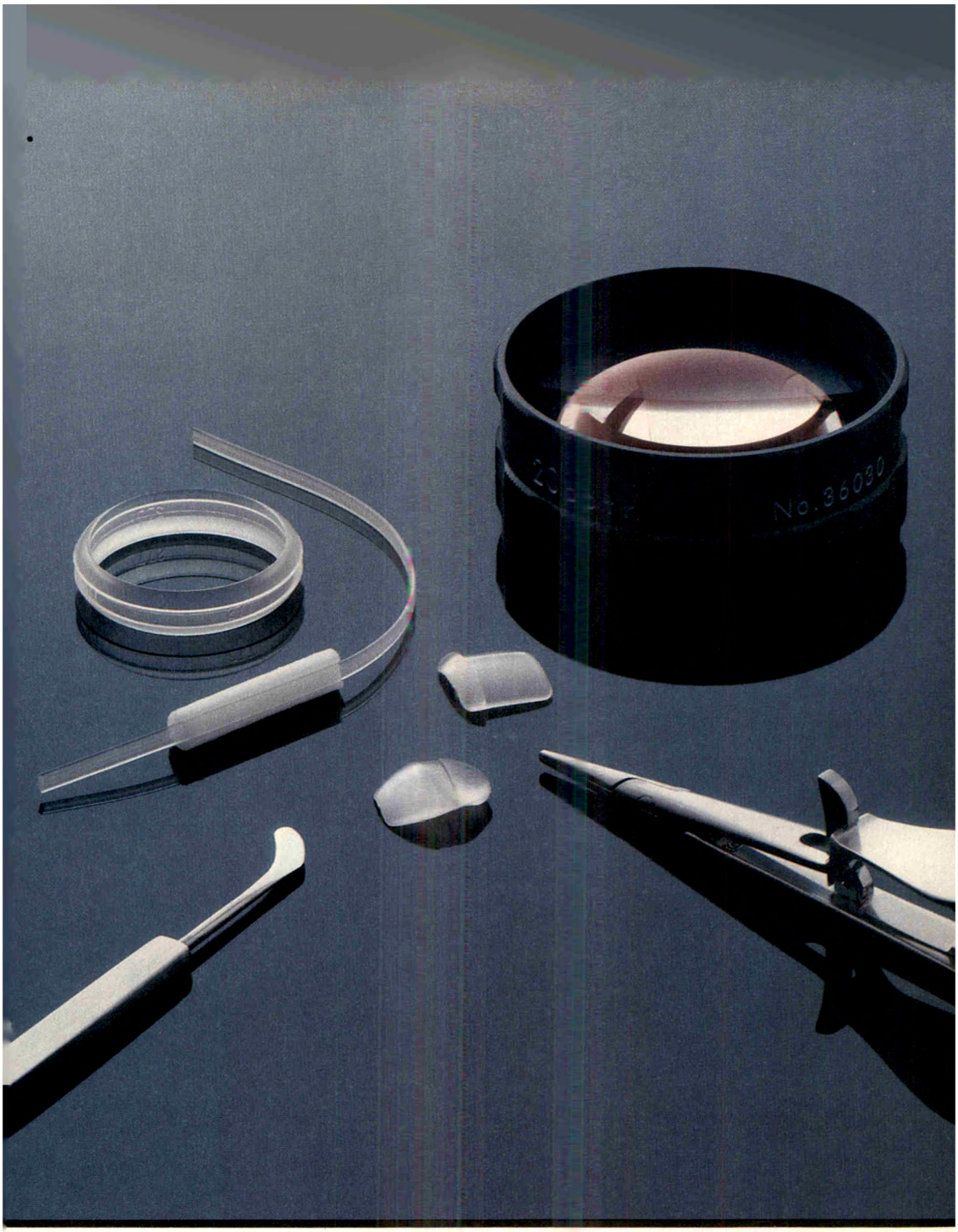
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and, the Wedge™, an implant developed to address the problems of the "fishmouth" phenomenon.

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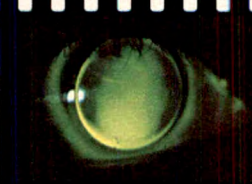
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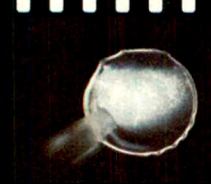
ew patient (has
ever worn lenses),
= 42.00/42.50,
3.00 D.



High astigmat,
cylindrical cornea,
K = 42.00/47.00,
– 4.00 D.



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post-op, K = 43.50/
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Soft lens with
protein/mineral
build-up, edge fluting;
patient complains
of poor vision.



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distorted, K =
58.00.

Now available in 8.5 & 9.5 mm diameters.

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Patients who have never worn lenses... patients who are tolerating compromised vision or annoying discomfort with their current lenses... highly astigmatic patients... aphakic patients... outright hard or soft lens failures... all are ideal candidates for POLYCON lenses.

POLYCON lenses add the important benefits of oxygen permeability to the desirable characteristics of PMMA lenses, to provide crisp, clear vision and avoid many of the problems associated with lens wear, particularly those due to corneal edema such as spectacle blur, limited hours of wear and discomfort.

That's because POLYCON lenses are made from a material specifically designed for contact lenses which contains silicone and methyl methacrylate, producing a lens that is thin, stable, durable and wettable... a lens with excellent optics which also provides oxygen transmission, virtually eliminating corneal edema.[1,2,3]

So you no longer have to choose between clear vision and clear corneas. Join the growing number of practitioners who are choosing POLYCON lenses, and offer your patients both.

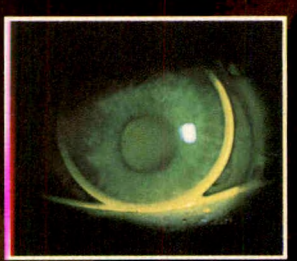
SYNTEX

SYNTEX OPHTHALMICS, INC.
PHOENIX, ARIZONA 85021



PMMA patient, complains of flare, glare and associated night vision problems.

PMMA patient for 4 years, complains of spectacle blur, discomfort; central corneal clouding.



POLYCON®

(silafocon A) CONTACT LENS

The clear choice in rigid lenses

Please see following page for a summary of prescribing information.

POLYCON®
(silafocon A) CONTACT LENS

Brief Summary
(For full prescribing information, see package insert.)

INDICATIONS
The POLYCON contact lens is indicated for the correction of visual acuity for persons with nondiseased eyes having ametropias, including those with high degrees of corneal or refractive astigmatism, as well as aphakia.

CONTRAINDICATIONS
Acute and subacute inflammations of the anterior segment of the eye; any eye disease which affects the cornea or conjunctiva; insufficiency of lacrimal secretion; corneal hypoesthesia; or any systemic disease which may affect the eye or be exacerbated by wearing contact lenses.

WARNINGS
(1) If the lens becomes less comfortable to the wearer than when first placed on the wearer's cornea, a foreign body may be present. The patient should remove the lens immediately. If discomfort persists, an eye care practitioner should examine the patient as soon as possible to determine if any corneal abrasion, ulceration, irritation or infection is present.

(2) Aphakic patients should not be fitted during the postoperative period until the surgeon determines that the eye has healed completely.

(3) Only use Preflex® Sterile Cleaning Solution, Flex-Care® Sterile Solution, and Adapettes® Sterile Lubricating Solution with the POLYCON lens. Store ONLY in Flex-Care® Sterile Solution. Patients must adhere to recommended daily lens care regimen. FAILURE TO DO SO MAY RESULT IN DEVELOPMENT OF SERIOUS OCULAR INFECTIONS.

(4) Wearers must not use any medicaments or ophthalmic solutions other than Adapettes® solution prior to inserting the lens or while the lens is on the eye.

(5) Remove before sleeping or in the presence of noxious and irritating vapors.

PRECAUTIONS
THE POLYCON CONTACT LENS MUST BE BOTH CLEANED AND DISINFECTED DAILY. ONE PROCEDURE DOES NOT REPLACE THE OTHER. To disinfect the lens, it is stored in fresh Flex-Care® solution for a minimum of four hours daily in the patient lens storage case. After removal of the lens from the storage case, rinse the lens thoroughly with Flex-Care® solution and apply Adapettes® lubricating solution to the lens surfaces as a wetting agent prior to inserting the lens in the eye.

ADVERSE REACTIONS
Rainbows or halos around objects or blurring of the vision. Excessive tearing, unusual eye secretions, and photophobia.

Preflex,® Flex-Care® and Adapettes® are trademarks of Burton Parsons & Company, Inc.

1-80

References

1 Multi-clinic study of 418 patients, data on file, Syntex Ophthalmics, Inc.

2 Korb JE, Finnemore VM: Corneal edema with polymethylmethacrylate vs. gas-permeable rigid polymer contact lenses of identical design. To be published.

3 Williams CE: New design concepts for permeable rigid contact lenses. *JAOA* 50:331-336, 1979.

SYNTEX

SYNTEX OPHTHALMICS, INC.
PHOENIX, ARIZONA 85021

DMV



DMV Remover
Hard lenses



DMV II (Vented)
Hard lenses



S.L.R.
Soft lens
Remover / Inserter

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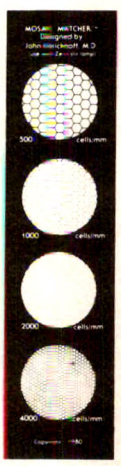
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Just match the mosaic seen with the smallest (flare) beam of the slit lamp with the similar mosaic on the MOSAIC MATCHER.

- So simple, fast, inexpensive—use it routinely for **implant**, cataract, and corneal cases.
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Send $20 check for complete instructions for viewing the mosaic and laminated chart to:

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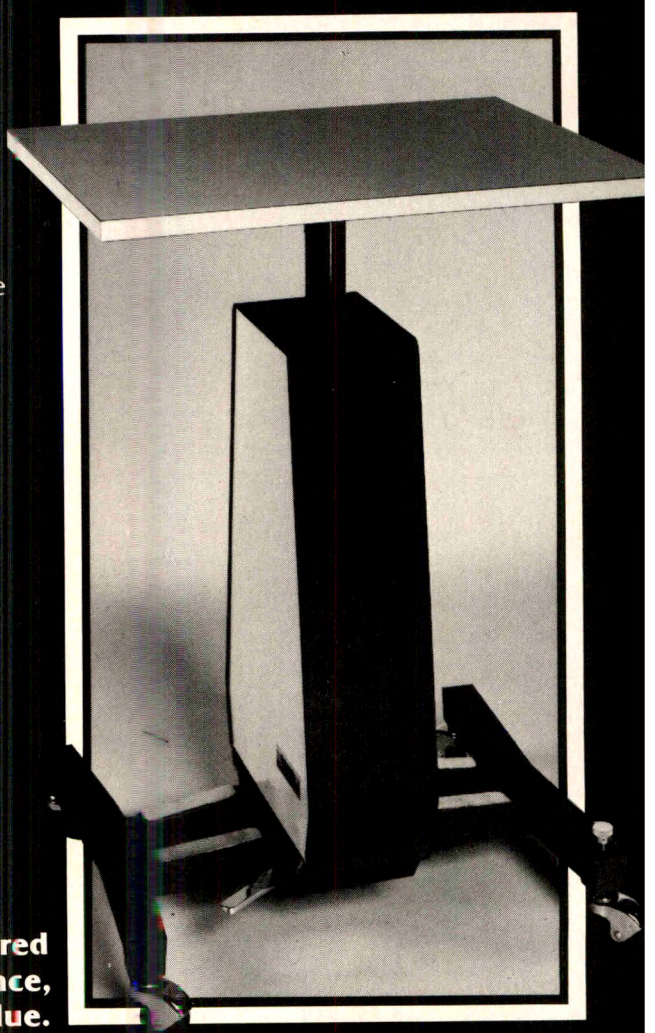
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Sound like your office?

Well, Acuity Systems, the leader in advanced instrumentation and systems for health care professionals, has come up with a way for you to streamline your office and make it more productive.

Introducing the Intelligent Office™: the first total system of practice management.

The Intelligent Office is a three-part, computer-based approach to running your practice. Whether you use just one of the modules, two, or all three, it makes your working day more efficient and more profitable.

It also lets you give better patient care, and enjoy your work more.

What's more, the Intelligent Office saves hours of staff time. And since that time can be devoted to other tasks, everyone in your office will become more productive.

The Acuity Systems office computer is the smart way to handle paperwork.

The Acuity Systems office computer handles all the routine, repetitive paperwork that ties up both your front office staff and you. Your secretary can operate it easily, because it actually prompts her with simple, step-by-step instructions. And it's completely contained in your office, so only you have access to confidential patient and practice information.

Acuity Systems' office computer keeps complete patient accounts, processes insurance forms, and schedules appointments. At the touch of a button, it produces itemized bills, appointment lists, and labels for appointment reminder postcards. And it also generates valuable management reports, detailing aging accounts, trial balances, a variety of statistical practice analyses, and more.

What's more, the office computer also functions as a word processor.

In this mode, it can generate personalized form letters for referral replies, insurance inquiries, patient recalls, and other repetitive correspondence.

As a result, paperwork flows through your office faster, saving staff time. Your cash flow improves, because patients receive bills on the spot and insurance claims are processed sooner. And you have the management information you need to make intelligent business decisions.



The Acuity Systems office computer uses a keyboard display terminal, printer, and disc memory storage to handle the paperwork that ties up your front office.

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Acuity Systems' objective Auto-Lensmeter is the standard of the industry. It cuts neutralization time to one second per lens, yet it's so simple to use that anyone on your staff can handle it. A patented alignment system makes finding the optical center foolproof. Then, push a few buttons, and it prints out the complete prescription.

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lenses. And your staff will be able to isolate the cause of a patient's complaint while he waits, avoiding no-fee re-examinations.

Our Rx1 Auto-Refractor® saves time and improves patient care.

The Rx1 Auto-Refractor dramatically reduces refraction time per patient—even for children and aphakics. In seconds, it gives you an accurate, objective printout of sphere, cylinder, axis, and IPD measurements—everything you need to write your prescription.

Since the Rx1 Auto-Refractor is completely operator objective, it can be operated by anyone in your office. That means you'll have valuable extra hours in every day to devote to other patients, or to seeing new patients.



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What's more, your patients will appreciate your using the most modern instrumentation to give them superior eye care. They'll be less anxious about making the wrong decision, because the Rx1 Auto-Refractor simplifies the subjective refraction decision-making process. So they'll walk out of your office feeling satisfied—and ready to refer their families and friends.

We've helped thousands of doctors solve their office problems.

The Intelligent Office is a lot more than sophisticated electronics.

It also represents everything that Acuity Systems has learned from working with thousands of doctors over the past 10 years. And from the moment you contact us, we put that knowledge to work for you.



The Rx1 Auto-Refractor gives you sphere, cylinder, axis, and IPD readings—everything you need to write your prescription. And this new design requires less space in your office.

First, our Practice Management Analyst takes a long, hard look at the way your office works, and then suggests needed improvements. He can tell you which parts of the Intelligent Office system you need. And he may surprise you by suggesting other things, like a change in patient traffic flow, or a reassignment of staff duties.

Then, when you buy any of the Intelligent Office modules, an Acuity Systems representative will come to your office to install it and teach your people how to use it. And he'll check back with you periodically, to train new employees and make sure you're getting the most from your equipment.

Finally, there's Acuity Systems' nationwide service network. It assures you that, should you ever have a problem, one of our experts will be right there to help you.

Stop letting your office run you.

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2. I do approximately _____ refractions a week.
3. I have _____ exam lanes and _____ assistants.
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GLAUCOMA, 2nd ed. *Edited and with contributions by* PAUL A. CHANDLER, M.D., *Massachusetts Eye and Ear Infirmary, Boston, Massachusetts; and* W. MORTON GRANT, M.D., *Harvard Medical School, Boston, Massachusetts. (8 Contributors).* Now completely updated to provide recent findings and up to date techniques and procedures, this new edition is an important text for effective solutions to problems concerning glaucoma that are seen in daily practice. Unusually clear instruction and practical guidance for the recognition and management of the many forms of glaucoma are the hallmarks of this exceptionally fine book. Reviews of previous edition: "The long awaited masterwork...a classic."—*New England Journal of Medicine.* "Recommended enthusiastically."—*American Journal of Ophthalmology. 381 pp. (7 x 10), illus., 1979, $26.00.*

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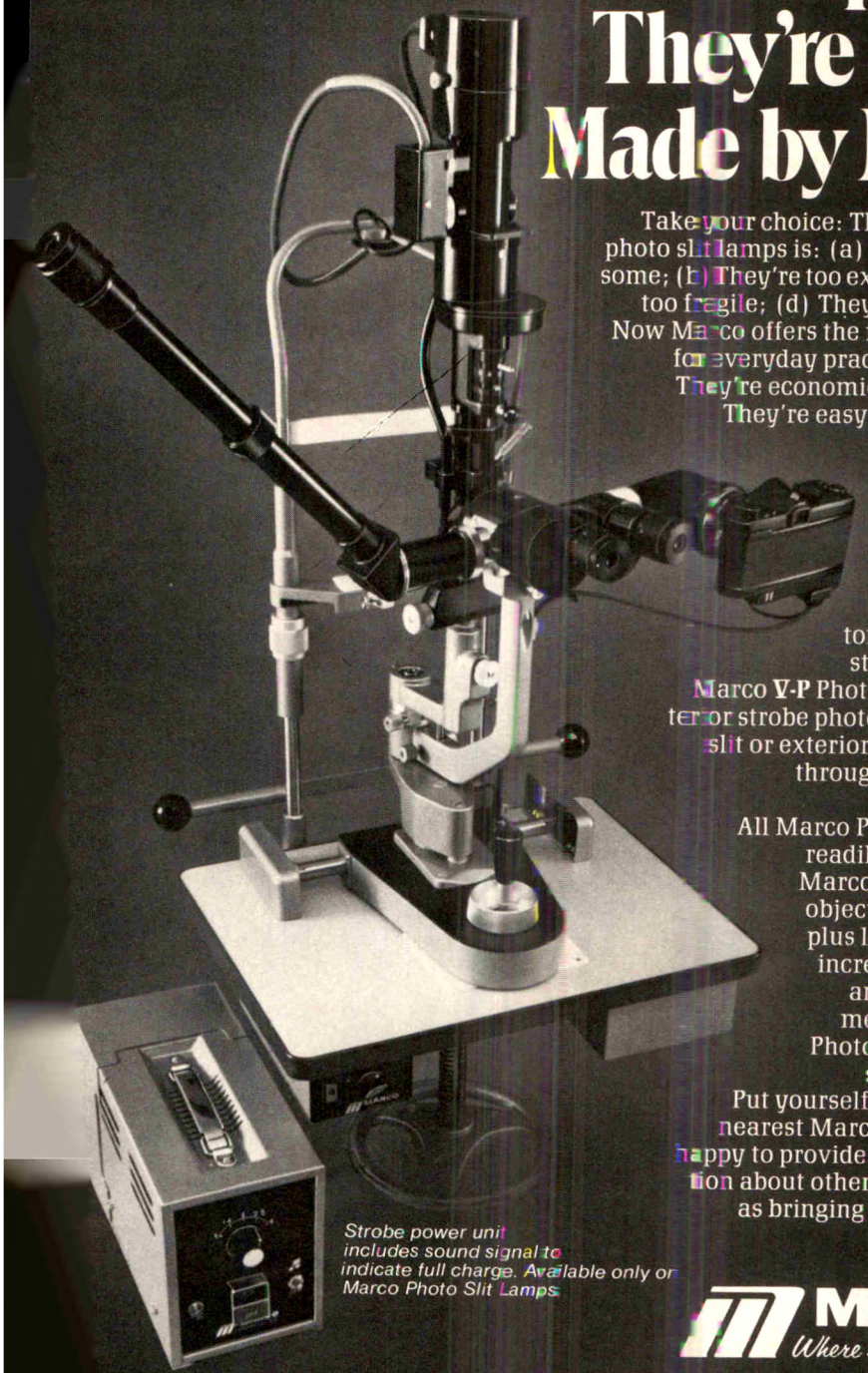
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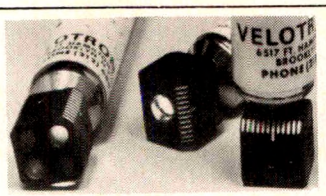
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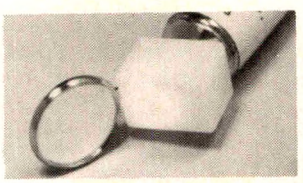
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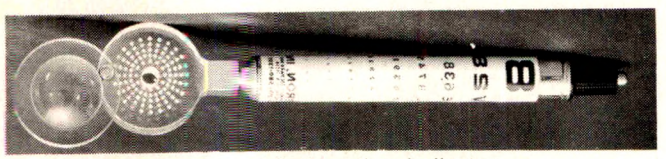
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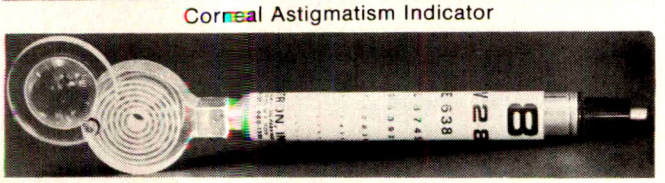
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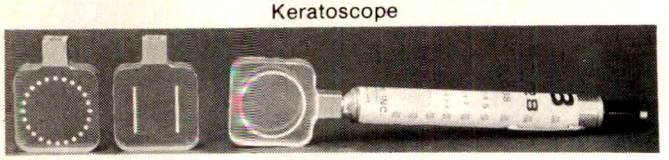
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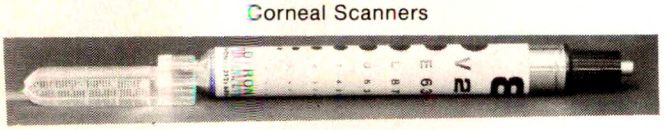
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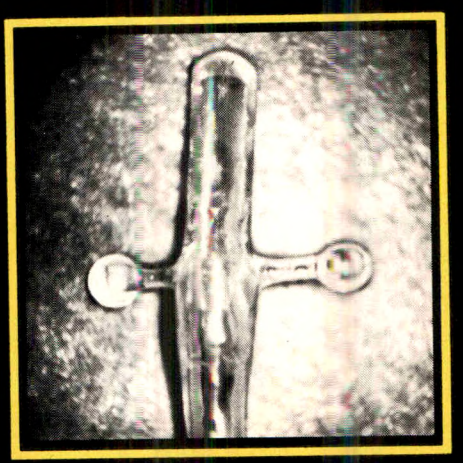


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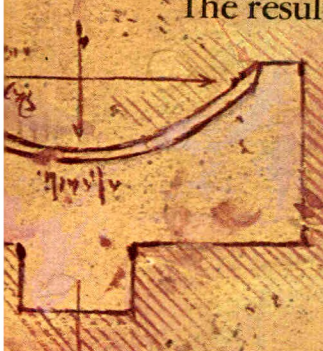
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Bausch & Lomb Scientific Update:

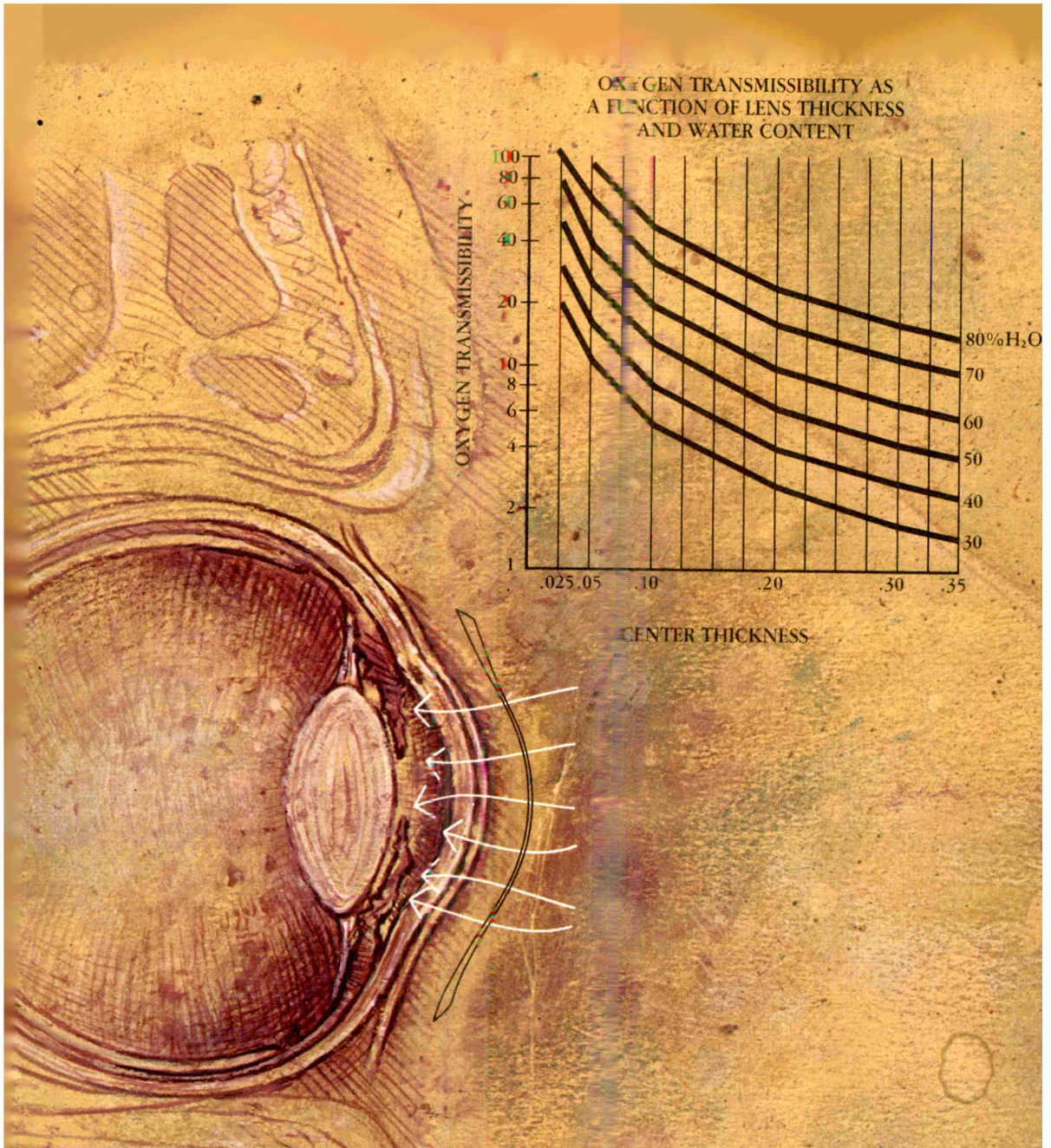
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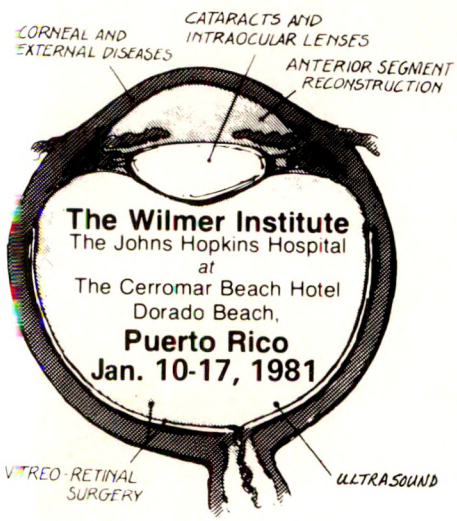
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BRIEF SUMMARY (For full prescribing information see package insert.)
VISION CORRECTION USE

INDICATIONS: SOFLENS Contact Lenses are indicated for vision correction use in persons with non-diseased eyes and in aphakia. **CONTRAINDICATIONS:** SOFLENS Contact Lenses are contraindicated in the presence of any active untreated infection or abnormality of the anterior segment of the eye. **WARNINGS: Abrasions and Infections**—If a lens becomes less comfortable than it was when first placed on the wearer's cornea, the lens should be removed immediately and the wearer's eye and the lens examined for the possible presence of a foreign body. If any eye abrasion, ulceration, irritation or infection is present or any abnormal eye condition is observed concurrent with lens wear, the lens should be removed immediately and a physician consulted. **Aphakic Patients:** Aphakic patients should not be fitted with SOFLENS Contact Lenses during the postoperative period until, in the opinion of the surgeon, the eye has healed completely. **Lens Care and Handling:** Persons who require only vision correction and who would not, or could not adhere to the recommended daily care of SOFLENS Contact Lenses or who are unable to place and remove the lenses should not be provided with them. Failure to follow handling and cleaning instructions could lead to serious eye infections which might result in corneal ulcers. Malfunction and rusting of the metal interior of the BAUSCH & LOMB™ Disinfecting Unit I (ASEPTOR®) as well as discoloration and cracking of the lens case has been reported after varying periods of use. If such occurs, appropriate replacement is indicated to avoid interference with the disinfection procedure. Keep the BAUSCH & LOMB™ Disinfecting Unit I (ASEPTOR®) and II (ASEPTRON™) clean and dry at all times. **Medicants and Eye Drops:** When the lenses are used by persons requiring only vision correction no ophthalmic solutions or medicants should be used. No conventional (hard) contact lens solutions and eye drops, except BAUSCH & LOMB™ Lens Lubricant should be used by SOFLENS Contact Lens wearers prior to placement, or while the lens is in place, on the eye. Also, no solutions, including conventional (hard) contact lens solutions should be used. Only daily cleaner, lens lubricant, saline and the solution made from the SOFLENS™ Enzymatic Contact Lens Cleaning Tablets are to be used on a SOFLENS Contact Lens when the lens is off the eye. **Wearing Restrictions:** SOFLENS Contact Lenses when used only for vision correction should be removed before sleeping or swimming and in the presence of noxious and irritating vapors. Insufficient lacrimal secretions or hypoesthesia may be restricting factors to the wearing of soft contact lenses. **PRECAUTIONS: Storage**—SOFLENS Contact Lenses must be stored ONLY in saline solution or BAUSCH & LOMB™ Sterile Disinfecting Solution. If left exposed to air, the lenses will dehydrate, become brittle, and break readily. If a lens dehydrates, it should be resoaked in saline solution or BAUSCH & LOMB™ Sterile Disinfecting Solution until it returns to its soft, pliable state which may take as long as forty minutes. Saline solution must be used DAILY for cleaning, storing, and disinfection of lenses. The carrying case must be emptied and refilled with fresh saline solution just before disinfecting the lenses. **Lens Handling:** Hands must be washed, rinsed thoroughly, and dried with a lint-free towel before handling the lenses. Cosmetics, lotions, soaps, and creams must not come in contact with the lenses since eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until the hair spray has settled. A SOFLENS Contact Lens may be damaged by nicking or tearing if care is not exercised during placement on or removal from the eye, replacing or removing it from the carrying case or in the cleaning process. **Disinfecting:** Saline solution must be used DAILY for cleaning, storing, and disinfection of lenses. After removal from the eye, the SOFLENS Contact Lens must be rubbed gently with Daily Cleaner then rinsed with saline solution to remove mucus and film from the lens surface. The BAUSCH & LOMB™ Lens Carrying Case (LENSGARD™) must be emptied and refilled ⅔ full with fresh saline solution just before disinfecting the lenses and must always be tightly closed to prevent leakage of saline from the case and subsequent dehydration of the lenses. In order to remove protein deposits which may form on the lenses, wearers should use the SOFLENS™ Enzymatic Contact Lens Cleaning Tablets according to the directions for use which accompany the tablets. To help remove tear residues and mucus as well as other deposits that tend to accumulate on the lens surface, patients should use Daily Cleaner according to the directions for use which accompany the product. Disinfecting with a BAUSCH & LOMB™ Disinfecting Unit I (ASEPTOR®) or II (ASEPTRON™) is necessary to kill microorganisms. If a BAUSCH & LOMB™ Disinfecting Unit I (ASEPTOR®) or II (ASEPTRON™) is not available, lenses may be disinfected by immersing your lenses in BAUSCH & LOMB™ Saline Solution in the carrying case and dropping the sealed case into a pan of already boiling water. Remove the pan from heat after ten minutes and allow the water to cool before removing your lenses. When this boiling method of disinfection is used, the lenses can be damaged if the boiling water is allowed to completely evaporate. Chemical disinfection with BAUSCH & LOMB™ Sterile Disinfecting Solution has been tested microbiologically and shown to be an effective disinfection procedure for the SOFLENS hydrophilic contact lens. If chemical disinfection is used the SOFLENS hydrophilic contact lens must be cleaned and rinsed daily with BAUSCH & LOMB™ Sterile Daily Cleaner and BAUSCH & LOMB™ Sterile Disinfecting Solution. The lens carrying case must be emptied and refilled with fresh BAUSCH & LOMB™ Sterile Disinfecting Solution prior to disinfecting the lens. Fresh BAUSCH & LOMB™ Sterile Disinfecting Solution must be used daily for storing and disinfecting the lens. **WARNING:** BAUSCH & LOMB™ STERILE DISINFECTING SOLUTION SHOULD NOT BE USED WITH HEAT. **Fluorescein:** Never use fluorescein while the patient is wearing the lenses because the lenses will become discolored. Whenever fluorescein is used, flush the eyes with sterile normal saline solution and wait at least one hour before replacing the lenses. Earlier replacement may cause the lenses to absorb residual fluorescein. **ADVERSE REACTIONS:** Serious corneal damage may result from wearing a SOFLENS Contact Lens which has been soaked in a conventional contact lens solution. Eye irritation may occur within a short time after putting on a hypertonic lens. Removal of the lens will relieve the irritation. A lens adheres very rarely to an eye as a result of sleeping with the lens on, or wearing a hypotonic lens. If a lens adheres for any reason, apply fresh saline and wait until the lens moves freely before removing it. Clinical studies indicate that corneal edema, as manifested by symptoms such as rainbows, halos around lights, or foggy vision, may occur in less than 5% of SOFLENS Contact Lens wearers. If these symptoms occur, the lenses should be removed and professional consultation obtained. Excessive tearing, unusual eye secretions, and photophobia are not normal. If these symptoms occur, the patient should be examined to determine their cause. Neovascularization of the cornea has been observed in some aphakic patients fitted with the SOFLENS Contact Lens, which may require discontinuation of the lens. Medical consultation should be obtained in such an instance.

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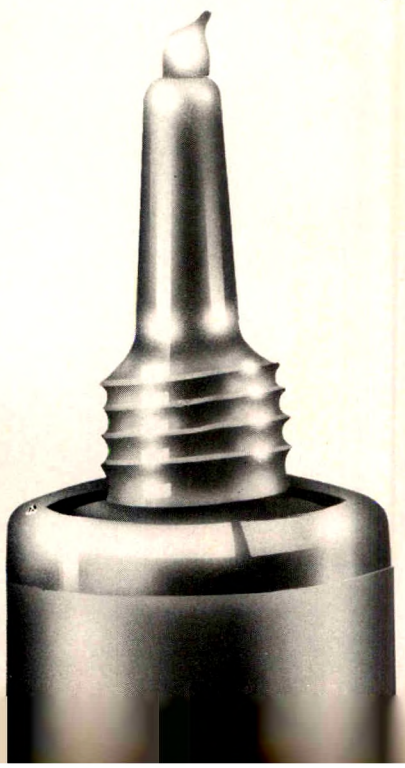
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PRECAUTIONS: Extended ophthalmic use of topical steroid therapy may result in glaucoma with possible damage of the optic nerves, posterior subcapsular cataract formation, or may aid in the establishment of secondary ocular infections from fungi or viruses liberated from ocular tissues. It is advisable that intraocular pressure be checked frequently. In those diseases causing thinning of the cornea, perforation has been known to occur with the use of topical steroids. As with any antibiotic preparation, prolonged use may result in the overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

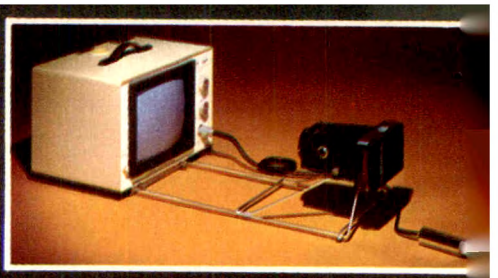
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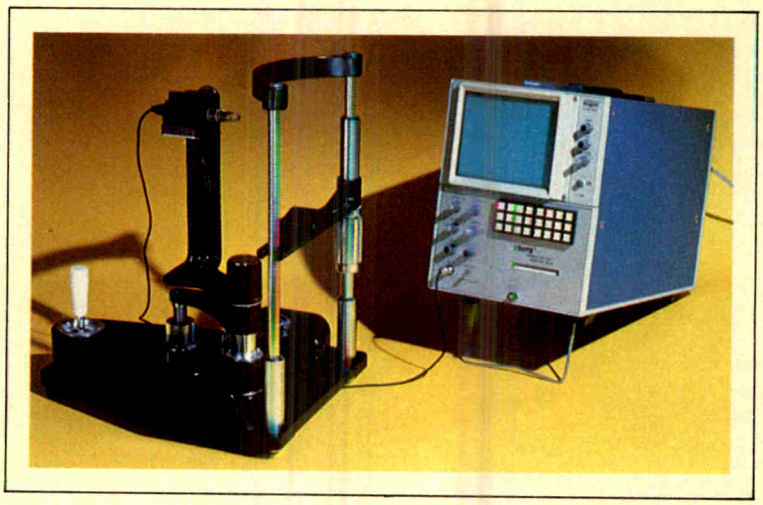
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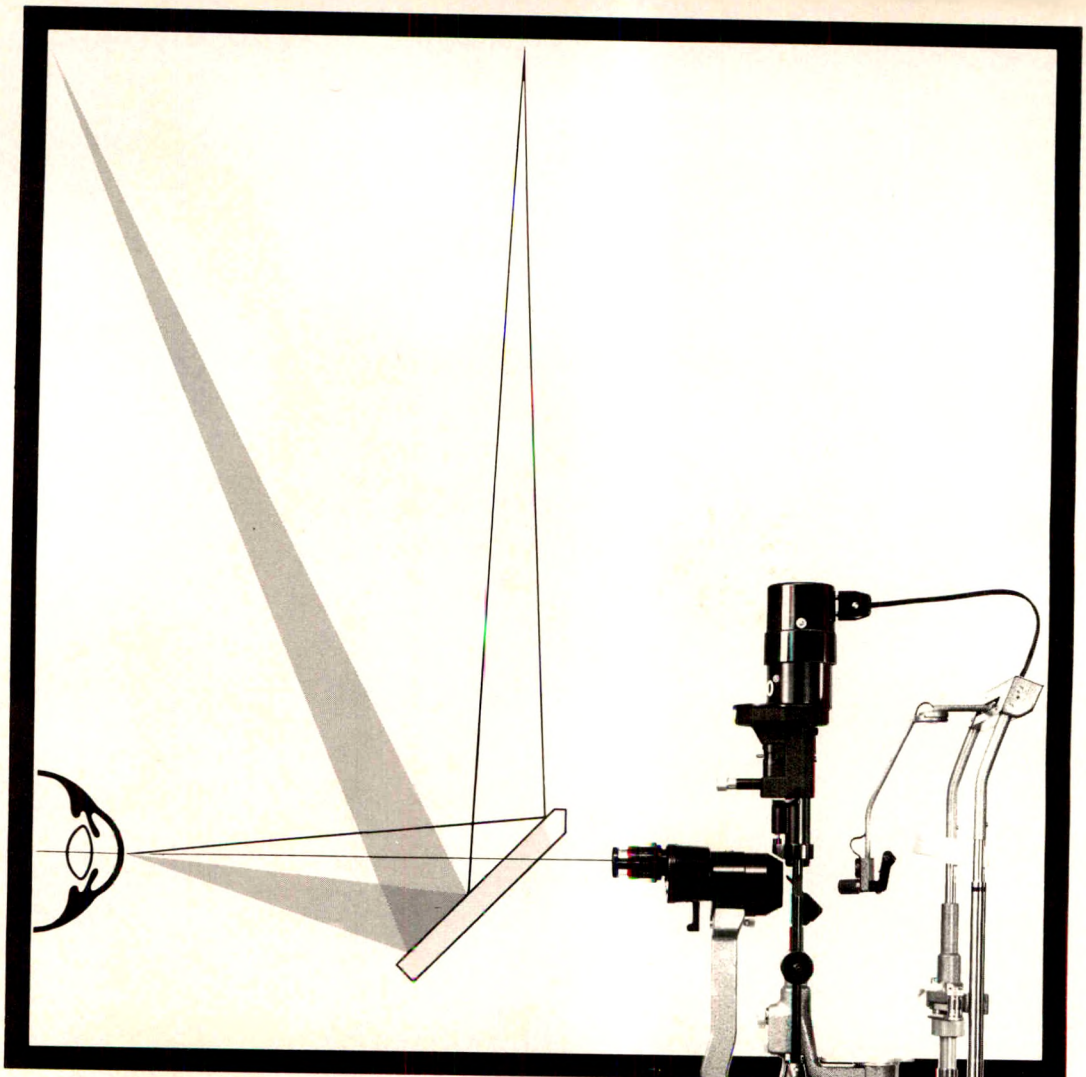
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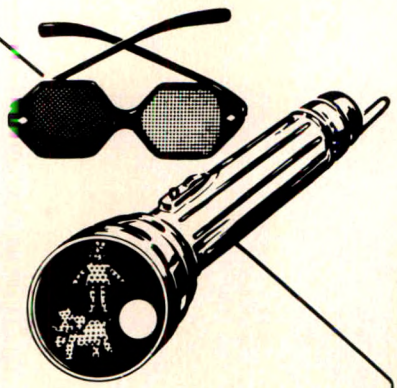
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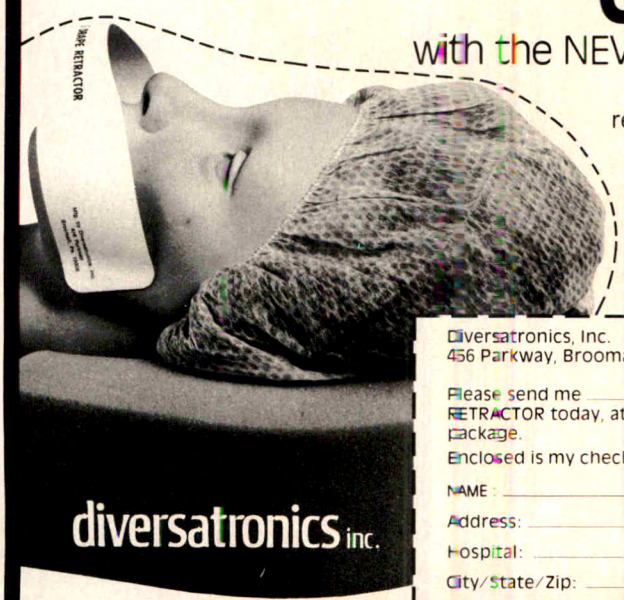
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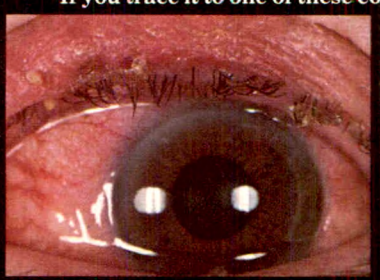
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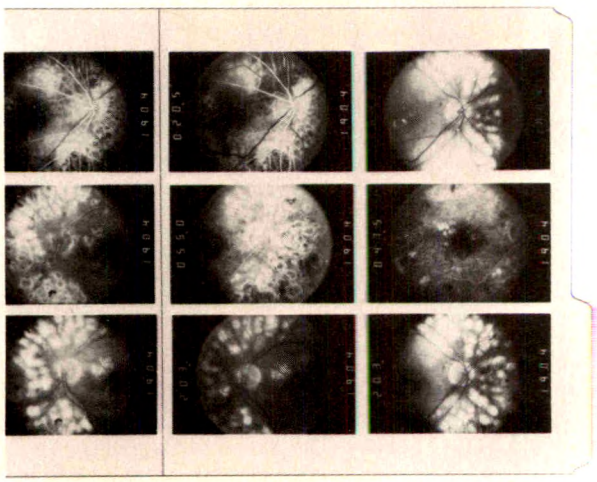
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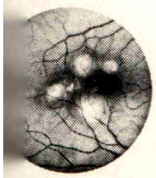
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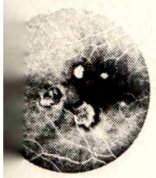
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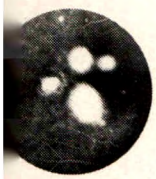
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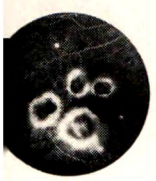




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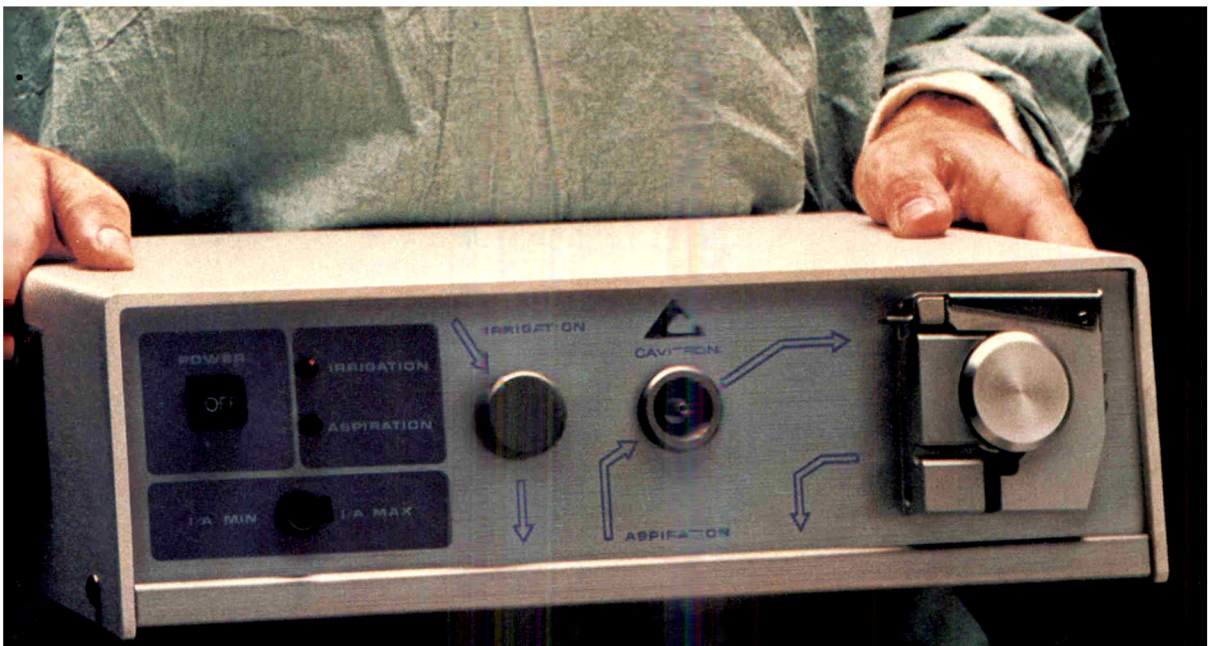


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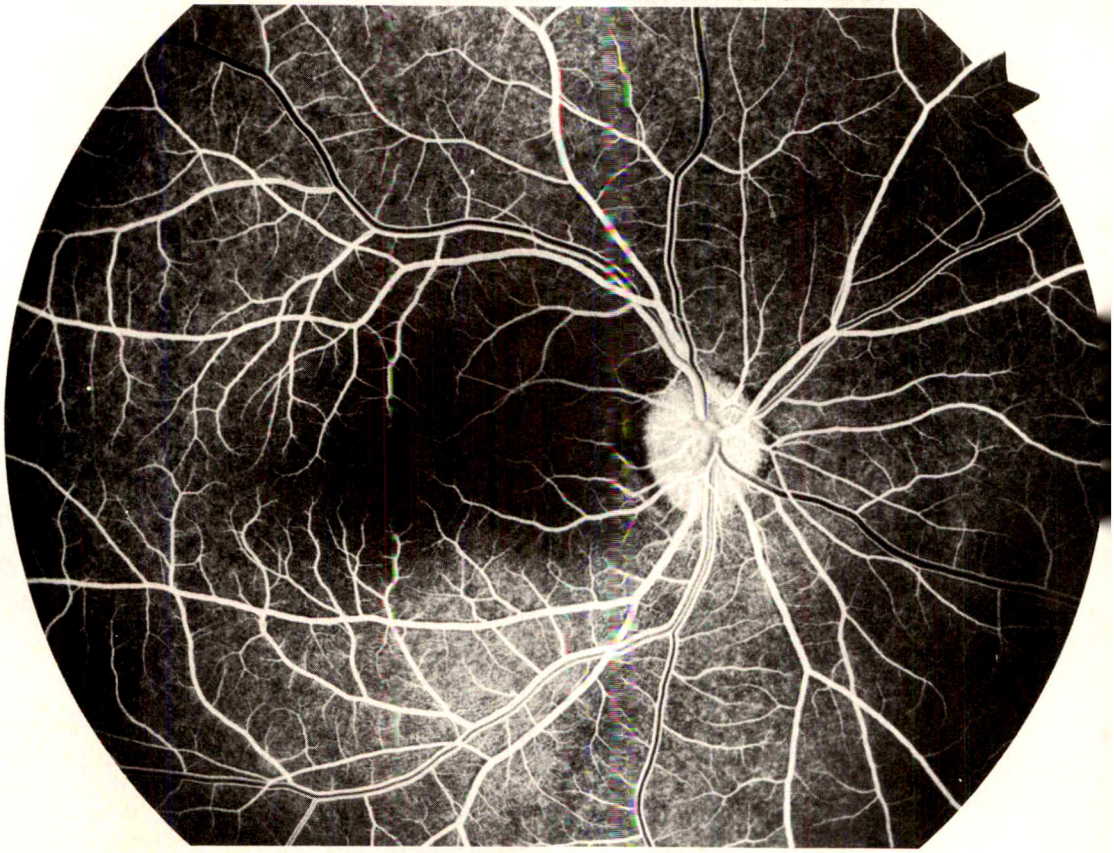
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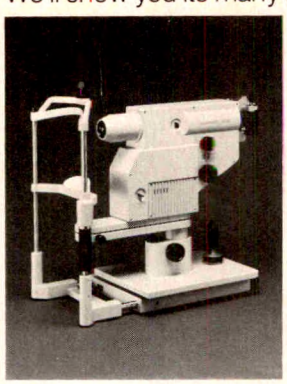
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Description. Vira-A (vidarabine, adenine arabinoside, or Ara-A) is an antiviral drug for topical ophthalmic administration. The chemical name is 9-β-D-arabinofuranosyladenine. Each gram of the ophthalmic ointment contains 30 mg of vidarabine monohydrate equivalent to 28.11 mg of vidarabine in a sterile, inert, petrolatum base.

Clinical Pharmacology. Vira-A is a purine nucleoside obtained from fermentation cultures of *Streptomyces antibioticus*. Vira-A possesses *in vitro* and *in vivo* antiviral activity against Herpes simplex types 1 and 2, Varicella-Zoster, and Vaccinia viruses. Except for Rhabdovirus and Oncornavirus, Vira-A does not display *in vitro* antiviral activity against other RNA or DNA viruses, including Adenovirus.

The antiviral mechanism of action has not been established. Vira-A appears to interfere with the early steps of viral DNA synthesis. Vira-A is rapidly deaminated to arabinosylhypoxanthine (Ara-Hx), the principal metabolite. Ara-Hx also possesses *in vitro* antiviral activity but this activity is less than that of Vira-A. Because of the low solubility of Vira-A, trace amounts of both Vira-A and Ara-Hx can be detected in the aqueous humor only if there is an epithelial defect in the cornea. If the cornea is normal, only trace amounts of Ara-Hx can be recovered from the aqueous humor.

Systemic absorption of Vira-A should not be expected to occur following ocular administration and swallowing lacrimal secretions. In laboratory animals, Vira-A is rapidly deaminated in the gastrointestinal tract to Ara-Hx.

In contrast to topical idoxuridine, Vira-A demonstrated less cellular toxicity in the regenerating corneal epithelium of the rabbit.

Indications and Usage. Vira-A Ophthalmic Ointment, 3%, is indicated for the treatment of acute keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus types 1 and 2. Vira-A is also effective in superficial keratitis caused by Herpes simplex virus which has not responded to topical idoxuridine or when toxic or hypersensitivity reactions to idoxuridine have occurred. The effectiveness of Vira-A Ophthalmic Ointment, 3%, against stromal keratitis and uveitis due to Herpes simplex virus has not been established.

The clinical diagnosis of keratitis caused by Herpes simplex virus is usually established by the presence of typical dendritic or geographic lesions on slit-lamp examination. In controlled and uncontrolled clinical trials, an average of seven and nine days of continuous Vira-A Ophthalmic Ointment, 3%, therapy was required to achieve corneal re-epithelialization. In the controlled trials, 70 of 81 subjects (86%) re-epithelialized at the end of three weeks of therapy. In the uncontrolled trials, 101 of 142 subjects (71%) re-epithelialized at the end of three weeks. Seventy-five percent of the subjects in these uncontrolled trials had either not healed previously or had developed hypersensitivity to topical idoxuridine therapy.

The following topical antibiotics: gentamicin, erythromycin, chloramphenicol; or topical steroids: prednisolone or dexamethasone, have been administered concurrently with Vira-A Ophthalmic Ointment, 3%, without an increase in adverse reactions.

Normally, corticosteroids alone are contraindicated in Herpes simplex virus infections of the eye. If Vira-A Ophthalmic Ointment, 3%, is administered concurrently with topical corticosteroid therapy, corticosteroid-induced ocular side effects must be considered. These include corticosteroid-induced glaucoma or cataract formation and progression of a bacterial or viral infection.

Vira-A is not effective against RNA virus or adenoviral ocular infections. Vira-A is also not effective against bacterial, fungal, or chlamydial infections of the cornea or nonviral trophic ulcers.

Contraindication. Vira-A Ophthalmic Ointment, 3%, is contraindicated in patients who develop hypersensitivity reactions to it.

Warnings. ***Use in Pregnancy:*** Vira-A (vidarabine) parenterally is teratogenic in rats and rabbits. Ten percent Vira-A ointment applied to 10% of the body surface during organogenesis indu fetal abnormalities in rabbits. When 10% Vira-A ointment was plied to 2 to 3% of the body surface of rabbits, no fetal ab malities were found. This dose greatly exceeds the total rec mended ophthalmic dose in humans. The possibility of embry or fetal damage in pregnant women receiving Vira-A (vidara ophthalmic ointment), 3%, is remote. The topical ophthalmic d is small, and the drug relatively insoluble. Its ocular penetratic very low. However, a safe dose for a human embryo or fetus not been established. Consequently, Vira-A should be used when clearly indicated.

It is not known whether Vira-A is excreted in human milk. general rule, because many drugs are excreted in human r nursing should not be undertaken while a patient is under tr ment. However, excretion of vidarabine in breast milk is unli because Vira-A is rapidly deaminated in the gastrointestinal t

Mutagenic Potential: Results of *in vitro* experiments indic that vidarabine can be incorporated into mammalian DNA and induce mutation in mammalian cells (mouse L5178Y cell li Thus far, *in vivo* studies have not been as conclusive, but ther some evidence (dominant lethal assay in mice) that vidarat may be capable of producing mutagenic effects in male g cells.

It has also been reported that vidarabine causes chromosc breaks and gaps when added to human leukocytes *in vitro*. W the significance of these effects in terms of mutagenicity is fully understood, there is a well-known correlation between ability of various agents to produce such effects and their ab to produce heritable genetic damage.

Oncogenic Potential: Chronic parenteral (IM) studies vidarabine have been conducted in mice and rats.

In the mouse study, there was a statistically significant crease in liver tumor incidence among the vidarabine-trea females. In the same study, some vidarabine-treated male m developed kidney neoplasia. No renal tumors were found in vehicle-treated control mice or the vidarabine-treated fem mice.

In the rat study, intestinal, testicular, and thyroid neoplasia curred with greater frequency among the vidarabine-trea animals than in the vehicle-treated controls. The increases thyroid adenoma incidence in the high-dose (50 mg/kg) ma and the low-dose (30 mg/kg) females were statistically sigr cant.

Hepatic megalocytosis, associated with vidarabine treatme has been found in short- and long-term rodent (rat and mou studies. It is not clear whether or not this represents a p neoplastic change.

The recommended frequency and duration of administrat should not be exceeded (see Dosage and Administration).

Precautions. The diagnosis of keratoconjunctivitis due to Herp simplex virus should be established clinically prior to prescribi Vira-A Ophthalmic Ointment, 3%.

Patients should be forewarned that Vira-A Ophthalmic Oi ment 3%, like any ophthalmic ointment, may produce a tempora visual haze.

Although viral resistance to Vira-A has not been observed, th possibility may exist.

Adverse Reactions. Lacrimation, foreign-body sensation, cc junctival injection, burning, irritation, superficial puncta keratitis, pain, photophobia, punctal occlusion, and sensitiv have been reported with Vira-A Ophthalmic Ointment, 3%. Th following have also been reported but appear disease-relate uveitis, stromal edema, secondary glaucoma, trophic defects, cc neal vascularization, and hyphema.

Overdosage. Acute massive overdosage by oral ingestion of th ophthalmic ointment has not occurred. However, the rap deamination to arabinosylhypoxanthine should preclude ar difficulty. The oral LD50 for vidarabine is greater than 5,020 m kg in mice and rats. No untoward effects should result from inge tion of the entire contents of a tube.

Overdosage by ocular instillation is unlikely because any e cess should be quickly expelled from the conjunctival sac. To frequent administration should be avoided.

Dosage and Administration. Administer approximately one ha inch of Vira-A Ophthalmic Ointment, 3%, into the lower co junctival sac five times daily at three-hour intervals.

If there are no signs of improvement after 7 days, or comple re-epithelialization has not occurred by 21 days, other forms therapy should be considered. Some severe cases may requi longer treatment.

After re-epithelialization has occurred, treatment for an adc tional seven days at a reduced dosage (such as twice daily) recommended in order to prevent recurrence.

How Supplied. N 0071-3677-07 (Stock 18-1677-139)

Vira-A Ophthalmic Ointment, 3%, is supplied sterile ophthalmic ointment tubes of 3.5 g. The base is a 60:40 mixtu of solid and liquid petrolatum. U

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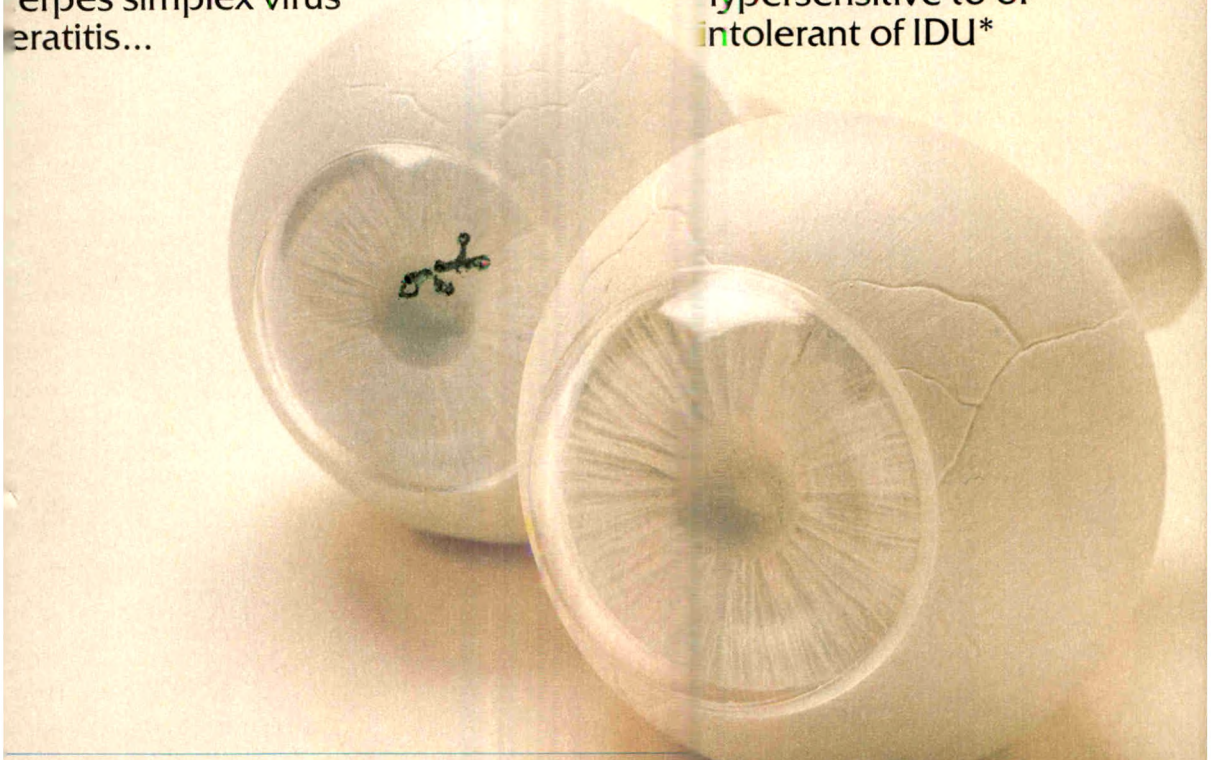
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linical Evidence	No. of Subjects	Description	Effective Response
ira-A ontrolled Trials	81	Previously untreated	70 subjects (86%)
ira-A ncontrolled Trials	142	75% had been resistant or hypersensitive to IDU	101 subjects (71%)

OTE: In the controlled and uncontrolled trials, 70 and 101 subjects, espectively, re-epithelialized at the end of three weeks on Vira-A.

ata on file, Medical Department, Parke-Davis

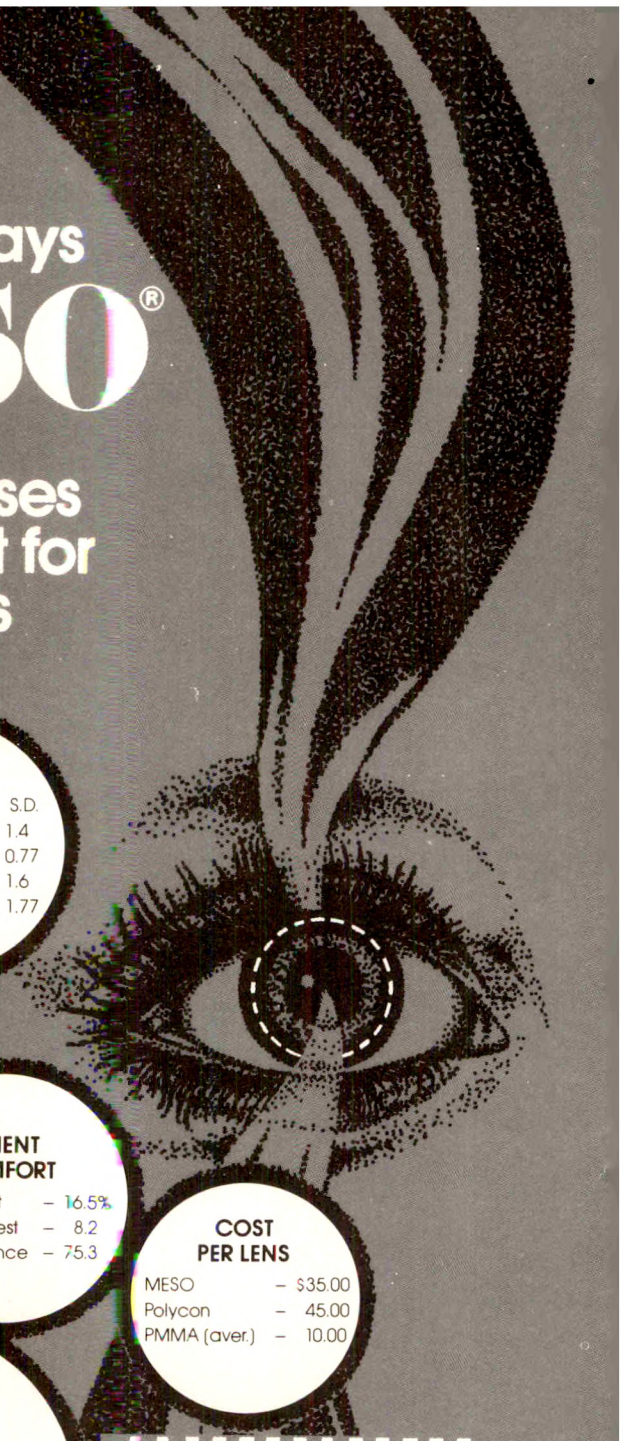
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Polycon	no	no	yes
PMMA	yes	yes	yes
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ription: The MESO (cabufocon) contact lens is composed of the polymer ose acetate butyrate.

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- Wetting angle: 46.4° dry, and 26.9° after soaking for 24 hours. (measured using the Poster Bubble Chamber technique)
- Water absorption: 1.8% by weight (ASTM D570)

he water content of the MESO (cabufocon) material when fully hydrated is 1.8% o the polymer's free hydroxyl and carbonyl content.

on: The MESO (cabufocon) contact lens, when placed on the human cornea, as a refracting medium to correct myopia, hyperopia, astigmatism and aphakia.

ation: The MESO (cabufocon) contact lens is indicated for the correction of l acuity in non-diseased eyes and aphakia.

ra-indication: MESO (cabufocon) contact lenses are contraindicated in the ence of any of the following conditions:

1. Active conjunctivitis.
2. Active or chronic trachoma infection.
3. Active herpes infection.
4. Any condition which has led to extensive corneal neovascularization.
5. Corneal hypothesia except that resulting from aphakia.
6. Any situation in which the wearer is unable to maintain good lens care regimen.

hings: The MESO (cabufocon) contact lens must be stored only in Flex-Care or ol solution.* No other storage solutions are recommended. Patients must adhere to ecommended daily care regimen. Failure to do so may result in ocular infection. f the lens becomes more uncomfortable to the wearer than when first placed on ye the lens should be removed, checked for dirt or scratches, and cleaned. If this dition persists, or if the eye becomes red or painful, the lens should be removed ediately. If symptoms persist, your eye-care practitioner should be consulted. The MESO (cabufocon) contact lens should be removed before sleeping or ming and should not be worn in the presence of noxious fumes or vapors. If ual blurring occurs, the lens should not be worn until the condition subsides.

autions:

care – Storage: When the MESO (cabufocon) contact lens is removed from the it should be stored only in Flex-Care or Flexsol sterile soaking solution after ning with Preflex* and rinsing with Flex-Care or Normol solution.* The lens should be pletely covered with the storage solution. Fill the lens case receptacle till just w the rim.

Before placing the MESO (cabufocon) lens on the eye, rinse the lens with Flex-Care ormol solution. Discard the old rinsing solution in the case, rinse the case with -Care or Normol, and dry the case. Refill with fresh storage solution each time the is stored.*

The MESO (cabufocon) lens, unlike the hydrophilic soft lens, does not dry out or ome brittle if exposed to air. If the lens does become dry, it should be soaked in age solution for a minimum of four hours before reinsertion on the eye.

Fluorescein may be used with the lens.

s cleaning: Ordinary mucous and oily deposits are removed with daily cleaning he lens with Preflex as described in your patient booklet.

verse reactions: As with any contact lens, corneal edema and corneal abrasions occur if the lens is not properly fit, or if the wearing time is excessive.

e and administration: A gradual build-up of wearing time is recommended. A ommended schedule is as follows:

A) On the day of the initial fitting, two to four hours continuous wear.
B) Increase one hour each day for one week until eight continuous hours have been achieved.
C) If checkup is normal, wearing time is again increased one hour a day until full time (12 hours or more) is achieved.

Wearing schedule should be determined by the eye care practitioner.

Studies have not been performed to support safety for extended wear of this s.

w supplied: Each lens is supplied in a sealed sterile packet. The packet is identified h the dioptric power, base curve, center thickness and manufacturer's lens number. When the lens is removed from the packet, it should be stored in Flex-Care or ksol solution for at least four hours prior to dispensing to ensure maximum patient mfort.

UTION: Federal law prohibits dispensing without prescription.

Complete list of recommended solutions for Cleaning, Rinsing and Soaking SO lenses:

aning: Preflex* Cleaning Solution
Soft Mate* Cleaning Solution

sing: Flex-Care* Rinsing, Storing and Disinfecting Solution
Soft Mate* Rinsing Solution
Normol* Rinsing Solution

aking: Flex-Care* Rinsing, Storing and Disinfecting Solution
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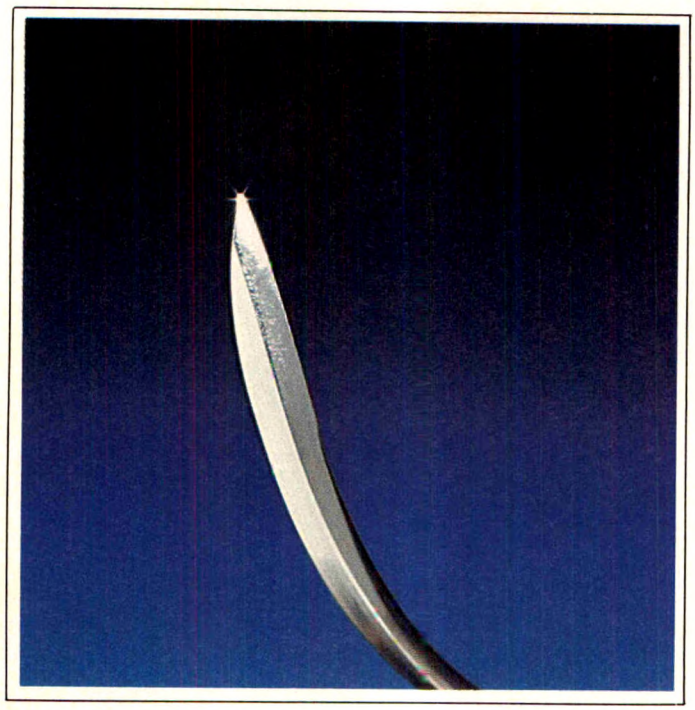
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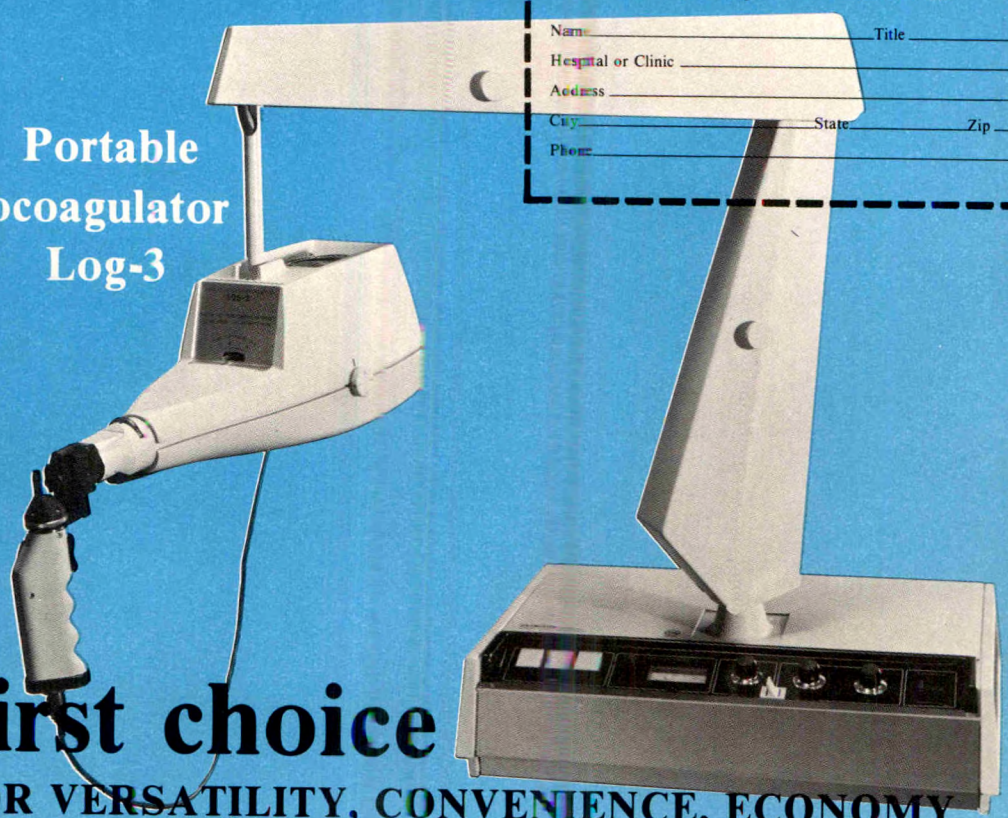
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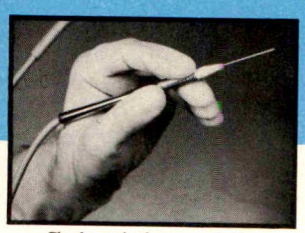
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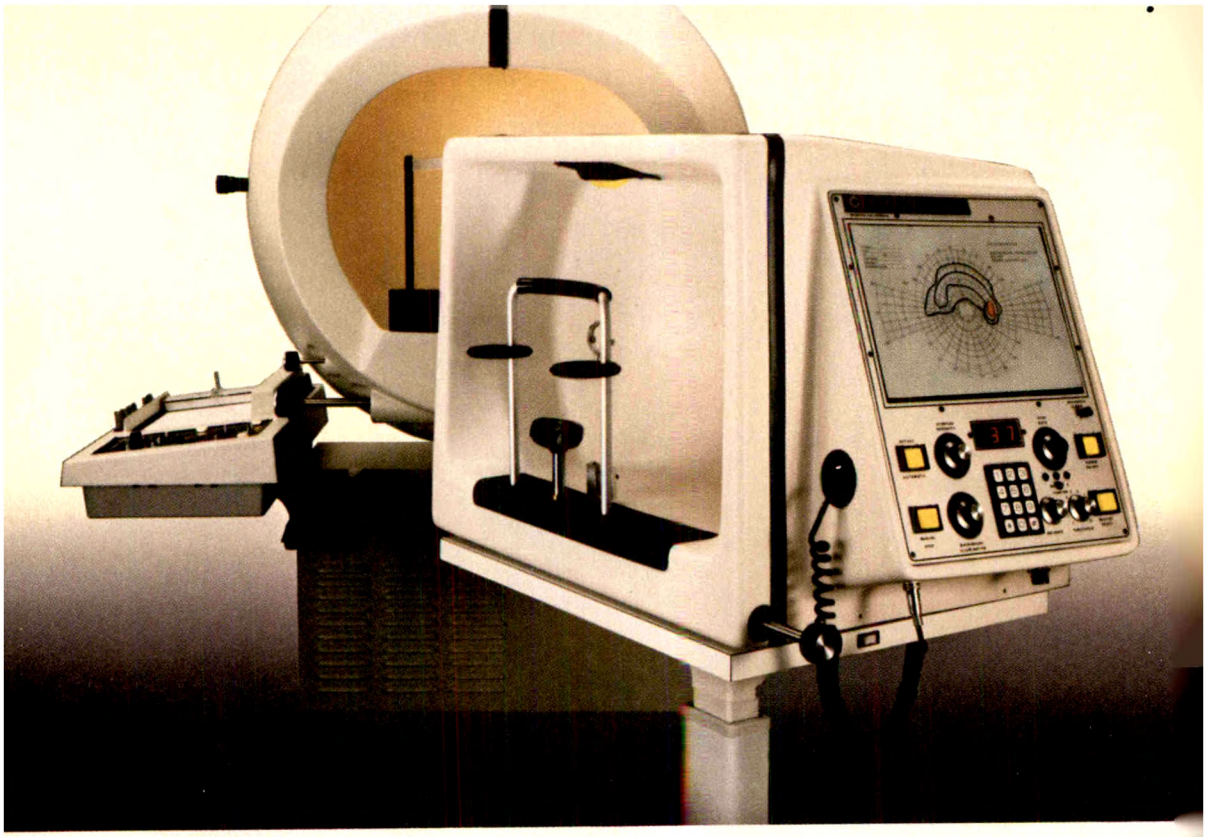
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ACTIONS The gram-positive bacteria against which gentamicin sulfate is active include coagulase-positive and coagulase-negative staphylococci, including certain strains that are resistant to penicillin; Group A beta-hemolytic and nonhemolytic streptococci, and *Diplococcus pneumoniae*. The gram-negative bacteria against which gentamicin sulfate is active include certain strains of *Pseudomonas aeruginosa*, indole-positive and indole-negative *Proteus* species, *Escherichia coli*, *Klebsiella pneumoniae* (Friedlander's bacillus), *Haemophilus influenzae* and *Haemophilus aegyptius* (Koch-Weeks bacillus), *Aerobacter aerogenes*, *Moraxella lacunata* (diplobacillus of Morax-Axenfeld), and *Neisseria* species, including *Neisseria gonorrhoeae*. Although significant resistant organisms have not been isolated from patients treated with gentamicin at the present time, this may occur in the future as resistance has been produced with difficulty *in vitro* by repeated exposures.

INDICATIONS GARAMYCIN **Ophthalmic Solution** and **Ointment** are indicated in the topical treatment of infections of the external eye and its adnexa caused by susceptible bacteria. Such infections embrace conjunctivitis, keratitis and keratoconjunctivitis, corneal ulcers, blepharitis and blepharoconjunctivitis, acute meibomianitis, and dacryocystitis.

CONTRAINDICATIONS GARAMYCIN **Ophthalmic Solution** and **Ointment** are contraindicated in patients with known hypersensitivity to any of the components.

WARNINGS GARAMYCIN **Ophthalmic Solution** is not for injection. It should never be injected subconjunctivally, nor should it be directly introduced into the anterior chamber of the eye.

PRECAUTIONS Prolonged use of topical antibiotics may give rise to overgrowth of nonsusceptible organisms, such as fungi. Should this occur, or if irritation or hypersensitivity to any component of the drug develops, discontinue use of the preparation and institute appropriate therapy.

Ophthalmic ointments may retard corneal healing.

ADVERSE REACTIONS Transient irritation has been reported with the use of GARAMYCIN **Ophthalmic Solution.**

Occasional burning or stinging may occur with the use of GARAMYCIN **Ophthalmic Ointment.**

DOSAGE AND ADMINISTRATION GARAMYCIN **Ophthalmic Solution:** instill one or two drops into the affected eye every four hours. In severe infections, dosage may be increased to as much as two drops once every hour.

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HOW SUPPLIED GARAMYCIN **Ophthalmic Solution—Sterile,** 5-ml. plastic dropper bottle, sterile, boxes of one and six. Store away from heat.

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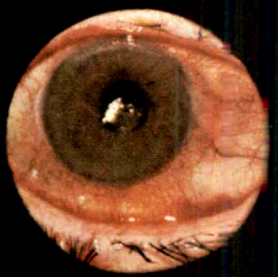
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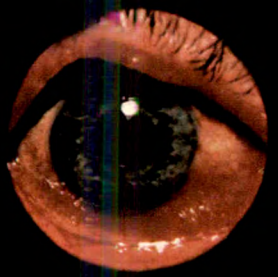
:ute catarrhal conjunctivitis
episcleritis. Etiology: S. aureus.
Before treatment.



Corneal ulcer.
Etiology: S. aureus.
Before treatment.



Acute hemorrhagic conjunctivitis.
Etiology: Pneumococcus.
Before treatment.

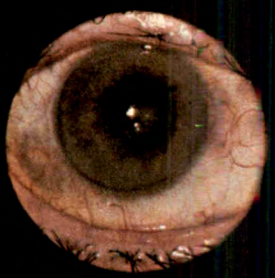


Corneal marginal ulcer.
Etiology: S. aureus.
Before treatment.

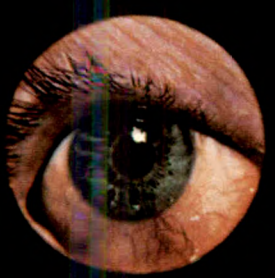




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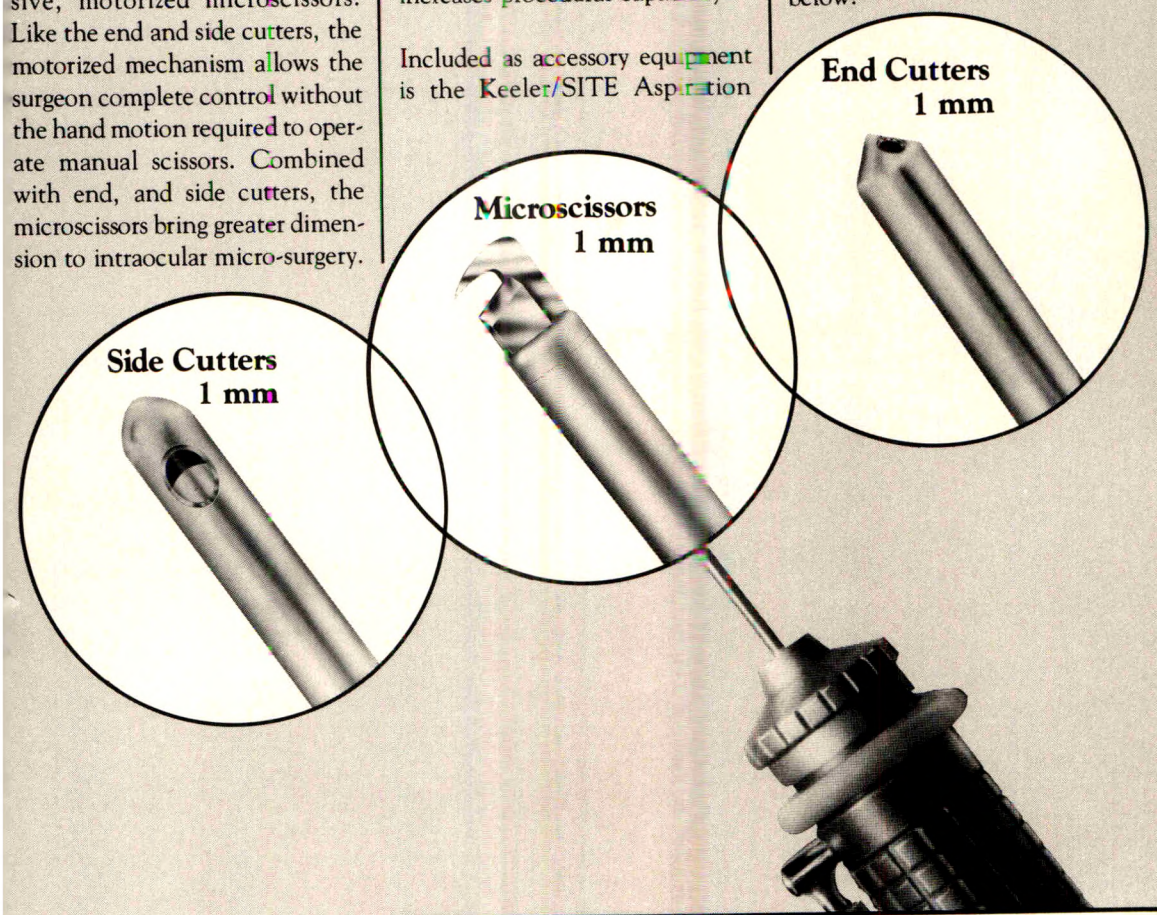
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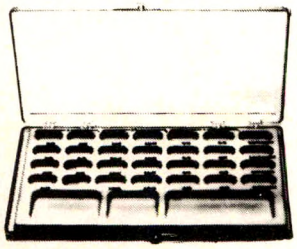
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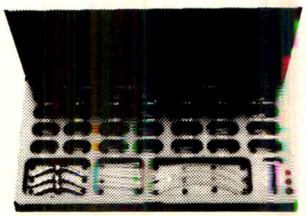
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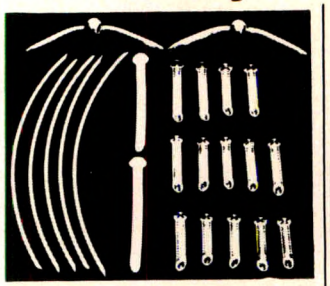
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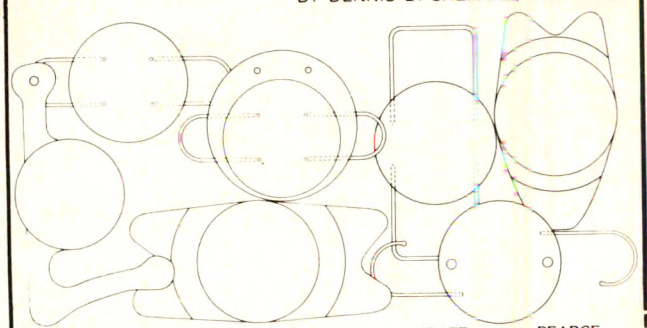
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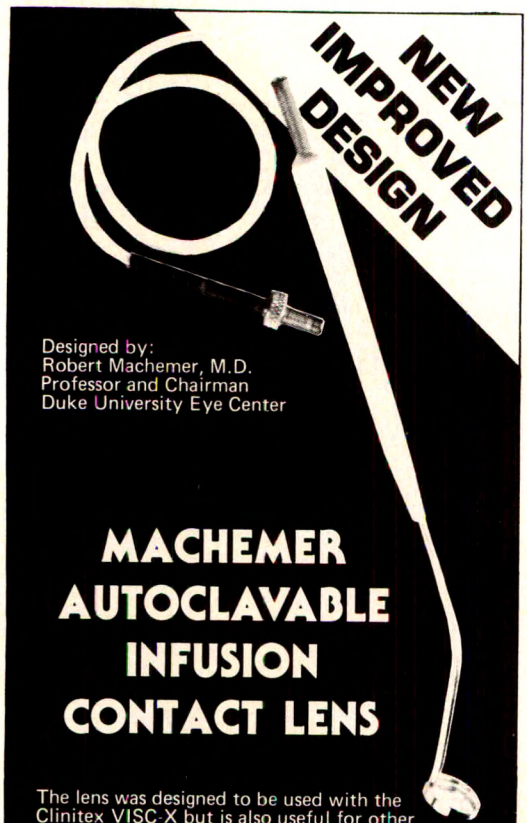
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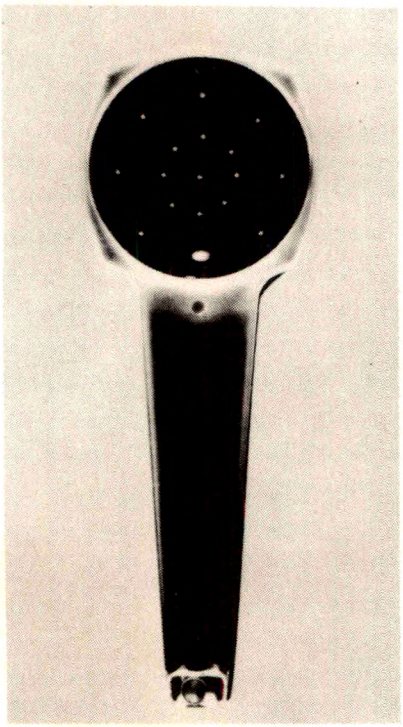
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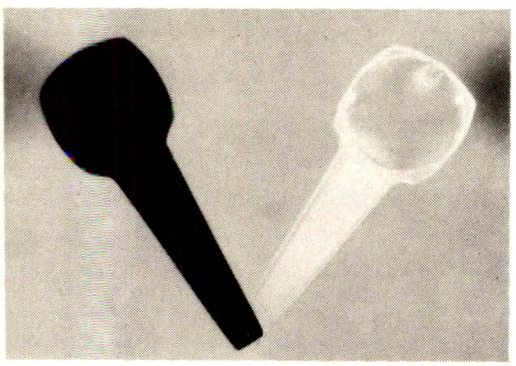
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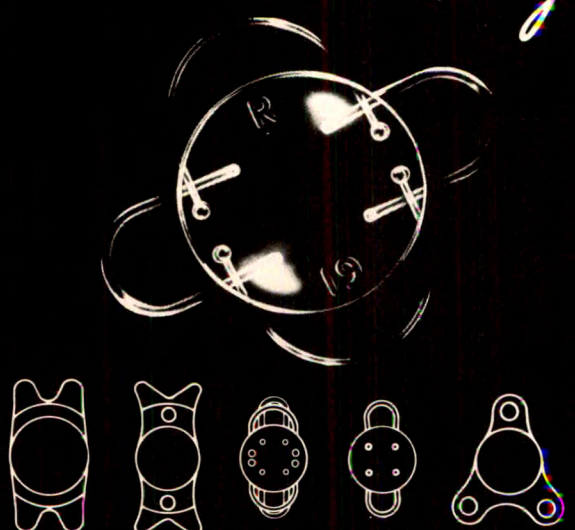
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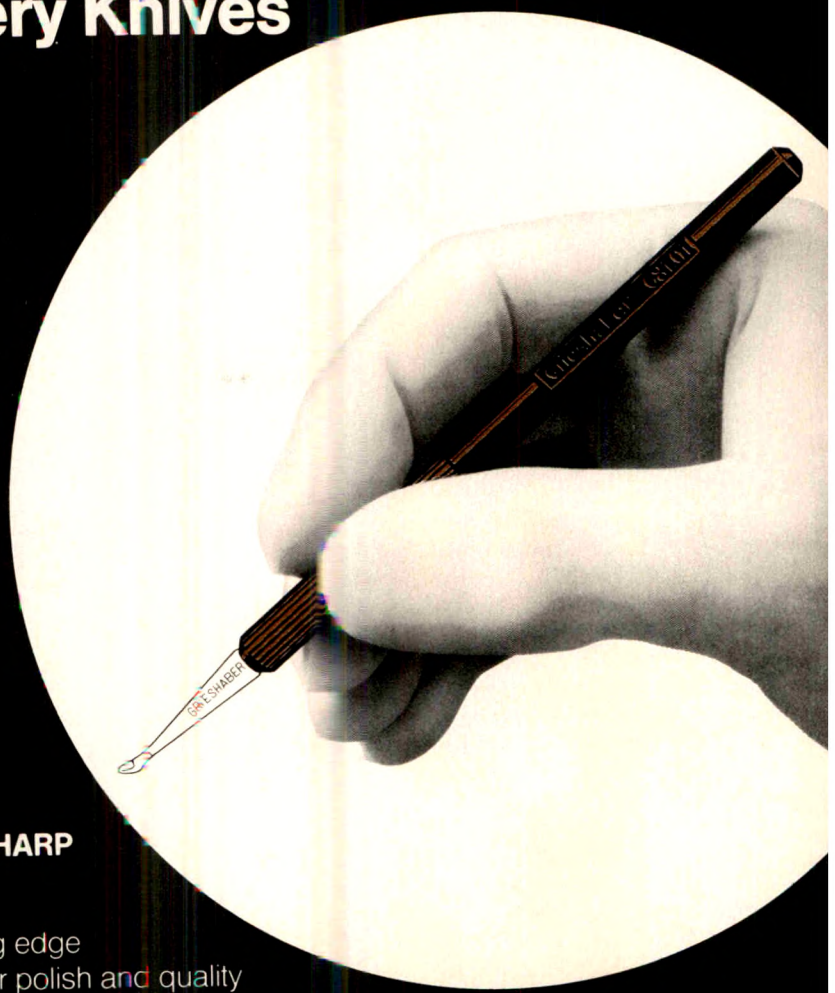
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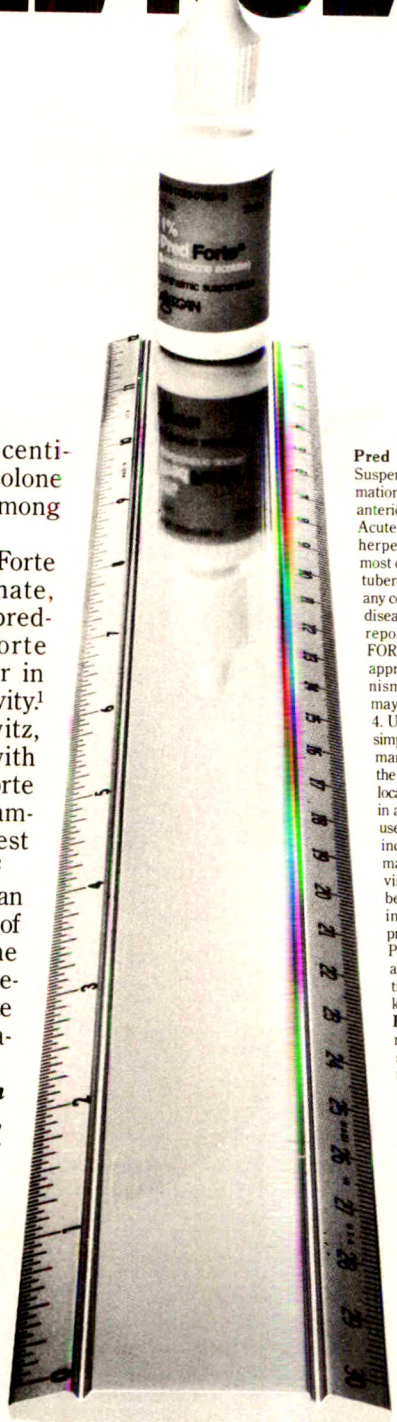
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REFERENCE NOTES: **1.** *Leibowitz, H. M. and Kupferman, A. Bioavailability and therapeutic effectiveness of topically administered corticosteroids. Trans Am Acad Ophthalmol Otolaryngol 79 (1): op 78-op 88, 1975.* **2.** *Ibid.* **3.** *Ibid.*

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DIABETIC MACULAR EDEMA IN JUVENILE- AND ADULT-ONSET DIABETES

JESSE SIGELMAN, M.D.

New York, New York

Diabetic macular edema is the greatest single cause of visual impairment in diabetic patients.[1] Previous studies of diabetic macular edema[2-9] have been directed primarily toward evaluating the efficacy of photocoagulation and medical treatment. In all of these studies, the patient groups have been composed of a mixture of patients with juvenile-onset and adult-onset diabetes. In the angiographic and histopathologic studies of diabetic maculopathy,[9-11] there also has been no differentiation on the basis of the type of diabetes.

Early studies[8,12] reported subjective impressions by the investigators that diabetic macular edema appeared mostly in patients with adult-onset diabetes and that there might be an inverse relationship between macular disease and the development of diabetic retinal neovascularization. The Hammersmith Hospital diabetic retinopathy study[13] was the first to give statistical evidence for a difference in diabetic macular disease between patients with juvenile-onset and adult-onset diabetes. In the Hammersmith study, 75% of the patients with diabetic maculopathy had adult-onset and 25% had juvenile-onset diabetes. Although this study drew no correlation between the development of macular edema and the appearance of proliferative retinopathy, it did report that the rapid development of proliferative retinopathy was most commonly seen in patients with juvenile-onset diabetes. From these findings the authors inferred that the intermediate stage of diabetic retinopathy was different in the two types of diabetes. Vascular hypermeability was more common in the patients with adult-onset diabetes than in those with juvenile-onset diabetes. Vaso-occlusive retinopathy occurred predominantly in the central retinas of patients with adult-onset diabetes and in the peripheral retinas of those with juvenile-onset diabetes.

My purpose was to characterize the differences in macular disease in the two types of diabetes. I studied the progression of the maculopathy and its association with proliferative diabetic retinopathy. The findings confirmed the previously reported impressions that there is a difference in macular disease between the two groups of diabetics.

SUBJECTS AND METHODS

The E. S. Harkness Eye Institute files contained fluorescein angiographic files for 103 patients referred because of diabetic retinopathy between 1974 and 1977 (Table 1). Each of these patients, accord-

From the Department of Ophthalmology, New York Hospital-Cornell Medical Center, Manhattan Eye, Ear and Throat Hospital, New York, New York. This study was supported by a grant from the Glorney-Raisbeck Foundation.

Reprint requests to Jesse Sigelman, M.D., 515 E. 71st St., New York, NY 10021.

TABLE 1

SUMMARY OF MACULOPATHY CATEGORIZATION

Data	Maculopathy Category							
	Background		Focal		Diffuse		Cystic	
	Juvenile-Onset	Adult-Onset	Juvenile-Onset	Adult-Onset	Juvenile-Onset	Adult-Onset	Juvenile-Onset	Adult-Onset
No. of patients	18	14	6	5	6	27	8†	11‡
Age (yrs)								
Median	48	57	50	64	45	67	53	65
Range	18-58	42-74	32-58	51-79	25-68	48-84	24-65	54-73
No. of eyes								
Total	35*	28	12	10	12	54	16	22
Dense vitreous hemorrhage	2	—	—	—	1	8	—	2
Premacular fibrosis	—	—	—	—	—	—	1	—
Duration of diabetes (yrs)								
Median	21	12	23	15	23	13	23	14
Range	4-44	1-28	12-30	10-38	10-34	1-34	8-39	4-33

*One eye was enucleated after trauma.

†Two patients had background maculopathy in the better eye.

‡One patient had focal maculopathy and one patient had diffuse maculopathy in the better eye.

ing to the referring physicians, had demonstrated at least some evidence of diabetic retinopathy. The progression of each patient's disease had been obtained from the patient. I categorized all patients who developed diabetes before the age of 40 years as having juvenile-onset diabetes and all patients who developed diabetes after the age of 40 years as having adult-onset diabetes.

Each patient had had a complete examination of both eyes. Although this included the measurement of visual acuity with the patient's correction, it did not include a manifest refraction in most cases. For this reason, I did not use visual acuity as the basis for categorization in this study. Each patient had undergone full retinal examination, including binocular indirect ophthalmoscopy of the posterior and peripheral retina and stereoscopic biomicroscopy. All reexaminations were performed by the individual who did the first examination.

Photographic fluorescein angiography of the posterior pole of both eyes had been performed in all cases except those with ocular media transparency loss caused by vitreous hemorrhage, cataract, or both.

I determined the density of microaneurysms in the posterior pole by their appearance during the arteriovenous phase when they appeared as hyperfluorescent spots. I estimated the status of the macular capillary bed by the extent of areas without capillary perfusion in the arteriovenous phase. I used only areas where fluorescence was not blocked by preretinal or intraretinal hemorrhage to determine capillary perfusion.

I used angiographic photographs taken approximately 45 minutes after injection to determine the degree of accumulation of fluorescein leakage.

I assigned each eye to one of four categories of macular disease based on the angiographic pattern: (1) Background maculopathy consisting of sparse microaneurysms without leakage (Figs. 1 and 2). I identified the microaneurysms by spots of hyperfluorescence in the arteriovenous phase. The microaneurysms showed staining without significant focal leakage or microcystic macular edema in late-stage angiography. (2) Focal maculopathy which consisted of microaneurysms in discrete areas within the posterior pole (Fig. 3). The microaneurysms caused fluorescein leakage into the macula with microcystoid edema in late-phase angiograms. (3) Diffuse maculopathy consisting of a diffuse bed of microaneurysms throughout the posterior pole in early-phase angiograms and diffuse fluorescein leakage and microcystoid edema of the macula in late-phase angiograms (Fig. 4). (4) "Cystic" macular edema. I chose this term in order to differentiate this category from the microcystoid edema present in the focal and diffuse leakage categories. The eyes with cystic edema showed diffuse leakage in early-phase angiography but differed from those with the other maculopathies in showing the pooling of fluorescein in late-phase angiography into large circular hyperfluorescent pools centered around the fovea (Fig. 5). The fluorescein poles corresponded to biomicroscopically evident cystic degeneration of the macula.

Only the first and second categories of maculopathy allowed me to assess the status of the capillary bed. In the last two categories, the fluorescein leakage was so rapid and diffuse that I could not assess the capillary bed accurately. Neovascularization of the retina at the posterior pole had been determined by biomicroscopic examination and confirmed by fluorescein angiography. Neovascularization in the retinal periphery had been determined either by biomicroscopy or by binocular indirect ophthalmoscopy. The presence of vitreous hemorrhage had been determined by binocular indirect ophthalmoscopy.

For patients in whom the maculopathy

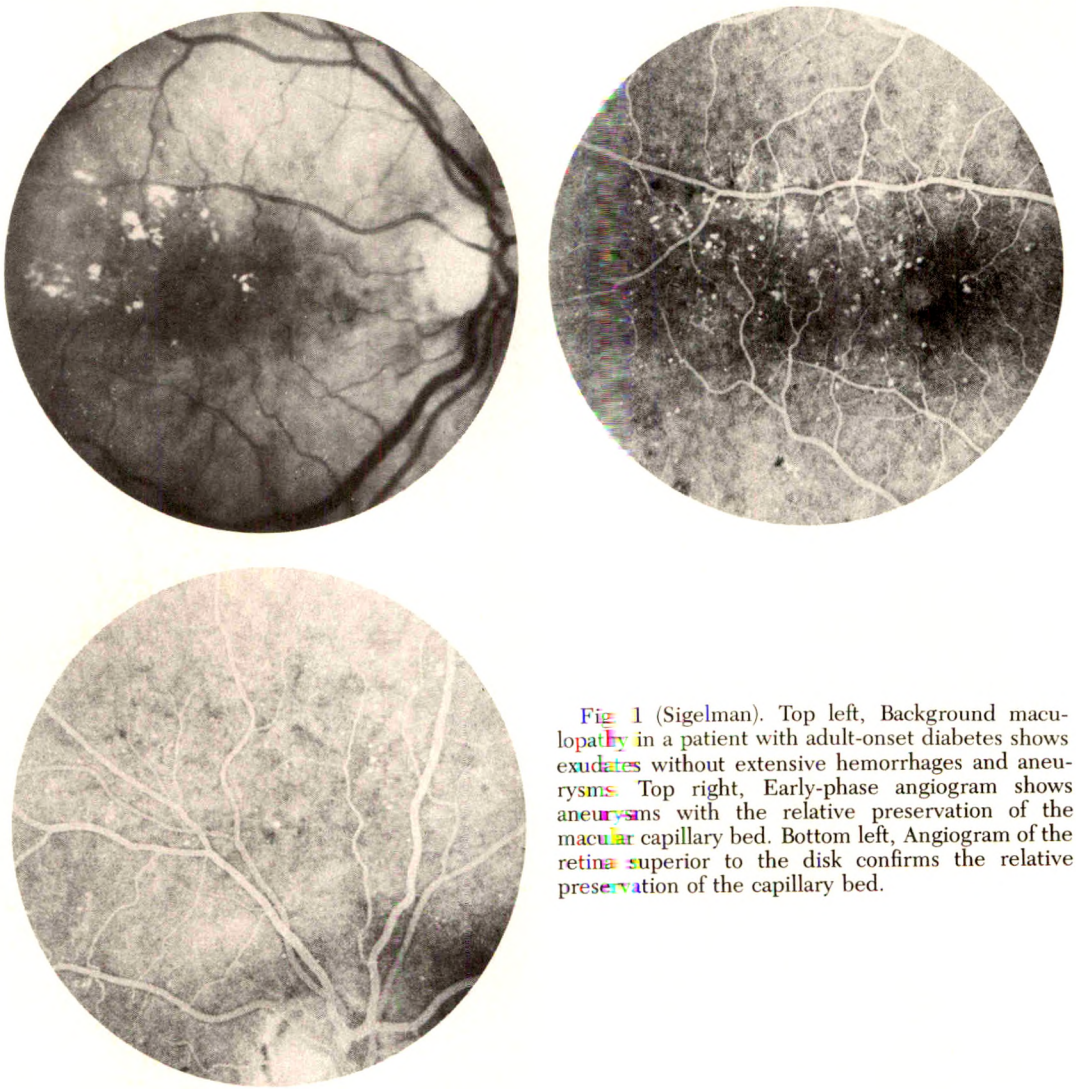


Fig. 1 (Sigelman). Top left, Background maculopathy in a patient with adult-onset diabetes shows exudates without extensive hemorrhages and aneurysms. Top right, Early-phase angiogram shows aneurysms with the relative preservation of the macular capillary bed. Bottom left, Angiogram of the retina superior to the disk confirms the relative preservation of the capillary bed.

was asymmetric in the two eyes, I categorized the patient on the basis of the angiographic status of the worse eye, recording the maculopathy in each eye separately. In cases where the fundus of one eye was obscured by a vitreous hemorrhage, I categorized the patient on the basis of the eye that had been clear enough for examination and angiography. I excluded ten of the 103 patients from the study because there were insufficient clinical data, five because of the poor quality of the angiograms, and five others because of inadequate information about the duration of their diabetes. Of the five patients in this last group, one had background retinopathy, one had focal maculopathy, one had diffuse leakage maculopathy, and two had cystic maculopathy. I categorized all eyes with vitreous hemorrhage as having neovascularization whether or not neovascularization could be observed through the hemorrhage.

Results

I subjectively estimated the degree of capillary loss in the patients with background and focal leakage. Obstruction in

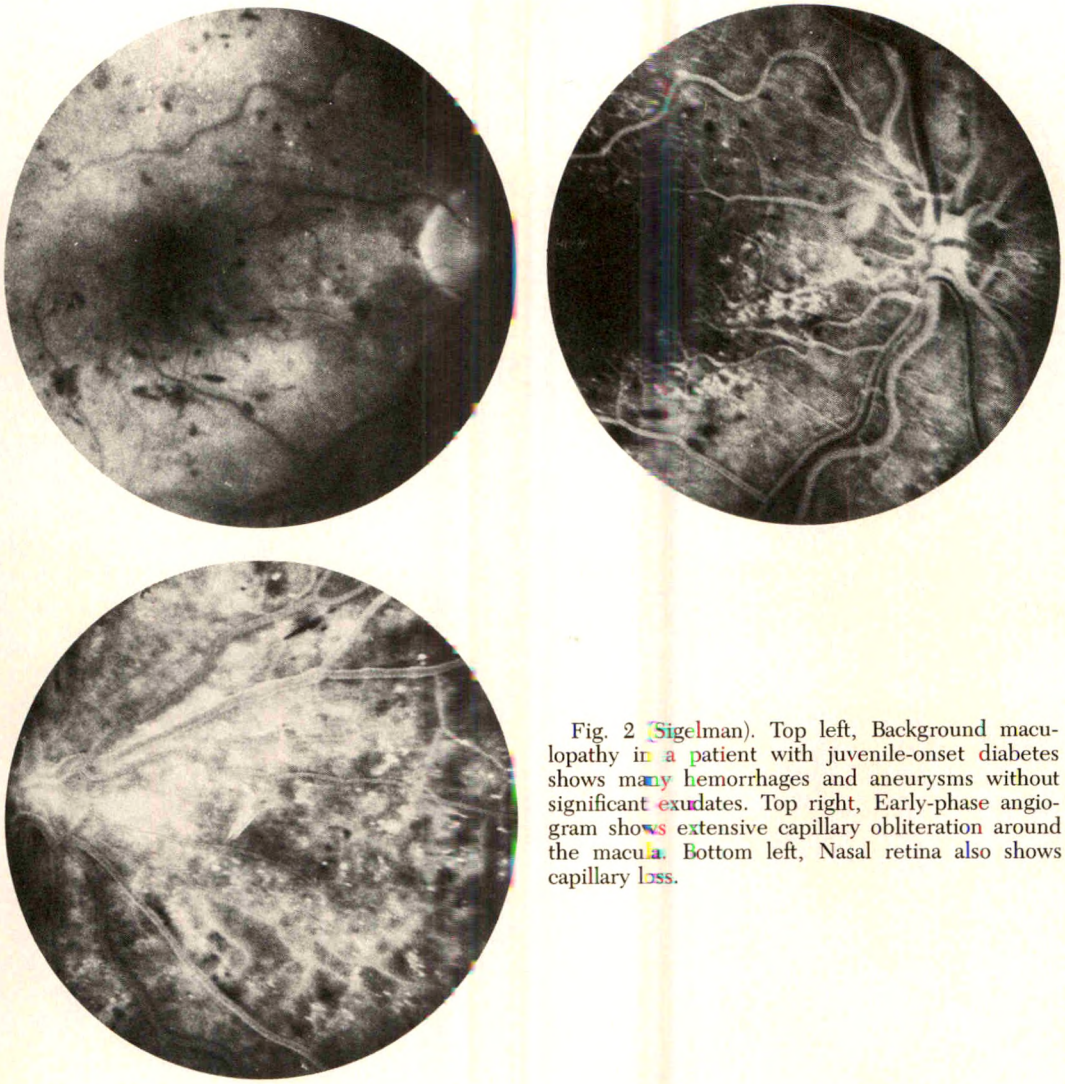


Fig. 2 (Sigelman). Top left, Background maculopathy in a patient with juvenile-onset diabetes shows many hemorrhages and aneurysms without significant exudates. Top right, Early-phase angiogram shows extensive capillary obliteration around the macula. Bottom left, Nasal retina also shows capillary loss.

the ocular media, cataracts, vitreous hemorrhages, and inadequate photographic technique prevented the precise measurement of capillary density in many eyes in the study. When it was discernible, capillary closure appeared more severe in the group with juvenile-onset diabetes than in that with adult-onset diabetes for the patients with both background maculopathy and focal leakage. For the patients with diffuse leakage and cystic maculopathy, the capillary bed was obscured by the diffuse, early fluorescein leakage. The only concomitant eye disease in these patients, besides cataract, had been recent episodes of unilateral iritis in two patients with focal maculopathy.

Discussion

It is the two types of primary diabetes that are associated with retinopathy. These are juvenile-onset and adult-onset diabetes. The two groups are distinguished on the basis of clinical, immunologic, and histopathologic criteria.[14] Adult-onset diabetes is associated with the onset of diabetes after the age of 40

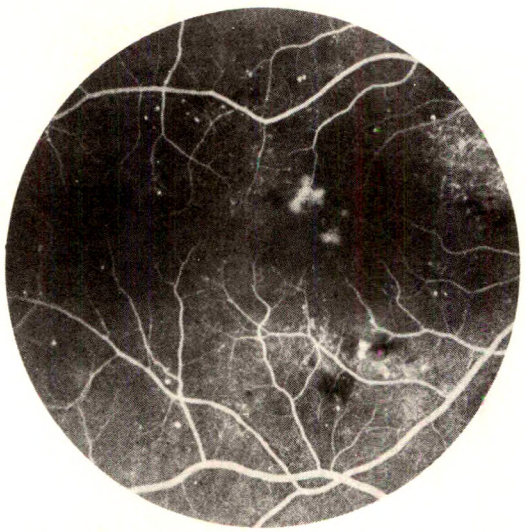


Fig. 3 (Sigelman). Focal leakage maculopathy shows discrete beds of aneurysms with relative preservation of the capillary bed in a patient with adult-onset diabetes.

years, obesity, gradual onset, preservation of the pancreatic islets of Langerhans,[15] insulin resistance, and slow progression of disease without brittle, rapid serum glucose responses to pharmacologic diabetic control. There is no association with cell-surface antigens or with specific antibodies. Juvenile-onset diabetes is associated with the onset of diabetes before the age of 40 years, sudden onset, destruction of the pancreatic islets of Langerhans, insulin deficiency, brittle, rapid serum glucose responses to pharmacologic diabetic control, and the presence of specific cell surface antigens. Juvenile-onset diabetes is unassociated with obesity.

Near the onset age of 40 years, the characteristics of the groups overlap so that the age of onset alone is only a statistical means of differentiating between the two groups. Each type of diabetes may occur at any age although they are well separated at the extremes of age. Without documentation of the other criteria, the age at onset is the only means of separating the two types of diabetic patients. Using the age at onset as the sole criterion has resulted in placing an unknown number of patients in this study into the wrong category.

The reason for categorizing the maculopathy on the basis of fluorescein angiography is that angiography is the best method of demonstrating the components of each stage of maculopathy. It shows the

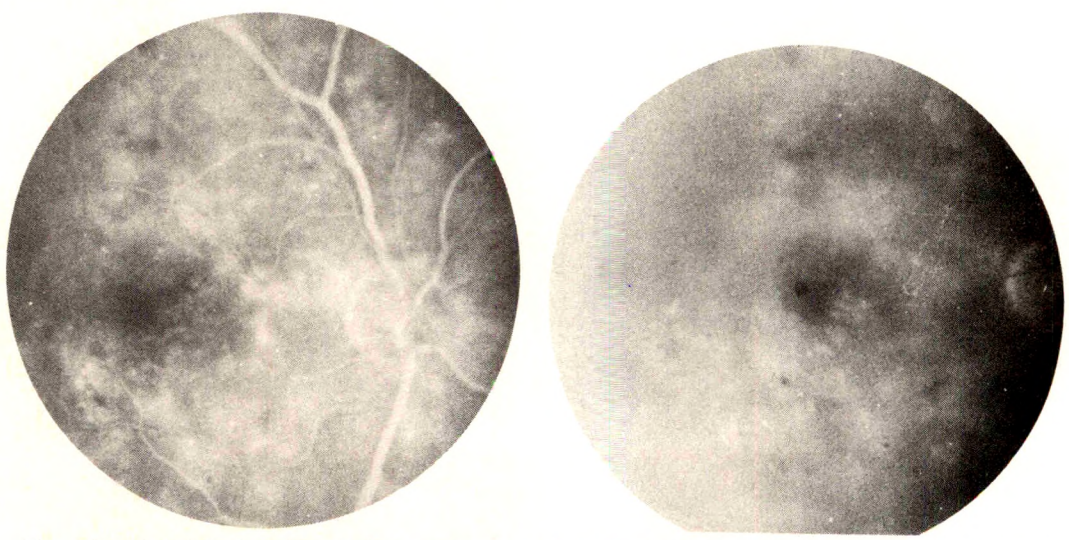


Fig. 4 (Sigelman). Left, Diffuse leakage pattern in early phase. Capillary pattern is not discernible. Right, Late phase shows diffuse leakage pooling into microcysts.

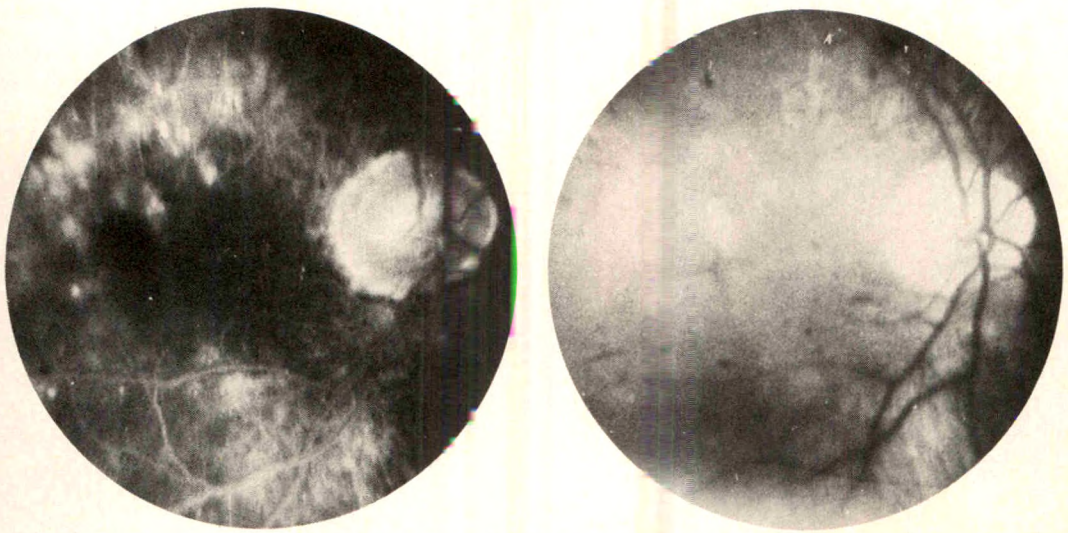


Fig. 5 (Sigelman). Cystic edema. Left, Early phase shows multiple leakage sites surrounding the macula. Right, Late phase shows the pooling of fluorescein into large cysts.

microaneurysms, capillary obliteration, intraretinal shunts, breakdown of the blood-retinal barrier, microcystic edema, and cysts that are the sequential elements in the progression of maculopathy.[16-18] Using visual acuity to grade the progression of maculopathy rather than angiographic observations gives less significant results because visual acuity may be altered significantly by optical phenomena, by small variations in the location of intraretinal hard exudates and hemorrhages, or by the loss of transparency of the crystalline lens.

Each category represents one stage of the progression of the maculopathy. I emphasized the underlying vascular disease because its progression is the cause of the diabetic macular changes that cause the loss of macular function.[19] The intraretinal hemorrhage, edema, hard exudates, microcysts, and large cysts are the sequelae of the development of retinal ischemic foci[6] with breakdown in the blood-retinal barrier and with capillary closure.

The most significant finding was the higher rates of disk or retinal neovascularization and vitreous hemorrhage, in each category of maculopathy, for the group with juvenile-onset diabetes compared to that with adult-onset diabetes (Table 2).

A potential inaccuracy in this calculation is the assignment to the wrong category of patients in whom the view of the macula of one eye was blocked by a dense vitreous hemorrhage. Because I categorized all patients with dense vitreous hemorrhages on the basis of the fellow eye, I may have underestimated the maculopathy.

The maximum possible effect of such an error on the group with juvenile-onset diabetes would be to move two of the patients with background maculopathy and vitreous hemorrhage and one of the patients with diffuse maculopathy and vitreous hemorrhage into more advanced maculopathy categories. This would have the effect of decreasing by two the seven patients with juvenile-onset diabetes categorized as having background maculopathy and neovascularization. The six patients with juvenile-onset diabetes categorized as having diffuse maculopathy

TABLE 2

NEOVASCULARIZATION AND VITREOUS HEMORRHAGE

Data	Maculopathy Category							
	Background		Focal		Diffuse		Cystic	
	Juvenile-Onset	Adult-Onset	Juvenile-Onset	Adult-Onset	Juvenile-Onset	Adult-Onset	Juvenile-Onset	Adult-Onset
No. of eyes	12	4	1	0	8	16	7	5
No. of patients	7	3	1	0	6	11	5	5
Age (yrs)								
Median	41	68	32	—	45	64	50	60
Range	18-58	55-74	32	—	33-65	48-84	24-61	54-68
Duration of diabetes (yrs)								
Median	21	21	14	—	23	13	18	9
Range	4-44	9-25	14	—	8-34	1-34	8-39	4-19

and neovascularization would be decreased by one.

Of the eleven patients with adult-onset diabetes categorized as having diffuse maculopathy and neovascularization, a maximum of eight would be moved into another category. Even if these changes were appropriate only for the group with juvenile-onset diabetes, the association of neovascularization with each category of maculopathy would be greater for that group of patients than for the group with adult-onset diabetes. The symmetry of the degree of maculopathy in the two eyes of the patients in this study suggests that the need for these changes is not likely.

The higher rate of neovascularization in each category of maculopathy in the group with juvenile-onset diabetes suggests that separate factors cause maculopathy and neovascularization.

Maculopathy, as I defined it in this study, results from vascular leakage in and surrounding the macula. Neovascularization results from retinal ischemia.[11,20] This study suggests that the same degree of vascular leakage is associated with a higher degree of ischemia in the group with juvenile-onset diabetes. The corollary of this finding is that capillary leakage is associated with more extensive capillary closure in the group with juvenile-onset diabetes. My subjective observations of fluorescein angiograms of patients in this study with background and focal maculopathy support this concept. This difference may reflect a difference in the capillary disease in the two types of diabetes. Additionally, the greater age of the group with adult-onset diabetes may contribute to the lower rate of neovascularization because older tissues tend to respond less actively to the ischemic stimulus. The therapeutic implication of this finding is that the presence of neovascularization at each level of maculopathy should be more readily suspected in the group with juvenile-onset diabetes.

The degree of maculopathy in the fellow eyes of almost all patients was equal at almost all levels of maculopathy. This finding supports the validity of using one eye as a control in the prospective study of therapeutic regimens.[2,19]

In adult-onset diabetes, the duration of diabetes usually is not accurately ascertained because of the gradual onset and the time lag between the onset of asymptomatic chemical diabetes and the later onset of symptoms. The imprecise determination of the age at onset of diabetes in this group makes it difficult to draw conclusions about the relationship of the duration of diabetes and the degree of maculopathy.

The duration of diabetes can be established accurately in juvenile-onset diabetes because of its sudden symptomatic onset. Comparisons of the duration of diabetes in the two groups must take into account that duration is underestimated in adult-onset diabetes. The median duration of diabetes in this study was shorter for each category of maculopathy in the group with adult-onset diabetes than in the group with juvenile-onset diabetes. This finding may not be meaningful, however, because of the likelihood of underestimating the duration in the group with adult-onset diabetes.

The age and duration in each category of maculopathy for patients with and without neovascularization were approximately the same. These factors suggest that the risk of neovascularization may be more closely related to the degree of maculopathy and to the type of diabetes than to the patient's age or duration of diabetes.

The similarity of the median ages of the two groups for patients in each category of maculopathy implies that the progression of maculopathy is not closely related to age for either group. Although the

duration of diabetes might be expected to correlate with the degree of maculopathy, this was not found to be the case in the group with juvenile-onset diabetes, for whom the data on the duration of diabetes are fairly reliable. This finding implies that the progression of maculopathy is rapid once it appears. This conclusion may be erroneous because all the patients in this study were referred because of retinopathy.

Summary

A retrospective study of 103 patients referred because of diabetic retinopathy graded the degree of maculopathy on the basis of fluorescein angiography. Patients with juvenile-onset diabetes (that is, onset before the age of 40 years), had a higher rate of neovascularization of the disk or retina at early stages of maculopathy than patients with adult-onset diabetes. Greater capillary closure at each degree of maculopathy in the group with juvenile-onset diabetes may explain this difference.

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LONG-TERM ANATOMIC AND FUNCTIONAL RESULTS OF VITRECTOMY FOR DIABETIC RETINOPATHY

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Closed vitreous surgery through a pars plana approach is often used to treat severe complications of proliferative diabetic retinopathy, including nonabsorbing vitreous hemorrhage and retinal detachment. When the specific anatomic objectives of the procedure have been achieved, vision often improves soon after the operation. Severe postoperative complications occur most commonly during the first weeks after the operation. These include intraocular hemorrhage, retinal detachment, and rubeosis iridis.

Some authors[1,2] have observed that, after an initially successful vitrectomy, the course of the proliferative retinopathy is markedly changed. Preretinal (elevated) fibrovascular proliferation does not seem to occur when the scaffold of the adjacent posterior vitreous surface is absent.[2,3] It has been our impression in these cases that the posterior segment remains anatomically stable and the incidence of blinding complications is reduced. Blankenship and Machemer[4] reported that 82% of 17 diabetic eyes with a successful result six months after vitrectomy retained good vision five years after the operation. Still, the long-term results of vitrectomy in diabetic eyes have been uncertain.

This study was performed to evaluate the long-term visual results, anatomic effects, and complications of vitrectomy in diabetic eyes. We chose to study cases in which a successful result was achieved during the first six months after the operation. We studied these eyes to learn the outcome over a longer period of time. We excluded those eyes in which the operation was judged unsuccessful soon after surgery because eyes with blinding complications during the early postoperative period seldom have improved vision later.

MATERIAL AND METHODS

We studied a consecutive series of 146 eyes that had an initially successful result after vitreous surgery for complications of proliferative diabetic retinopathy. The indications for surgery had included nonabsorbing vitreous hemorrhage, traction retinal detachment, combined traction and rhegmatogenous detachment, and progressive growth of fibrovascular tissue damaging the optic nerve or macula. We defined initially successful surgery as visual acuity of 6/240 (5/200) or better at any time during the first six months after the operation, along with the absence of untreatable blinding complications such as inoperable rhegmatogenous retinal detachment or uncontrolled neovascular glaucoma. We included eyes in the series if the visual acuity was 6/240 (5/200) or better, even if residual vitreous hemorrhage or traction retinal detachment was present, because we felt that these abnormalities did not necessarily preclude a later successful result. Of the 146 eyes selected, we eliminated 16 from the study. Seven of these 16 eyes were in patients lost to follow-up, and nine eyes could not be reexamined because the patients had died.

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We reviewed the medical records and examined the remaining 130 eyes (119 patients). The average age of the patients was 48 years; the youngest patient was 21 years old and the oldest was 79 years old. The minimum follow-up time was 18 months, and the longest follow-up time was 68 months. The mean follow-up interval was 32.5 months, and the median follow-up interval was 30 months. We performed the final examination on 111 eyes; other ophthalmologists who reported their findings examined the other 19 eyes.

Of the 130 eyes, 115 (88%) had had preoperative vitreous hemorrhage, and 32 eyes (25%) had had preoperative retinal detachment involving the macula. Vitrectomies were performed with the Douvas Rotoextractor or the O'Malley Ocutome introduced through a pars plana approach. In most of the 130 eyes the surgical objectives achieved included (1) removal of any vitreous hemorrhage, (2) removal of portions of the vitreous gel and excision of the posterior vitreous surface posterior to the equator, and (3) closure of any retinal tears. Epiretinal membranes were removed or segmented when possible.

Sixty-two eyes (48%) had had some preoperative lens opacity, and the crystalline lens was removed in 94 eyes (72%). Many clear lenses were removed, because this was the practice at the time these operations were done. Peripheral retinal cryotherapy with an encircling scleral buckle was used in 93 eyes (72%). This was a prophylactic measure in 87 eyes to prevent later retinal detachment[2]; this technique was used for treatment of a retinal break in six eyes. Prophylactic peripheral retinal cryotherapy, without a scleral buckle, was applied in 28 additional eyes (22%). Only nine eyes (7%) had no cryotherapy applied to the peripheral retina. We no longer apply retinal cryotherapy or perform peripheral scleral buckling for prophylactic purposes.

Complications during the vitreous surgery included bleeding (18%), iatrogenic retinal holes (13%), retinal dialysis (5%), and retinal detachment (5%). Most of these complications spontaneously resolved or were successfully treated, as would be expected in a series composed of cases that appeared successful during the first six months after the operation.

The final examination included measurement of best corrected visual acuity, external evaluation, slit-lamp examination applanation tonometry, and indirect ophthalmoscopy. We examined the retina and vitreous cavity by biomicroscopy with a contact lens. We examined the internal aspect of the superotemporal pars plana sclerotomy site by indirect ophthalmoscopy aided by scleral indentation. We performed fluorescein angiography of the iris or fundus, or both, in some cases. During the examination, we tried to detect iris neovascularization, retinal neovascularization, retinal detachment, macular damage, and fibrovascular ingrowth extending from the sclerotomy site.

We analyzed the data from each patient's medical record and information from the final examination to learn whether these eyes remained visually and anatomically stable, and to determine the type and incidence of later complications. For each eye, we compared the best visual acuity recorded during the first six months after the operation to the visual acuity at the final examination to determine whether vision remained stable, improved, or worsened. For the entire group of eyes, we compared the incidence of each major complication during the first six postoperative months to the later incidence. We also compared the percentage of eyes needing

additional surgery during the initial six postoperative months to the percentage of cases needing additional surgery later.

Results

In this series of initially successful cases, 123 eyes (95%) had improved visual acuity and only seven eyes (5%) were unimproved or worse (Fig. 1).

Comparing the best visual acuity achieved during the first six postoperative months to the final visual acuity provides a measure of the long-term functional stability after vitrectomy (Fig. 2). Visual acuity remained the same in 31 eyes (24%) and improved in 64 eyes (49%).

Thirty-five eyes (27%) showed a decrease in the final visual acuity but only ten of these (8%) had a significant reduction, defined here as more than a two-fold increase in the visual angle subtended by the test letters. For low levels of visual acuity, we equated a decrease from 6/240 (5/200) to hand motions with a doubling of the visual angle. The causes of significant deterioration in visual acuity in these ten eyes were (1) neovascular glaucoma (two eyes), (2) cataract (two eyes), (3) progressive traction retinal detachment caused by residual epiretinal membranes (one eye), (4) macular scar (one eye), (5) rhegmatogenous retinal detachment causing permanently reduced visual acuity despite successful repair (one eye), (6) opacification of an epiretinal membrane covering the macula (one eye), and (7) unexplained causes (two eyes). Eyes with only a mild decrease in visual acuity usually had no apparent anatomic change, and we attributed the reduction to a worsening of intraretinal vascular changes affecting the macula in these diabetic patients.

Although ten eyes (8%) had a significant decrease in visual acuity after the first six postoperative months, 22 eyes

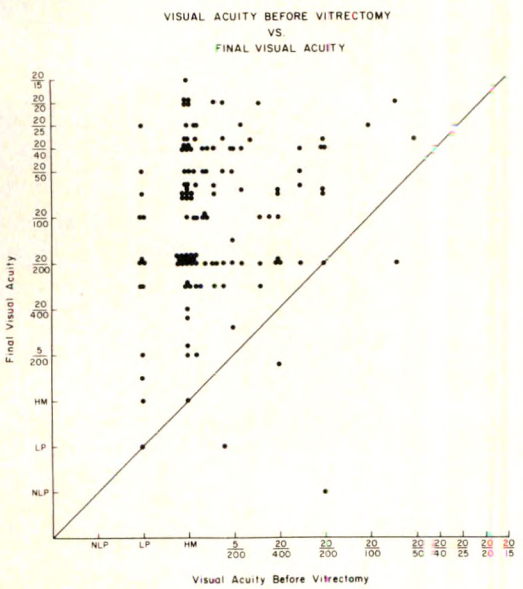


Fig. 1 (Rice and Michels). Each dot represents one of the 130 eyes. Dots above and to the left of the diagonal line represent eyes in which final visual acuity improved after vitrectomy. Dots below and to the right of the diagonal line represent eyes in which visual acuity was reduced.

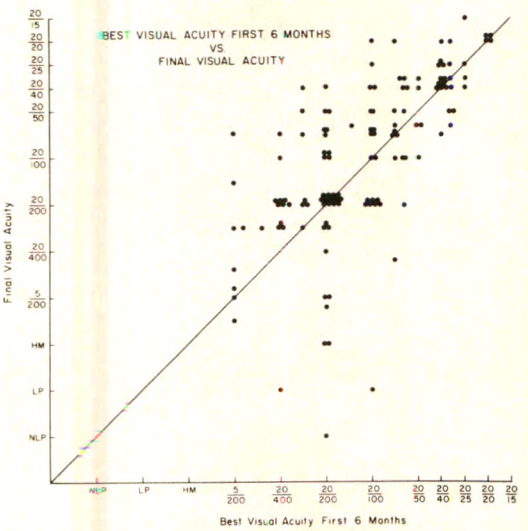


Fig. 2 (Rice and Michels). Each dot represents one of the 130 eyes. Dots above and to the left of the diagonal line represent eyes in which final visual acuity was better than the best visual acuity during the first six months. Dots below and to the right represent eyes in which visual acuity was worse.

(17%) had a significant improvement in final visual acuity, defined here as more than a halving of the subtended visual angle. There was no statistically significant difference in the visual results for the 50% of the eyes followed up for less than 30 months compared to the 50% followed up longer. In fact, the group followed up for a longer time, as a whole, fared slightly better.

Complications after vitreous surgery occurred in a number of cases, but complications were less frequent after the initial six-month postoperative period. Table 1 summarizes the time of onset of the more frequent severe complications. Iris neovascularization, which is a common complication after vitrectomy in diabetic eyes, was much more frequent during the first six months. Cases with severe iris neovascularization were usually treated with scatter retinal photocoagulation, and this often caused complete or nearly complete regression of the neovascular tissue on the iris (Table 2). Some milder cases seemed to be nonprogressive and were not treated by retinal photocoagulation.

Iris neovascularization occurred more frequently in aphakic eyes than in phakic eyes. Twenty-five of 102 aphakic eyes (25%) developed iris neovascularization whereas only three of 28 phakic eyes (11%) developed this complication. The difference between these two groups is not statistically significant (P = .09, Fisher's exact test), but there is a trend suggesting that aphakic eyes are more likely to develop postoperative iris neovascularization than phakic eyes.

TABLE 1

MOST COMMON POSTOPERATIVE COMPLICATIONS IN 130 EYES

Complication	Time of Onset	
	<6 mo	>6 mo
Iris neovascularization	25	7
Glaucoma (neovascular glaucoma and other)	6	7
Vitreous hemorrhage	10	9
Rhegmatogenous retinal detachment	8	1

TABLE 2

POSTOPERATIVE IRIS NEOVASCULARIZATION

Neovascularization Treatment Results	Onset of Neovascularization	
	<6 mos	>6 mos
No treatment		
Regressed, or mild and stable	7	2
Progressive, severe	1	1
Scatter photocoagulation		
Regressed, or mild and stable	14	4
Progressive, severe	1	0
Reattachment of detached retina, regressed	2	0
Total	25	7

Postoperative glaucoma, other than that caused by transient increase in the intraocular pressure during the immediate postoperative period, occurred in 13 eyes (10%). The time of onset was about equally divided between the first six months after surgery and the period after that. Most cases of glaucoma were a consequence of neovascularization of the anterior-chamber angle, although most cases of iris neovascularization did not progress to glaucoma, perhaps because scatter retinal photocoagulation caused apparent regression of the neovascular tissue in some cases. Glaucoma was not controlled with medication (intraocular pressure > 30 mm Hg) in seven of the 13 involved eyes. In six of the seven eyes with uncontrolled pressure, glaucoma was caused by iris neovascularization.

New vitreous hemorrhage unrelated to residual intraocular blood remaining after the initial vitrectomy occurred in 19 eyes

(15%). The site of origin of the postoperative hemorrhage usually could not be determined. Iris neovascularization may have been the cause in some aphakic eyes, but it may have been only an associated finding, indicating more widespread vascular disease in these cases. Vitreous hemorrhage did not seem to be a direct consequence of new extraretinal neovascularization, although in some eyes localized blood was clotted on the inner retinal surface, suggesting that bleeding was of retinal origin. In one eye with recurrent hemorrhages, we found retinal neovascularization at the final examination. Most hemorrhages cleared spontaneously, although some eyes needed additional surgery to remove blood. No eye had a dense vitreous hemorrhage at the time of final examination. Vitreous hemorrhage and glaucoma were the only serious complications that did not occur mainly during the first six months.

Retinal detachment occurred most frequently during the first six months after the operation. Eight eyes developed rhegmatogenous detachment within six months. Further surgery in seven of these eight eyes provided successful reattachment. Traction retinal detachment involving the macula occurred after surgery in three eyes because of contraction of residual epiretinal membranes located near the macula. In these cases, it was difficult to determine whether the onset of macular involvement occurred during the first six months after the operation or later. In other cases, areas of extramacular traction detachment persisted after vitrectomy, usually caused by contraction of residual epiretinal fibrovascular tissue. We observed this detachment in 26 eyes (20%). In 22 other cases, involvement of the macula by traction detachment had been present preoperatively, and the retina was successfully reattached after the operation. Therefore, both traction and rhegmatogenous retinal detachment occurred postoperatively in some cases, although these complications were rare more than six months after the operation.

Corneal opacification, excluding transient postoperative edema, occurred in three eyes. This was associated with markedly increased intraocular pressure in these eyes and did not seem to be caused by intraoperative damage to the corneal endothelium.

Visually significant cataracts developed postoperatively in four of 28 phakic eyes (14%). Two of these cataracts were removed, and we found an opaque lens at the final examination in two eyes. Clear lenses were removed in four additional eyes during secondary surgical procedures such as lavage of recurrent vitreous hemorrhage.

Both secondary surgical procedures and laser treatment were required most often during the first six months after vitrectomy (Table 3). Surgery during the early postoperative period was performed most often for retinal detachment, although after the initial six months various secondary operations were performed. Laser treatment during and after the initial postoperative period was mainly used to treat iris neovascularization by scatter retinal photocoagulation. Direct photocoagulation of new blood vessels in the anterior chamber angle was not performed. Persistent or recurrent areas of retinal neovascularization or other retinal vascular abnormalities were occasionally treated with focal photocoagulation if

TABLE 3

ADDITIONAL POSTOPERATIVE SURGERY AND LASER TREATMENT

Treatment	Time After Vitrectomy <6 mos	Time After Vitrectomy >6 mos
Surgery	16	7
Laser	18	8
Total	34	15

they were thought to be the cause of recurrent vitreous hemorrhage.

During the final examination we recorded all abnormalities. Table 4 lists the most prominent. We had also observed most of the abnormalities during previous examinations. The low incidence of active retinal neovascularization is particularly interesting. We observed retinal neovascularization in only three eyes. One of these three eyes had peripheral neovascularization beneath an area where the posterior cortical vitreous had not been removed. Another eye had a moderate amount of neovascularization in a residual epiretinal membrane. This was unusual because portions of epiretinal membranes were present postoperatively in 97 of the 130 cases (75%) in this series, and, in these cases, the vascular component had undergone involution so that the epiretinal membranes were white and atrophic. The third eye had a small patch of peripheral neovascularization unassociated with adjacent cortical vitreous or residual epiretinal membranes. In each of these three cases, the neovascular tissue was present along the plane of the inner retinal surface, and no eye had proliferation of elevated fibrovascular tissue.

TABLE 4

FINAL EXAMINATION

Pathologic Findings	No. of Eyes	%
Corneal edema (All had severe glaucoma)	3	2
Iris neovascularization	8	6
Glaucoma	13	10
Cataract (9 of 22 phakic eyes)	9	
Vitreous hemorrhage (Severe in only 1)	4	3
Retinal neovascularization	3	2
Vascularization of internal aspect of sclerotomy (1 of 107 eyes examined)	1	
Macular abnormalities	108	83

We examined the internal aspect of the superotemporal sclerotomy site by indirect ophthalmoscopy aided by scleral depression in 107 eyes. Twenty-one of these 107 eyes (20%) appeared to be free of any incarcerated tissue. Clear vitreous was incarcerated in the wound in 76 of the 107 eyes (71%), and ten eyes had white fibrous tissue extending from the wound. Of these ten eyes with localized fibrous proliferation, we observed prominent blood vessels within the tissue in only one. The blood vessels extended onto the residual peripheral lens capsule in this aphakic eye. These blood vessels were asymptomatic, and there was no history of recurrent vitreous hemorrhage.

Macular abnormalities were present in 108 eyes (83%) at the final examination, and accounted for some decrease in visual acuity. The most frequent abnormalities were mottling of the retinal pigment epithelium, intraretinal edema, and narrowing or sheathing, or both, of small retinal vessels in the posterior pole. These changes seemed to be nonprogressive when compared to descriptions in the medical records and previous fundus photographs and fluorescein angiograms of these patients.

DISCUSSION

Vitreous surgery for complications of proliferative diabetic retinopathy is a somewhat hazardous procedure, and major complications resulting in further loss of vision and inoperable structural or pathophysiologic complications can occur. However, severe blinding complications occur most commonly during the first 12 weeks after the operation. We have the impression that when early postoperative complications do not occur the eye is stable thereafter, with partial improvement in visual acuity and virtual absence of progressive structural damage

from further extraretinal fibrovascular proliferation.

The data in this study suggest that the eye is remarkably stable after initially successful vitrectomy. The final visual acuity was the same as or better than the best visual acuity recorded during the first six months after surgery in 95 of the eyes (73%), and in only ten eyes (8%) was the final visual acuity reduced by an amount greater than a doubling of the subtended visual angle. Significant anatomic or functional abnormalities suggesting instability, such as uncontrolled glaucoma, visually significant cataract, severe vitreous hemorrhage, active retinal neovascularization, or new retinal detachment involving the macula, were present in only 15 eyes (12%) at the final examination. Some of these conditions, such as retinal neovascularization, usually did not reduce visual acuity; others, such as cataract and vitreous hemorrhage, were potentially treatable so that visual acuity could be maintained or even improved.

The most important observation was the low incidence of active retinal neovascularization at the time of the final examination. This was present in only three of the 130 eyes, and in each of these cases a single area of flat neovascular tissue was present. This tissue was not causing apparent complications, although it is possible that this was the origin of postoperative vitreous hemorrhage in one eye. The low incidence of flat retinal neovascularization and the absence of further preretinal (elevated) fibrovascular proliferation support the impression that initially successful vitrectomy has an important long-term beneficial effect by preventing virtually all further fibrovascular proliferation from the retina, which is the usual cause of blinding complications. The mechanism by which this favorable effect occurs is unproven, although it is likely that the posterior vitreous surface is a necessary scaffold for proliferation of preretinal fibrovascular tissue.[2,3] The explanation for the low incidence of active neovascular tissue growing along the inner retinal surface is less certain, although adjacent cortical vitreous gel may also be important for flat growth of neovascular tissue. It is also possible that vitrectomy alters the physiology of the eye so that angiogenic substances produced by hypoxic retinal tissue diffuse more rapidly, thereby limiting the effect on adjacent retina.

Summary

A long-term follow-up study of 130 eyes that had had initially successful vitrectomies for complications of proliferative diabetic retinopathy showed that only ten (8%) eyes had significant worsening of visual acuity at final examination (18 to 63 months after surgery), compared to the best visual acuity recorded during the first six months after surgery. Active flat retinal neovascularization was present at final examination in only three eyes (2%), and in no case did elevated fibrovascular proliferation occur. The type and incidence of important complications during the early and later postoperative periods indicated a low rate of late complications.

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CLOSED VITRECTOMY FOR THE VITREOUS TOUCH SYNDROME

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Vitreous contact with corneal endothelium is associated with aphakic bullous keratopathy in a significant number of cases.[1,2] The introduction of microsurgical closed vitrectomy techniques has provided a method for safely removing formed vitreous from such eyes.[3] We report the surgical and visual results in 38 consecutive cases in which closed vitrectomies were performed for the vitreous touch syndrome.

SUBJECTS AND METHODS

We examined 38 eyes of 35 patients who had undergone an elective senile cataract extraction and subsequently developed the vitreous touch syndrome. In each instance the patient had experienced visual loss before examination at our institution, and each eye showed typical signs of bullous keratopathy associated with an area of vitreous touch. We did not subject any eyes with vitreous touch but without signs of corneal edema to surgery, and we did not perform vitrectomies on eyes in which apparently irreversible corneal changes had occurred.

The anterior chambers were of normal depth in all cases. The ages of the 35 patients ranged from 52 to 88 years and averaged 70 years. Twenty-three (66%) were women. The left eye was involved in 20 (53%) of the cases. Intracapsular cataract surgery had been performed in 27 (71% of the eyes, and phacoemulsification with posterior capsulectomy had been the procedure in the remaining 11 (29%).

Figure 1 shows the length of time between cataract surgery and our diagnosis of vitreous touch syndrome. Only three eyes (8%) were seen within three months of their cataract procedure, and 18 (43%) were seen within two years of cataract surgery. A precise documentation of the time the vitreous face initially made contact with the endothelium was unavailable in most cases. We were able to obtain a history of symptoms related to the onset of corneal edema in only 15 instances. These began four days to one year before our initial examinations. Most patients were referred after the discovery of corneal changes by their cataract surgeon.

The corrected preoperative visual acuities of these eyes are shown in Figure 1. There was no correlation between visual acuity and the duration of the patient's symptoms or the length of time between cataract surgery and the examination. Visual acuities were related to the severity and extent of the corneal edema.

We performed vitreous surgery through the pars plana in 33 cases and through the corneoscleral limbus in five. Vitreous was removed with the Douvas Rotoextractor in nine cases and with the Ocutome unit in the remaining 29. We did not use intraocular illumination because the hyaloid face could be seen adequately with the operating microscope at the time of surgery. At surgery, we attempted to remove enough formed vitreous to prevent its moving forward of the iris plane postoperatively. No attempts were made to penetrate the posterior vitreous face with the vitrectomy

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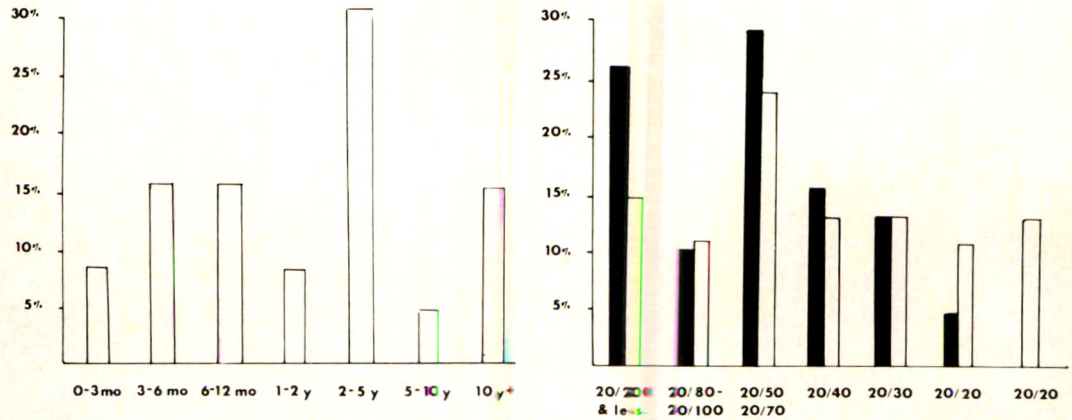


Fig. 1 (Wilkinson and Rowsey). Left, Length of time between cataract surgery and vitrectomies. Right, Visual acuities preoperatively (solid bars) and five to seven weeks postoperatively (open bars).

instrument or to remove large amounts of formed vitreous behind the iris.

Results

Most of the formed vitreous was removed from the anterior chamber and the vitreous touch syndrome was eliminated in all cases. Figure 1 shows the visual acuities recorded five to seven weeks postoperatively. Figure 2 compares the preoperative and postoperative visual acuities. No patient with preoperative visual acuity worse than 6/24 (20/80) obtained 6/12 (20/40) or better in the first seven weeks after surgery. Of eyes with preoperative visual acuities of 6/24 (20/80) or better, 19 (73%) obtained 6/12 (20/40) or better in the same follow-up period. Only one patient with preoperative visual acuity better than 6/12 (20/40) lost visual acuity during this brief follow-up period, and this was from 6/9 (20/30) to 6/12 (20/40). Most of the eyes maintained their preoperative visual acuities or showed improvement. Follow-up studies in these eyes have ranged from four months to five years, averaging 17 months. None of the ten eyes with visual acuities of 6/24 (20/80) or less in the early postoperative period improved significantly over the months that followed, and three of these have undergone penetrating keratoplasties. Two of the nine eyes with early postoperative visual acuities in the range of 6/15 to 6/22 (20/50 to 20/70) have maintained clear corneas for over two years. One eye has a visual acuity of 6/7.5 (20/25), but visual acuity in the other has dropped to 6/60 (20/200) because of ischemic optic neuropathy. Four of the nine eyes in this group have maintained visual acuities in the range of 6/15 (20/50) for a few months, and the remain-

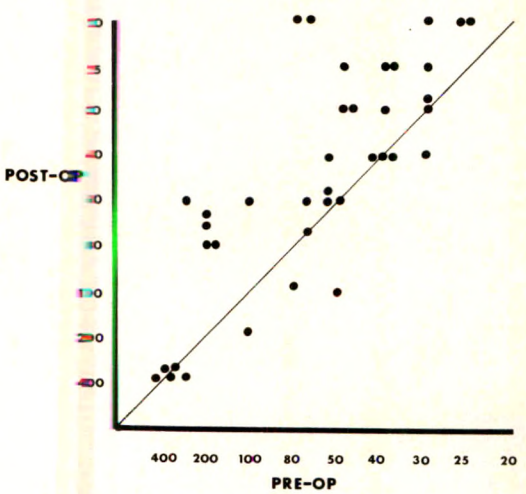


Fig. 2 (Wilkinson and Rowsey). Visual acuities preoperatively and five to seven weeks postoperatively.

ing three eyes have had a progression of their bullous keratopathy. Most eyes with visual acuities of 6/12 (20/40) or better five to seven weeks postoperatively have maintained visual acuity and clear corneas during follow-up. Figure 3 shows the visual outcome for these 19 eyes.

We performed preoperative and postoperative pachymetry readings on the four most recent cases. In the three eyes in which significant clearing of the corneal edema occurred with improved vision, corneal thickness was reduced by at least 0.1 mm within six weeks of vitreous surgery. In the fourth case corneal thickness was reduced only 0.03 mm, and a penetrating keratoplasty was required.

We were unable to correlate the anatomical and visual results with either the duration of the patient's symptoms or the length of time between cataract surgery and vitreous surgery. A precise documentation of the extent of vitreocorneal contact was not available in most cases, but eyes with relatively localized areas of central corneal edema and without evidence of widespread endothelial damage tended to fare better than those with a more diffuse disease process. We were unable to correlate the visual outcome with preoperative endothelial cell counts. The range of these cell counts in eyes in which visual acuity returned to 6/9 (20/30) or better was from 3,000 cells/mm^2 to 790 cells/mm^2.

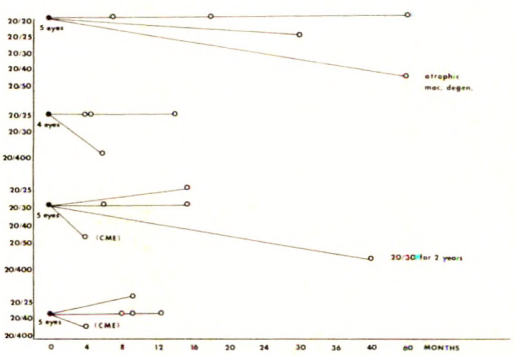


Fig. 3 (Wilkinson and Rowsey). Visual acuities during follow-up of 19 eyes that achieved 6/12 (20/40) or better in the early postoperative period. CME indicates cystoid macular edema.

Complications caused by vitreous surgery occurred in five eyes. A tiny vitreous strand attached to the vitrectomy corneal incision was detected one day after surgery in one eye. This was successfully removed 48 hours after the initial vitrectomy. Cystoid macular edema developed in this eye and in three others from five weeks to three months after surgery. In all four eyes, the visual loss associated with the onset of macular edema occurred after initial visual improvement and clearing of the cornea. The macular edema cleared in a few months in two of these eyes. Visual acuity in these eyes is now 6/9 (20/30) or better. The remaining two eyes have been followed up for four months, and they continue to show macular edema. Visual acuity is 6/15 (20/50) in one eye and 6/30 (20/100) in the other. A rhegmatogenous retinal detachment developed nine months after vitreous surgery in one of the eyes with macular edema. This detachment did not affect macular function, and it was repaired successfully. A tiny hemorrhage occurred at the pars plana incision site in a fifth eye, but the visual outcome was excellent.

Discussion

The vitreous touch syndrome following uncomplicated intracapsular cataract extraction occurs in 3% to 13% of cases.[4] Studies of such changes in the vitreous body suggest that most instances of contact between the vitreous and corneal endothelium take place within a few months of cataract surgery.[5] Although many of these eyes do not immediately develop clinical signs of corneal edema, the incidence of this problem may increase with the duration of contact between the vitreous gel and endothelial cells.[6] Since over one half of the eyes in this series developed corneal edema

more than two years after lens extraction, it appears that bullous keratopathy caused by the vitreous touch syndrome frequently requires years to become evident clinically.

The precise mechanism whereby vitreous causes apparent endothelial damage remains unknown, although recent animal studies suggest that solid collagenous elements in the formed vitreous may be harmful to endothelial cell function.[7] The cornea may initially compensate for relatively localized endothelial damage by replacing damaged endothelial cells with healthier cells from surrounding areas. Recent morphologic studies on mechanisms of endothelial healing indicate that focal corneal damage can ultimately result in an increase in cell size and a decrease in cell population over a large extent of the cornea.[8] Ultimately, compromised endothelial cells may become incapable of maintaining a normal state of corneal dehydration, and edema may occur in association with irreversible changes which extend well beyond the area of vitreocorneal contact.[9]

Many forms of therapy have been proposed for the vitreous touch syndrome.[1-4] Most cases that occur early, and particularly those associated with a shallowing of the anterior chamber, can be treated medically. In cases with a persistent attachment of the vitreous face to the cornea, in which progressive signs of aphakic bullous keratopathy are observed, surgical therapy appears to be indicated. Although the removal of vitreous in association with other maneuvers has been recommended in this syndrome for decades,[1,2] only recently have closed vitrectomy techniques made such vitreous removal safe.[3]

We were able to eliminate successfully the vitreous touch syndrome in each of the 38 eyes in this consecutive series, and few severe complications occurred. Although no eyes with a severe loss of visual acuity and extensive corneal edema had a dramatic improvement, in a majority of eyes with preoperative visual acuities of 6/15 (20/50) or better, we were able to eliminate a progression of the corneal edema and to provide good stable visual acuity. Pachymetry studies performed in a small number of these eyes showed a prompt thinning of the cornea after removal of the vitreous in eyes that ultimately had a good result. The visual results and pachymetry studies indicate that this progressive syndrome can be interrupted and frequently cured if surgery is performed when corneal edema is only relatively localized. Any eye exhibiting vitreocorneal contact may develop progressive bullous keratopathy if the patient lives long enough. We believe such eyes should be examined periodically, and any clinical indication of endothelial damage or corneal edema should prompt serial pachometry studies. Surgery is indicated after unequivocal signs of progressive corneal edema have occurred.

Summary

We used closed vitrectomy techniques to remove formed vitreous from the anterior chamber in a consecutive series of 38 eyes with the vitreous touch syndrome. The visual results in these cases were related to the severity of corneal edema and the visual acuity recorded preoperatively. Most patients with preoperative visual acuities of 6/15 (20/50) or better demonstrated a clearing of corneal edema and an improvement in vision. Of eyes achieving visual acuities of 6/12 (20/40) or better within six weeks of vitreous surgery, 14 (74%) maintained this level during follow-up. No eyes with preoperative visual acuities of 6/90 (20/300) or worse achieved good results. The incidence of complications of vitrectomy surgery was acceptable, although cystoid macular edema developed postoperatively in at least 10%. Closed vitrectomy techniques provide a safe and effective means of treating the vitreous

touch syndrome when corneal edema is localized.

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Ophthalmic miniature

Once he begins to believe that he, rather than his knowledge and technical skill, is the source of the cure, danger lurks in the image reflected back from his patient's eyes. If the doctor looks into those eyes, the image that he sees is—God. But that image is the enemy.

Lawrence Shainberg, *Brain Surgeon: An Intimate View of His World*
New York, Fawcett Crest, 1979

EXTENDED-WEAR HYDROCURVE AND SAUFLON CONTACT LENSES

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The development of extended-wear contact lenses has given the ophthalmologist an alternative to intraocular lenses for the correction of aphakia. Extended-wear lenses have undergone many clinical trials in the last five years,[1] with follow-up data available for periods ranging from six months to three years.[1] For the most part, the best corrected visual acuity obtained with these lenses has been excellent and comparable to the results obtained with intraocular lens implantation.[1,2] Nevertheless, experience gained to date indicates that extended-wear lenses will not replace intraocular lenses since they do have certain disadvantages.[3]

We previously published two other studies on extended-wear lenses[4,5]; our third report compares moderately and highly hydrophilic contact lenses.

SUBJECTS AND METHODS

All patients referred for extended-wear lenses underwent a complete ophthalmologic evaluation which included a careful anterior segment examination with Schirmer tear strip measurements, corneal pachymetry, slit-lamp biomicroscopy without fixation lights, corneal anesthesiometry, and clinical specular microscopy. After the patients signed informed consent forms, we took extraocular photographs.

We fitted the lenses after determining the refraction (manifest or cycloplegic examination, or both, when indicated), central corneal keratometry readings, and, when indicated, horizontal corneal diameters. From Jan. 1, 1978, through Nov. 1, the only lenses available to us were the 45% water-by-weight Hydrocurve II (HCII-45) and the 55% water-by-weight Hydrocurve (HCII-55) lenses. After that time we also fitted patients with 79% water-by-weight Sauflon lenses. Using standard techniques for extended wear,[1] we fitted the lenses. After the appropriate overrefraction, lenses were ordered and later dispensed.

Patients were reexamined 24 to 48 hours after the initial fitting, one week later, two weeks later, and monthly thereafter. Each patient received an information package telling him about possible adverse reactions. Patients were instructed to return immediately if they had any combination of pain, decreased visual acuity, or undue ocular congestion. At each examination we performed an overrefraction, if necessary, and a slit-lamp biomicroscopic examination of the anterior segment, including corneal pachymetry with lenses in place. We examined the lenses in situ for deposits, lens abnormalities, and the excursion of the lens upon blinking. The patients continued to use topically applied medications (except epinephrine-containing ophthalmic drops). The majority used baby shampoo for eyelid margin hygiene.

Those patients who developed an increase in corneal thickness without an associated decrease in best corrected visual acuity were instructed to increase their frequency of blinking and were given low-viscosity artificial tears to use as often as every 30 minutes while awake. Those patients who developed an in-

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crease in corneal thickness associated with a decrease in best corrected visual acuity were similarly treated and reexamined in 24 to 72 hours. In the cases in which the corneal and visual acuity factors failed to respond, we removed the lenses for a minimum of one week and refitted the patients with lenses which were flatter or more hydrophilic, or both.

Following the manufacturer's protocol, the Hydrocurve lenses were removed weekly, by us or by the patient, for cleaning, while the Sauflon lenses were left in situ and only removed for cleaning when clinically indicated. We replaced lenses when they were lost or when abnormalities appeared. We examined 11 acute red eyes immediately and performed bacterial and viral cultures of the conjunctival sac and lens when clinically indicated. Any corneal abnormality, such as neovascularization, was measured with the slit-beam ruler of the slit lamp and recorded in the chart.

There are different definitions of success,[2] but we considered our patients successfully fitted if they were able to wear the lenses continuously for more than two weeks while maintaining their best possible corrected visual acuity.

Results

From Jan. 1, 1978, through Oct. 1, 1979, 64 patients were referred for the fitting of extended-wear contact lenses. Of these, we considered 43 patients (70 eyes) acceptable candidates. Follow-up ranged from two to 20 months (Table 1).

We fitted 34 myopic eyes and 36 monocular aphakic eyes. Using the previous definition of success, 94% of the myopic cases (32 of 34) and 67% of the aphakic cases (24 of 36) were successful, with a combined overall success of 80% (56 of 70 eyes) (Table 2). There were only two unsuccessful cases in the myopic series. One patient lost interest after one month and did not wish to continue although the best corrected visual acuity in both eyes was 6/6 (20/20). A second patient with myopia wore HCII-45 lenses for five months and later complained of irritation. We switched her to HCII-55 lenses; although she maintained good visual acuity and wore the lenses continuously for two months, she requested that we drop her from the program.

Of the 14 aphakic eyes in the HCII-45 group (Table 2), we fitted nine successfully. Two patients who were able to maintain good visual acuity without corneal edema but who complained of ocular irritation were successfully refitted with HCII-55 lenses. Three others developed

TABLE 1

Duration of follow-up

Lens	No. of Eyes	Follow-up (mos) Range	Follow-up (mos) Average
HCII-45			
Myopic	26	5–16	12
Aphakic	14	2–17	8
HCII-55			
Myopic	6	20	—
Aphakic	10	3–13	9
Sauflon			
Myopic	2	2–12	5
Aphakic	12	2–12	5

TABLE 2

Successful cases

Lens	Total No.	Successful No.	Successful %
HCII-45			
Myopic	26	24	92
Aphakic	14	9	64
HCII-55			
Myopic	6	6	100
Aphakic	10	6	60
Sauflon			
Myopic	2	2	100
Aphakic	12	9	75
Total			
Myopic	34	32	94
Aphakic	36	24	66

corneal anoxia (corneal edema, ocular and ciliary congestion, and aqueous cells and flare with decreased visual acuity) and were fitted with HCII-55 lenses. We had success with six of ten aphakic eyes fitted with HCII-55 lenses (Table 2). One of the patients who had complained of ocular irritation with the HCII-45 lens continued to complain of ocular irritation with the HCII-55 lens, although by our criteria the eye was successfully fitted. Nevertheless, this patient asked to be dropped from the study and returned to successful daily wear with the HCII-55 lens. Three of the patients who developed corneal edema with HCII-45 lenses and were refitted with HCII-55 lenses again developed corneal edema and were subsequently fitted with Sauflon lenses.

We fitted nine of 12 aphakic eyes with Sauflon lenses (Table 2). Two of the three patients who developed edema with Hydrocurve lenses also developed edema with Sauflon lenses and were switched to daily-wear lenses. One patient with aphakia who was initially fitted with a Sauflon lens experienced decreased visual acuity and developed corneal edema. Daily wear of a hard contact lens was successful.

We tested various factors in this study, but analysis of the successfully fitted lenses failed to disclose any trends, that is, there were no statistically significant failures because of any one quality of a lens.

Analysis of the age distribution in the various groups failed to demonstrate any significant differences between the successful and failed cases (Table 3).

Failures caused by corneal edema or decreased visual acuity occurred within the first 30 days of continuous wear, with the majority of eyes developing edema within two weeks of continuous wear (Table 4). With corneal pachymetry we found that readings of 0.03 mm more than baseline thickness were significant.[6] Several aphakic eyes developed an increase in corneal thickness of 0.06 mm more than their baseline reading without an associated decrease in visual acuity (Table 4). Three of the four patients with aphakia in this category wore HCII-45 lenses. Twelve eyes (17%) developed an increase in corneal thickness of more than 0.08 mm, a finding always associated with a decrease in visual acuity of one or two lines (Table 4).

All of the aphakic eyes that developed corneal edema, with or without a decrease in visual acuity, were treated with low-viscosity artificial tears once each hour; we instructed these patients to blink more frequently and reexamined them within 24 to 48 hours. We gave

TABLE 3

AGE DISTRIBUTION

Lens	Success (Age in yrs)		Failures (Age in yrs)	
	Range	Average	Range	Average
HCII-45				
Myopic	16–37	25	24–47	37
Aphakic	62–76	69	51–72	65
HCII-55				
Myopic	24–26	25	—	—
Aphakic	64–72	70	51–72	63
Sauflon				
Myopic	15	—	—	—
Aphakic	66–70	67	51–72	64

TABLE 4

CORNEAL ANOXIA

Lens	Corneal Swelling	
	<0.06 mm	>0.08 mm
Aphakic		
HCII-45	3	4
HCII-55	1	3
Sauflon	0	5
Myopic	0	0

those patients whose corneal edema was unassociated with visual loss contact lenses with a higher water content; they were ultimately successful. The 12 aphakic eyes with visual loss and an increase in corneal thickness of more than 0.08 mm also tended to develop edema with flatter, more hydrophilic contact lenses. These patients ultimately switched to daily-wear lenses. Patients who developed edema with HCII-45 lenses were switched to HCII-55 lenses, but tended to develop edema with those lenses also. When switched to 79% Sauflon lenses, they still developed edema. Therefore, of the 12 aphakic eyes that developed edema, eight were from patients whose corneas became edematous with all lenses tested. This finding may reflect our patient selection, but suggests that a healthy cornea which decompensates with any well-fitting lens probably does so even when fitted with a looser-fitting or more hydrophilic lens.

Analysis of the best corrected visual acuities showed that 100% of the myopic and aphakic eyes achieved visual acuities equal to or better than 6/12 (20/40) (Table 5) and 64 of 70 (91%) eyes achieved equal to or better than 6/9 (20/30). For a patient with more than 1.5 diopters of corneal astigmatism, a spectacle overrefraction had to include an astigmatic correction to achieve the best possible visual acuity.

Numerous lens changes were required during the study to maintain the level of best corrected visual acuity. The reasons for lens changes included lens deformities (chipping or tearing), lens deposits, lens loss, and refractive error changes unassociated with changes in corneal curvature or thickness. Table 6 shows the number of lens changes required to achieve or maintain, or both, the best corrected visual acuity. A total of 198 lens changes were required to successfully fit 70 eyes (2.8 lens changes per eye per

TABLE 5

BEST CORRECTED VISUAL ACUITIES

Lens	Visual Acuity			
	6/4.5–6/6 (20/15–20/20)	6/7.5 (20/25)	6/9 (20/30)	6/12 (20/40)
HCII-45				
Myopic	22*	—	2	—
Aphakic	3†	3†	2*	1†
HCII-55				
Myopic	6	—	—	—
Aphakic	1‡	2*	0†	1
Sauflon				
Myopic	2	—	—	—
Aphakic	2†	2*	2	2†

*With two failures.
†With one failure.
‡With three failures.

TABLE 6
LENS CHANGES

No. of Changes	HCII-45		HCII-55		Sauflon	
	Myopic	Aphakic	Myopic	Aphakic	Myopic	Aphakic
0	10	1	—	3	—	1
1	2	4	—	3	—	1
2	4	4	—	1	—	3
3	4	2	2	1	2	—
4	4	—	—	—	—	2
5	—	—	2	—	—	—
6	2	6	—	—	—	—
Total lenses fitted	76	71	20	16	8	22

year). There was no significant difference in the number of lens changes required per eye for aphakic or myopic patients. The majority of lens changes were designed to produce looser-fitting (flatter-fitting) lenses and the majority of the remaining changes were due to lens-power changes, which were required after one or two weeks of wear. Patients who required power changes in their lenses saw well for a week or two and then complained of a mild distortion in distance vision. The plus-power changes required in the lenses were between 0.25 and 1.0 diopter and occurred with both myopic and aphakic lenses. Of the lens changes, 158 (80%) took place in the first two months. The major causes for lens replacement after that time were lens problems (chipping, cracking, loss, deposits, and the like) and not ocular complications.

Lens deposits, which occurred as soon as one week into the study, were the most frequent complication in the series (Table 7). If we noted even one deposit, we recorded it. Many patients could tolerate a surprising amount of lens deposits since only six of 198 lens changes were caused by deposits. We found that once a lens developed deposits, recurrent deposits would occur, even if it was thoroughly cleaned. Certain patients tended to produce more deposits than others[4]; we

TABLE 7
COMPLICATIONS

Complication	Eyes With Hydrocurve Lenses		Eyes With Sauflon Lenses	
	No.	%	No.	%
Deposits*	36	20	6	20
Ocular congestion	2	3	0	0
Conjunctivitis†	4	6	0	0
Neovascularization	4	6	2	3
Epithelial defect	0	0	1	1.5
Lenses lost*	6	9	3	10
Lens abnormality*	19	9	7	23
Corneal ulcer	0	0	0	0
Total lenses fitted	183		30	

*Per lens.
†Clinical conjunctivitis with negative culture.

encouraged these patients to use low-viscosity and hypo-osmotic artificial tears frequently and to maintain good eyelid margin hygiene with daily baby shampoo scrubs. This form of treatment controlled deposits in only one third of the deposit-forming eyes.

In this study, we fitted a total of 168 HCII-45 and HCII-55 lenses (56 eyes). Of these, 33 (20%) developed deposits. Thirty Sauflon lenses were used in the study and six (20%) of these developed deposits. Therefore, there was no significant difference in the incidence of deposits for moderately and highly hydrophilic contact lenses.

The complications of extended-wear contact lenses are well documented.[1,8] In our study, complications were few (Table 7). We carefully selected the patients who were fitted from a larger population of referred patients. Furthermore, we examined the patients frequently, requiring 14 visits per patient over a 12-month period. We encouraged the patients to return immediately if they developed any signs of pain, ocular congestion, or decreased visual acuity.

One complication was corneal neovascularization of less than 1 mm, which occurred in six aphakic eyes. With the frequent use of artificial tears, increased blinking, and the fitting of looser or more hydrophilic lenses, the vascularization was controlled in five of the six eyes. We did not use and do not recommend topically applied corticosteroids. In the one case that did not respond to our treatment, lens wear was stopped and restarted on a regimen of 24 hours on, 24 hours off. This controlled the neovascularization.

Two myopic eyes developed ocular congestion and an additional four had red eyes consistent with adenoviral keratoconjunctivitis (Table 7). Viral cultures were negative in these patients and the ocular signs and symptoms cleared within 48 to 72 hours after lens wear was stopped. Based on our previous experience with similar cases, we suspect that the cause of these red eyes may be the preservatives in the solutions used for disinfection.[9] There were no cases of giant papillary conjunctivitis in this study.

When we removed contact lenses for quarterly examinations, we found minimal superficial punctate epithelial staining in many of the patients who were asymptomatic. In only one patient did we find an epithelial defect underneath a soft contact lens; this occurred in the patient who had anoxic overwear syndrome.

The patients lost 15 (9%) Hydrocurve lenses and three (10%) Sauflon lenses (Table 7). Lens abnormalities, including chipping, tearing, and warping, occurred in 9% of the Hydrocurve lenses and 23% of the Sauflon lenses (Table 7). Infectious corneal ulceration, which has been reported with extended-wear lenses outside of the United States,[1] did not occur in any of our selected patients (Table 7).

Discussion

Analysis of these patients clearly demonstrates that extended-wear lenses can be successful with a minimum of complications. However, these lenses cannot be fitted without frequent and careful follow-up examinations, which require numerous lens changes. If patients are carefully selected, informed of possible complications, and examined frequently, the success rate will be high.

In this series, we fitted 79% of our selected patients with moderately hydrophilic (HCII-45 and HCII-55) or highly hydrophilic (Sauflon) lenses. Some patients who were unsuccessful with the moderately hydrophilic lenses were successful when refitted with the highly hydrophilic lenses, but the reverse was not true. The major reason for failure was the development of lens intolerance,

shown by increasing corneal edema (>0.08 mm) and a decrease in visual acuity of one or two lines, Patients who developed this level of anoxia did so with both types of lenses.

Some patients who developed anoxia did not respond to the refitting of lenses, the frequent use of artificial tears, or increased blinking, which suggests that oxygen exchange with these hydrophilic gel lenses was insufficient to meet the oxygen demands of their corneas. A recent study [10] suggested that minimal, if any, tear exchange takes place under hydrophilic gel lenses and that most of the transmitted oxygen must come through the lens. When we studied those patients who developed anoxia, we found that all but one had corneal thicknesses of more than 0.54 mm before fitting and all but one had central corneal endothelial cell densities of less than 1,200/mm^2 while the endothelial cell densities of the successful patients were greater than 1.900/mm. The increased corneal thicknesses and decreased endothelial cell densities suggest that we can preselect successful candidates.

Possibly many of these failed cases could have been fitted with a very thin (0.05-mm), minimally hydrophilic contact lens or a silicon contact lens able to transmit more oxygen.[1] Unfortunately, these lenses were not available at the time of this study.

Lens deposits were a frequent complication with all lenses used in this study (Table 7). Compared with our previous extended-wear series,[4,5] we were partially successful in modifying the incidence and degree of lens deposits by the frequent use of low-viscosity or hypo-osmotic artificial tears combined with baby shampoo eyelid scrubs. Although only six eyes required new lenses because of deposits, the incidence was significant enough to produce mild ocular irritation and numerous complaints. At present all we can do is increase the frequency of lens cleaning, but in the future we may be able to use lenses whose physical or chemical make-up will resist deposit formation.

Red eyes in contact-lens wearers should be treated as clinically indicated. In most instances, lens wear should be stopped, lenses should be disinfected, and the patient treated appropriately. We, as well as other investigators, have noticed an increased incidence of red eyes in patients using daily-wear and extended-wear contact lenses.[9] Many of these episodes may be caused by mild infections, but it is our impression that a toxic or allergic reaction to the preservatives in the lens solutions may play a role.

Corneal neovascularization occurred in six aphakic eyes in the present series (Table 7) and has been reported to occur in up to 7% of eyes fitted for extended-wear lenses.[1] Neovascularization can occur with or without clinical evidence of corneal edema, and can be controlled by increasing the level of oxygen available to the corneal surface by fitting looser or more hydrophilic lenses or changing the wearing schedule. Topically applied corticosteroids will not stop the progression of the vessels.

Continuous-wear lenses do produce a mild irritation of the corneal epithelium in the form of a superficial punctate keratitis. Many patients are asymptomatic and the epithelial changes are only noted when the lenses are removed. Although numerous corneal epithelial defects have been reported with extended-wear lenses, only one patient in this series developed an epithelial defect and that occurred in an aphakic patient who developed the anoxic overwear syndrome.

There were no corneal ulcers in this study, although corneal ulceration remains the major sight-threatening complication of extended-wear lenses. The

highest incidence of corneal ulceration (6%) occured in three of 50 unselected patients who were fitted with Sauflon lenses.[11] If patients are carefully selected and followed up, the incidence of this complication will remain low.

Our study, as well as many other studies published in the last two years, clearly demonstrated that extended-wear soft contact lenses are useful for correcting aphakia and myopia. There is no doubt that extended-wear contact lenses are not for everyone since patients have to be carefully selected, fitted, and followed up in order to obtain good results.

These lenses will not replace intraocular lenses, but do expand the ophthalmologist's ability to correct aphakic eyes. Both intraocular lenses and extended-wear lenses have advantages and disadvantages.[3] Analysis of results with extended-wear lenses to date[1] demonstrates that extended-wear contact lenses can be highly successful while producing a low incidence of ocular complications. The major complications with intraocular lenses occur about the time of surgery, whereas the major complications of extended-wear lenses occur after the lenses are fitted and continue to occur throughout the time the lenses are worn.

Summary

Fifty-six of 70 eyes selected for extended-wear lenses were successfully fitted with moderately hydrophilic (Hydrocurve II and Hydrocurve II-55) and highly hydrophilic (Sauflon) contact lenses. Thirty-two of 34 myopic eyes and 24 of 36 aphakic eyes were successfully fitted. The myopic eyes were followed up for two to 20 months and the aphakic eyes for two to 17 months. Ninety-one percent of all eyes achieved visual acuity better than or equal to 6.9 (20/30). Approximately 2.8 lenses per eye per year were required for success.

The major complication was corneal edema (anoxic overwear); some of these cases responded to the fitting of looser, more hydrophilic lenses, frequent use of artificial tears, and increased blinking. There was no difference in the incidence of lens deposits between the moderately and highly hydrophilic lenses, and in only six eyes did lens deposits necessitate a lens change. There were no corneal ulcers in this study.

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REMOVAL OF AN INTRAOCULAR FOREIGN BODY FROM THE OPTIC NERVE HEAD

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Penetrating injuries to the globe in which a metallic (iron) foreign body becomes embedded exclusively within the optic nerve head are rare occurrences that can produce immediate and severe damage to the optic nerve, with concomitant loss of vision.[1] The mechanism for initial loss in these cases is by direct damage to the axons and supportive cells of the optic nerve, by vascular occlusive phenomena, or both. Vascular occlusion can take place when there is secondary swelling of damaged tissue leading to compression of important vascular channels with resultant occlusions such as branch retinal artery occlusion. In other cases, the foreign body itself may directly compress a vascular channel, leading to a retinal vascular occlusion.

A delayed mechanism for visual loss in these cases[2-4] concerns iron-containing foreign bodies known to release ferrous ions that eventually saturate iron-binding molecules, for example, apoferritin. The excess unbound ferrous ions are then free to interfere with specific vital intracellular enzymatic systems such as the electron transport chain. Thus, the unbound ferrous ions poison certain cells that are highly active metabolically, leading to siderosis bulbi. Apparently the photoreceptor cells and the retinal pigment epithelium are sensitive to excess ferrous ions.[2] For these reasons it is essential to remove the iron-containing foreign body, if possible, before irreversible chemical damage to ocular tissues takes place. Late stages of ocular siderosis may also involve damage to the trabecular meshwork with resultant glaucoma.[5]

Magnetic foreign bodies embedded in the optic nerve head may not respond to strong magnetic fields applied at the pars plana region[6-8] or to magnetic intravitreal probes because the mass of the foreign body is small and it is too firmly embedded in optic nerve tissue. Such cases present complex management problems and require a direct surgical approach to the surface of the optic nerve head through incisions in the pars plana region.[9,10] A bimanual pars plana vitrectomy approach with the use of a microscalpel and foreign body forceps may be required to remove the metallic foreign body from the surface of the optic nerve head.[11] We describe a case that shows this technique.

CASE REPORT

A 29-year-old man was struck in the left eye while hammering a metal fender. He had a sharp pain in the left eye lasting less than a minute, followed by progressive blurred vision in the eye.

The ocular findings two hours after the initial injury were as follows: visual acuity, with correction, was R. E. 6/6−2 (20/20−2) and L. E.: counting fingers at 2 feet. Manifest refraction could not improve the visual acuity in either eye. Extraocular motility studies revealed a full range of motion of either globe in all standard fields of gaze and the eyes were straight in the primary position of gaze. Function of cranial nerves II, III, IV, V (ophthalmic division), VI, and VII was grossly intact bilaterally. Formal visual fields were not performed at this time, but confrontation visual fields were grossly intact bilaterally. The eyelids, ocular adnexae, and lacrimal gland apparatus were normal in both eyes. The palpebral

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and bulbar conjunctivae were within normal limits bilaterally except for a localized bulbar hemorrhage at the 7 o'clock meridian in the left eye, which was the entrance wound for the intraocular foreign body. The corneas and scleras were within normal limits; however, the bulbar hemorrhage in the left eye obscured the underlying sclera. The intraocular pressures were R. E.: 15 mm Hg and L. E.: 38 mm Hg by applanation tonometry. Slit-lamp examination of the anterior chambers showed that they were of moderate depth with grade 3 open anterior chamber angles for 360 degrees bilaterally. The right anterior chamber was clear and the left anterior chamber had 1+ flare and 1+ red blood cells. No hyphema was noted in the left eye. The irides were brown and the pupils were equal, round, dilated, and fixed because of mydriatic medications, which had been instilled by the referring ophthalmologist one hour before this examination. Therefore both pupils were 7 mm in diameter, round, and nonreactive to light or accommodative stimuli. A small iris nevus noted in the left eye was flat with well-circumscribed borders and appeared to be benign. The lenses were clear bilaterally. The right vitreous cavity was clear and unremarkable, whereas the left vitreous cavity had a moderately dense hemorrhage posteriorly overlying the optic nerve head. The anterior and midzones of the left vitreous body also contained 1 to 2+ red blood cells.

Ophthalmoscopic examination showed a normal size and shape to the right optic nerve head. The right optic nerve head had sharp borders with a cup:disk ratio of 0.2 in the horizontal meridian and a vein:artery ratio of 3:2. The macula was normal and the peripheral retinal examination with scleral depression did not disclose any retinal breaks or tears and a zone of 1+ white with pressure was present extending from the equator peripherally to the ora serrata and running for 360 degrees.

Ophthalmoscopic examination of the left eye disclosed a black metallic-looking object in the region of the optic cup, which was partially obscured by overlying fresh vitreous hemorrhage (Fig. 1). The vitreous hemorrhage obscured fine details of the optic disk and the macula. Peripheral indirect ophthalmoscopy with gentle scleral depression failed to show any retinal breaks or tears in the left eye. The lens periphery also appeared to be clear without any visible tract or damage on three-mirror contact lens biomicroscopy of the left eye.

On the basis of these findings, a diagnosis of a metallic intraocular foreign body embedded in the optic nerve head was made. The patient was immediately admitted to the hospital in preparation for a pars plana vitrectomy and removal of the foreign body. The patient had foreign body X-ray localization films that indicated the foreign body to be just behind the globe. The patient was given 0.5 ml of tetanus toxoid subcutaneously and systemic oxacillin intravenously. Initially, the hammer and object being hammered (fender) were found to be magnetic.

For the next several hours massive hemmorrhage continued into the vitreous cavity despite a high intraocular pressure of 38 mm Hg as measured by applanation tonometry. The vitreous hemorrhage precluded any view of the optic nerve, retina, or choroidal tissues with only a black reflex seen on indirect ophthalmoscopy. Next a pars plana vitrectomy[11] was done with a Douvas Rotoextractor with a fiber optic light pipe connected to a probe tip with a 0.6-mm cutting port. Continuous rotary cutting action with mild suction was used in conjunction with a Zeiss operating microscope and a Banco corneal contact lens for visualization of the posterior pole of the globe. Almost all of the massive vitreous hemorrhage and vitreous gel were removed by this approach. At this point, the metallic foreign body was clearly visible embedded in the optic cup portion of the optic nerve head and compressing the adjacent inferotemporal retinal artery (Fig. 2). A branch retinal artery occlusion with infarcted (white-opaque) retina was noted in the inferotemporal quadrant for the first time. Retrobulbar tolazoline HCl (Priscolene) was given without any noticeable improvement. The intraocular pressure was decreased as well.

The Douvas Rotoextractor probe with accompanying light pipe was removed and an electromagnet (10,000 Gauss) was applied over the pars plana incision site in an effort to dislodge and remove the metallic foreign body from the optic nerve head, but numerous attempts at the pars plana site as well as with magnetized intraocular probes were unsuccessful in removing the foreign body. Because the surface of the foreign body was flush with the surface of the optic nerve head, forceps could not be used to remove it without considerable damage to the optic nerve and surrounding retinal and choroidal tissues. Because there was still a continuous ooze of red blood cells into the vitreous with poor visibility, the pars plana incisions were closed with 6-0 silk and the conjunctiva closed with 6-0 plain gut. Postoperatively, topical corticosteroids and mydriatics were given. High intraocular pressure in the injured eye was controlled with acetazolamide (Diamox) orally, which was subsequently tapered ten days after surgery. The eye remained normotensive.

Three days postoperatively A- and B-scan ultrasonography was performed (Fig. 3) and showed the foreign body to be acoustically dense and highly reflective of ultrasound waves. The B-scan indicated the position of the foreign body in the anterior region of the optic nerve head. An acoustically normal retrobulbar fat pattern and optic nerve shadow were evident. The vitreous was acoustically clear as expected after vitrectomy.

A fluorescein angiogram was performed three days after the first vitrectomy and showed an inferotemporal retinal artery occlusion with some retrograde flow of dye in the inferotemporal retinal artery (Figs. 2 and 4), along with a delay in retinal arterial filling time in the same quadrant. During the next several weeks the patient's eye quieted down and a superior altitudinal field defect was recorded on visual field testing of the left eye. His best corrected visual acuity was 6/21 (20/70).

The metallurgic analysis of the hammer and auto-

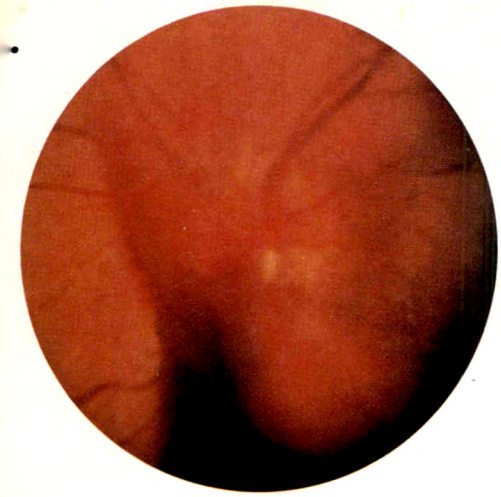
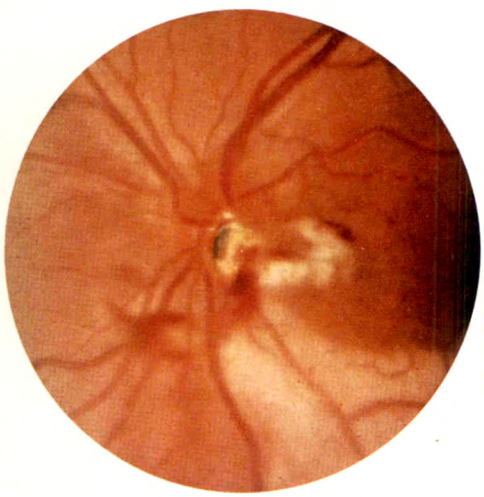


Fig. 1 (Zinn). Left eye, Fundus photograph of the patient two hours after sustaining a penetrating injury to the globe that resulted in a piece of steel being embedded in the cup of the optic nerve head. Vitreous hemorrhage partially covers the foreign body. There is no evidence of a branch retinal artery occlusion at this point. Within the next several hours a massive vitreous hemorrhage developed obscuring all details of the optic nerve and retina.



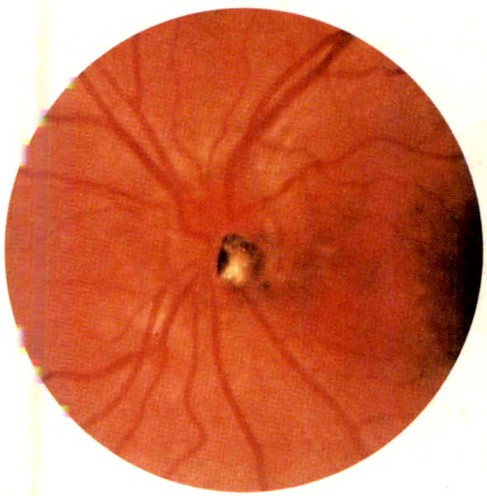


Fig. 2 (Zinn). Left, Fundus photograph of the patient three days after the first pars plana vitrectomy. Some residual preretinal hemorrhage is noted inferior to the optic nerve head. The metallic foreign body can be clearly seen in the center of the optic nerve head, embedded in the optic cup and compressing the inferotemporal retinal artery, which resulted in an inferotemporal retinal artery occlusion. Note the infarcted retina which is opaque. Several cotton wool spots are present at the temporal border of the optic nerve head. Right, Fundus photograph of the patient seven weeks after the first pars plana vitrectomy. Note that the retinal edema has cleared and a membrane partially covers the foreign body embedded in the optic nerve head.

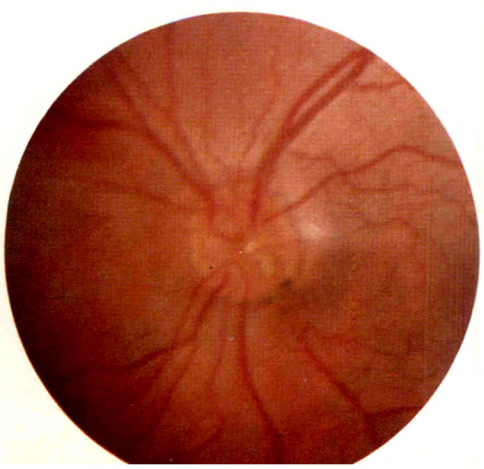


Fig. 6 (Zinn). Left eye, Fundus photograph ten months after removal of the foreign body from the optic nerve head. The gray area in the optic cup and in the inferotemporal quadrant of the optic nerve head is the remains of the original deep crater that housed the foreign body. The inferotemporal retinal artery on and adjacent to the optic nerve head is attenuated and barely visible on ophthalmoscopy. Yet one-quarter of a disk diameter distal to the optic nerve border, the inferotemporal retinal artery has a normal-looking blood column. Fine striae are also noted on the internal surface of the nasal macula. The retinal edema from the original branch retinal artery occlusion has cleared.

mobile fender that the patient was hammering at the time of injury showed both to be 98% iron and highly magnetic. Because of the possibility of ocular siderosis the patient was readmitted to the hospital eight weeks after the initial injury and had a second pars plana vitrectomy with removal of the intraocular foreign body from the optic nerve head. A 360-degree limbal peritomy was made and each of the four rectus muscles secured with a 4-0 silk suture. A pars plana incision 3 mm long and parallel with the equator was made 5 mm posterior to the corneoscleral limbus in the inferotemporal quadrant. A 4-0 Dacron horizontal mattress suture was used to secure the wound. A second pars plana incision was made in the superonasal quadrant with an Ocutome stiletto knife (20-gauge size) for the Ocutome endoillumination probe. The Zeiss operating microscope and Banco contact lens were used to visualize the posterior segment of the globe. A microscalpel was made by bending the tip of a 20-gauge needle with a needleholder. The angle of the tip was approximately 90 degrees to the major axis of the shaft. The back of the microscalpel was then hooked to neoprene tubing that was connected to a bottle of nonlactated Ringer's solution. The height of this bottle was 10 inches above the eye.

Because the vitreous was clear and its gel had been previously removed from the eye, another vitrectomy was not necessary. The foreign body was removed in the following manner: The tip of the microscalpel blade (20-gauge needle connected to the infusion system) was inserted into the vitreous cavity through the inferotemporal pars plana incision. The preplaced 4-0 Dacron suture was drawn up to provide a watertight seal. At the second pars plana incision site (superonasal quadrant) the Ocutome fiber optic endoilluminator was also inserted into the vitreous cavity. Both instruments were advanced to the region overlying the optic nerve head. The microscope illumination system was turned off and only the endoillumination system was used. The microscalpel was used to incise a moderately dense membrane that covered the foreign body and the tissue was deflected temporally. The surface of the foreign body had small pits. The tip of the microscalpel engaged one of the pits on the surface of the foreign body. The microscalpel was then used to rock the foreign body out of its crater within the cup of the optic nerve head. Once free of the optic nerve head, the foreign body rolled onto the surface of the macula. The microscalpel was withdrawn and Neubauer foreign body forceps were used to remove the foreign body from the surface of the macula and the eye through the pars plana incision in the inferotemporal quadrant. The pars plana incisions were closed with 6-0 silk, the 4-0 silk sutures were removed from the rectus muscles, and the conjunctiva was closed with 6-0 plain gut.

The patient's postoperative course was uneventful with treatment consisting of topical corticosteroids and mydriatic agents. The best corrected postoperative visual acuity was 6/7.5 (20/25) and repeat visual fields did not reveal any change in the altitudinal field cut (Fig. 5). Visual acuity and visual fields have remained unchanged for an 18-month follow-up period. The crater in which the foreign body was lodged filled in with some scar tissue several months later (Fig. 6) and has not changed in appearance over the past 15 months.

A fluorescein angiogram was performed one year after the removal of the foreign body (Fig. 7) and indicated little if any perfusion of the disk capillaries where the foreign body was originally embedded. Also, there was a delay in the retinal arterial filling and arteriovenous circulation time in the inferotemporal quadrant where the original branch retinal artery occlusion occurred.

Discussion

Runyan[1] stated that if a foreign body is 1 mm or greater in diameter and its location is in the center of the optic nerve head, no useful vision will be retained even if the foreign body is re-

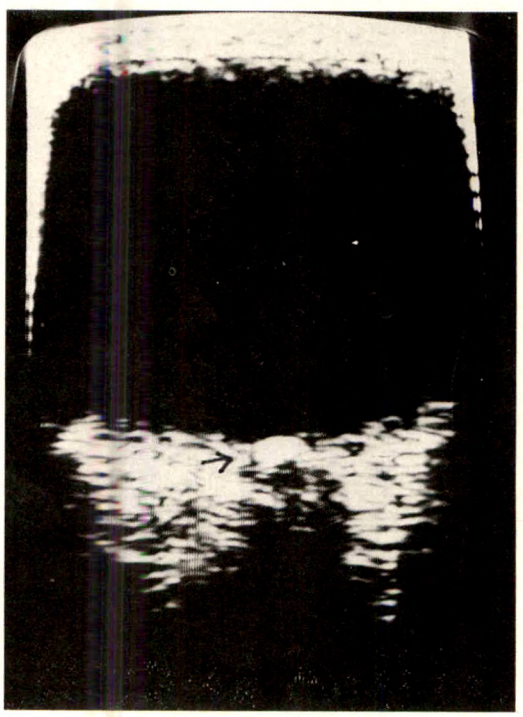


Fig. 3 (Zinn). B-scan ultrasonograph three days after initial pars plana vitrectomy showing an acoustically dense (highly reflective) foreign body embedded in the anterior portion of the optic nerve head (arrow). The retrobulbar fat pattern and optic nerve shadow were acoustically within normal limits.

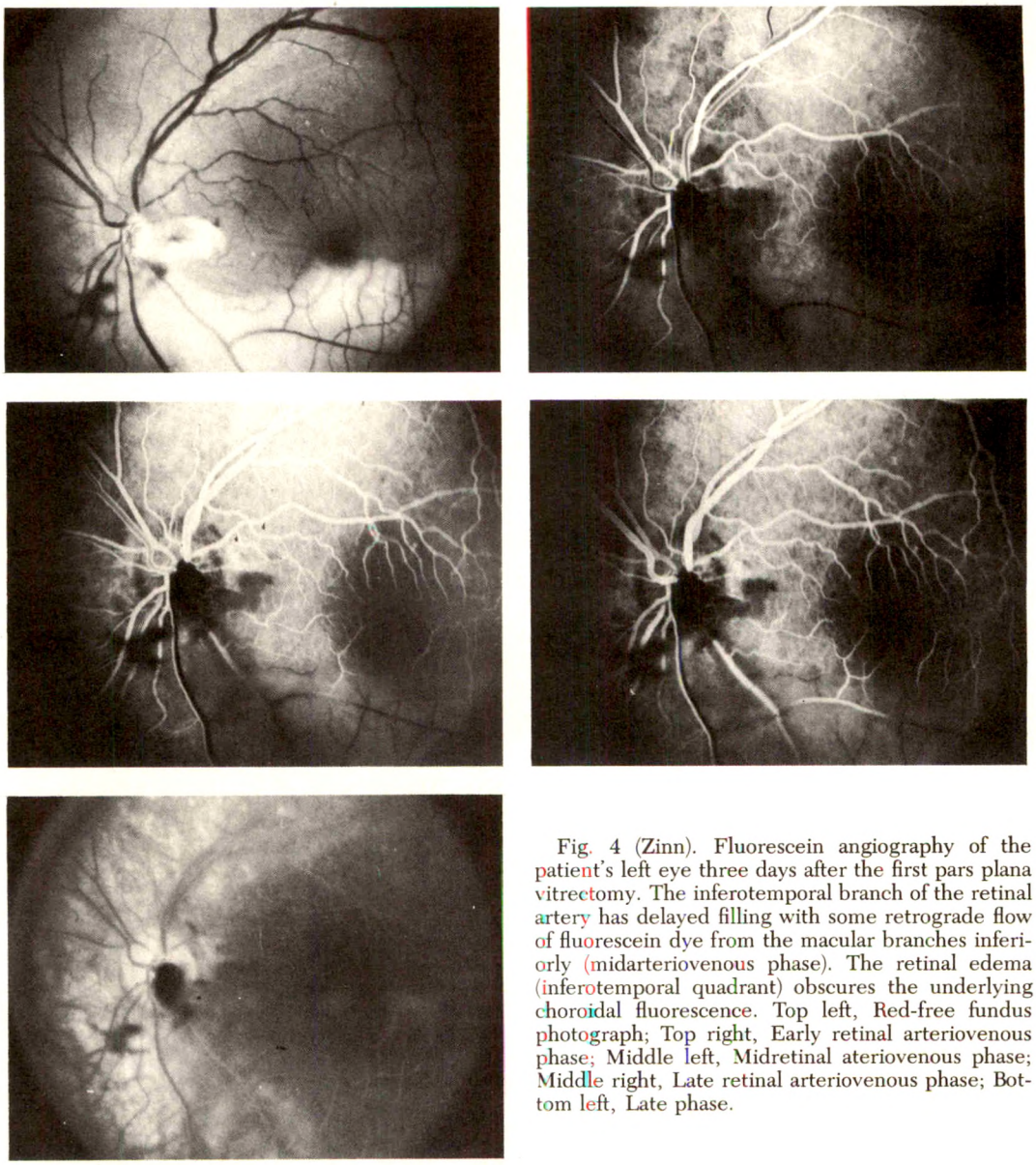


Fig. 4 (Zinn). Fluorescein angiography of the patient's left eye three days after the first pars plana vitrectomy. The inferotemporal branch of the retinal artery has delayed filling with some retrograde flow of fluorescein dye from the macular branches inferiorly (midarteriovenous phase). The retinal edema (inferotemporal quadrant) obscures the underlying choroidal fluorescence. Top left, Red-free fundus photograph; Top right, Early retinal arteriovenous phase; Middle left, Midretinal ateriovenous phase; Middle right, Late retinal arteriovenous phase; Bottom left, Late phase.

moved. In Runyan's case, the patient had light perception in only one quadrant and therefore no attempt was made to remove the foreign body from the optic nerve head. In 1970 Wolter[6] reported on the extraction of a firmly embedded piece of steel from the margin of the optic nerve head using a pars plana approach. However, in this case numerous attempts to use an electromagnet at the pars plana failed to dislodge the foreign body, which was the same in our case. Wolter[6] had to devise a special intravitreal forceps from a spinal tap needle to grasp the foreign body and remove it from the eye through a pars plana incision.

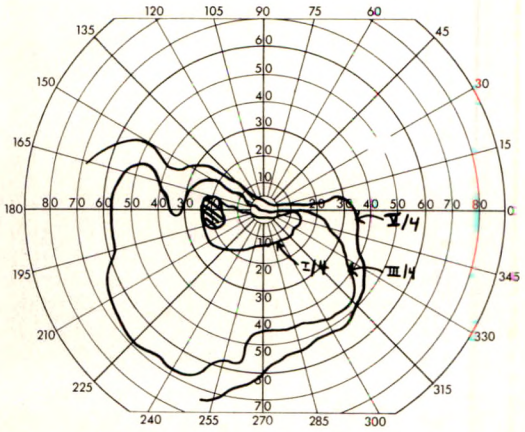


Fig. 5 (Zinn). Visual field two months after removal of the foreign body from the optic nerve head. An altitudinal field cut is noted.

The decision to make a second attempt to remove the foreign body in this case was based on the strong possibility of ocular siderosis.[2-5] The metallurgical analysis of the possible sources of the foreign body (hammer or auto fender) indicated that each had an iron content of 98%. The analysis also disclosed that each metal was highly magnetic. In view of the failure to dislodge the foreign body with strong magnets (pars plana and intravitreal probes), another more direct approach was designed.

In this case a dense membrane partially covered the optic nerve head foreign body and so a microscalpel was fashioned from a 20-gauge needle. This in turn was used to incise the capsule and reflect it to the temporal border of the optic nerve head. This maneuver provided access to the finely pitted surface of the foreign body whose surface was flush with that of the optic nerve head. Gentle traction applied through the tip of the microscalpel to the pitted surface was used to rock the foreign body out of its crater without damaging the surrounding optic nerve and retinal tissue.

The membrane dissection and rocking maneuver of the foreign body in our case could never have been carried out without excellent endoillumination and stereomicroscopic visualization. Also, the bimanual approach[12] was indispensable to the overall surgical management of this case. Additionally, A- and B-scan ultrasonography was important in determining the depth of foreign body penetration into the optic nerve, thereby aiding and influencing the surgical approach.

At the time of the initial examination of the patient no evidence of a branch retinal artery occlusion was present (Fig. 1). However, it was thought that secondary swelling of injured optic nerve tissue in the presence of the deeply embedded foreign body was responsible for the development of the branch retinal artery occlusion and capillary loss in the optic nerve head itself (Fig. 2). The developing branch retinal artery occlusion was obscured by an evolving massive vitreous hemorrhage and was not recognized until the vitrectomy was performed. Interestingly, fluorescein angiography three days after the vitrectomy disclosed not only a branch retinal artery occlusion but actual retrograde flow of fluorescein in the blocked inferotemporal retinal artery (Fig. 4) which has persisted for more than a year after the original injury (Fig. 7).

It is clear from previous reports[1,6-8] and our case that an electromagnet may not be successful in removing small deeply embedded magnetic foreign bodies from optic nerve, retinal, and choroidal tissues. Therefore, preoperatively the ophthalmic surgeon should try to obtain as much diagnostic information on the size, location, and nature of the metallic foreign body as possible. This includes X-ray localization and A- and B-scan ultrasonography.[13] Also, if possible, metallurgic analysis of the hammer and the object being hammered can be invaluable, and testing for the presence of magnetism in these materials is important.

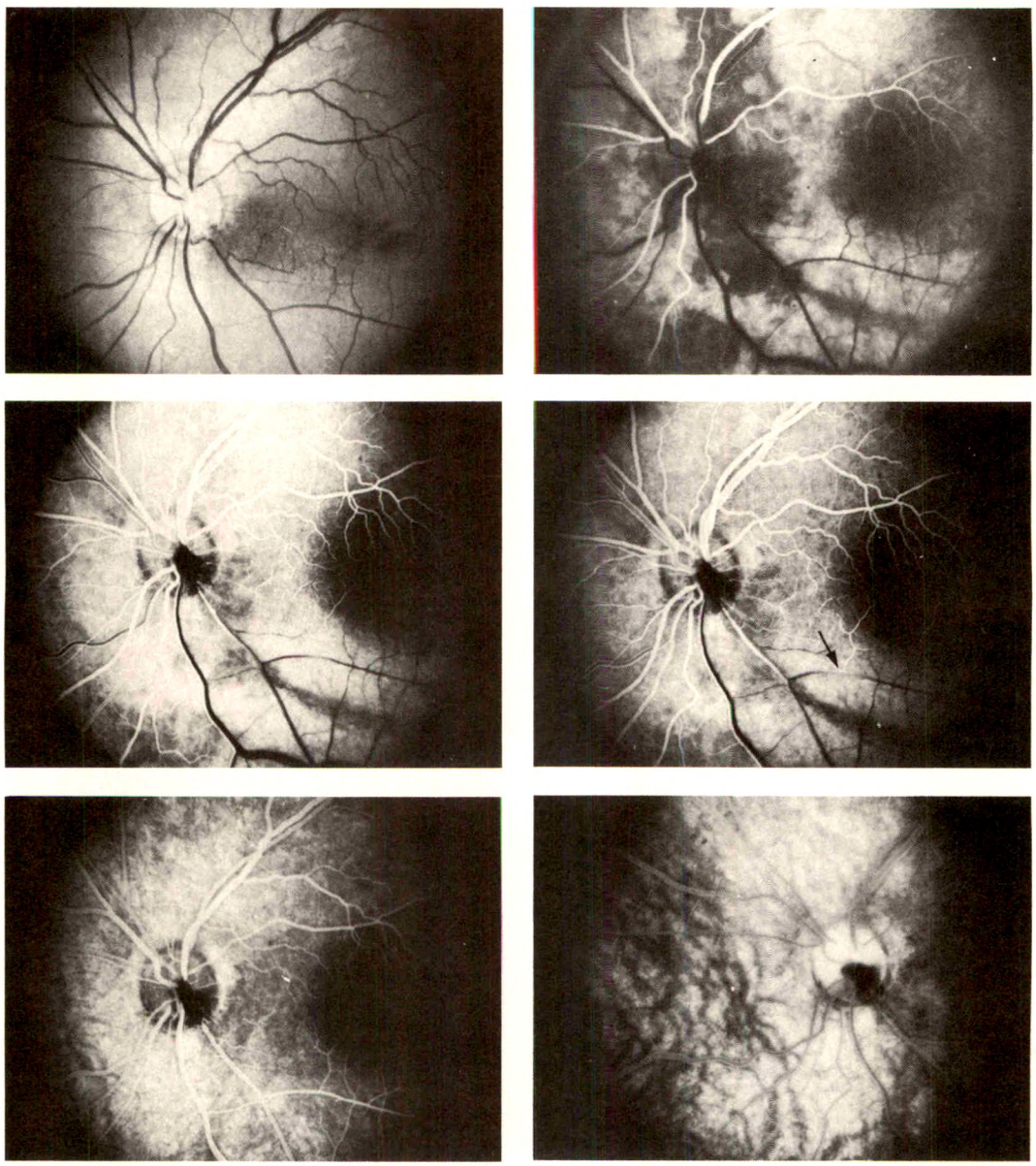


Fig. 7 (Zinn). Left eye, Fluorescein angiogram one year after removal of foreign body from the optic nerve head. Red-free photograph of disk and macular area gives an atrophic appearance to the inferotemporal quadrant of the optic nerve with a residual shallow crater. There is fine wrinkling of the internal surface of the retinal between the temporal edge of the optic nerve head and the nasal macula.

Prompt retinal anteriolar filling of fluorescein dye was evident in all quadrants except in the inferotemporal quadrant where there is only a minor delay in arterial filling. Some retrograde retinal arterial filling was found as described previously. However, there is a prolonged retinal arteriovenous circulation time in the inferotemporal quadrant. The optic cup and the inferotemporal area of the disk have no capillary perfusion present throughout the angiogram sequence. No abnormal leakage of fluorescein dye was noted throughout the angiogram. Top left, Red-free fundus photograph. Top right, Early retinal arterial phase. Middle left, Early retinal arteriovenous phase. Middle right, Midretinal arteriovenous phase with retrograde flow in retinal arteriole (arrow). Bottom left, Late retinal arteriovenous phase. Bottom right, Late phase.

Summary

A 29-year-old man sustained a metallic (98% iron) foreign body in the optic nerve head, which resulted in a massive vitreous hemorrhage and a secondary branch retinal artery occlusion. An initial pars plana vitrectomy was successful in removing the vitreous hemorrhage, but numerous attempts with pars plana and intravitreal electromagnets failed to dislodge the foreign body. A second operation using a microscalpel and foreign body forceps with the bimanual pars plana technique was successful in removing the steel foreign body from the optic nerve-head.

Acknowledgment

Yale Fisher, M.D., provided the photograph for Figure 3.

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ACUTE LEUKEMIC INFILTRATION OF THE VITREOUS DIAGNOSED BY PARS PLANA ASPIRATION

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We have recently managed a patient with acute lymphoblastic leukemia; the leukemic nature of his dense vitreous cellular infiltration was diagnosed by vitreous aspiration. In this patient the infiltration of the vitreous by acute lymphoblastic leukemia, to our knowledge a previously unreported occurrence, was the first clinical sign of involvement of the central nervous system.

CASE REPORT

We examined a 30-year-old man on Oct. 22, 1979. His chief complaint was progressive, painless loss of vision in his left eye over a seven- to ten-day period.

In November 1978, he had been examined for weakness and excessive ease of bruising. The diagnosis was acute lymphoblastic leukemia, null cell, L_2 type, by the French-American-British Cooperative Group classification.[1] He began receiving chemotherapy with doxorubicin, vincristine, cytarabine, and prednisone for induction. He suffered two relapses, which eventually responded to several courses of combinations of vincristine, cytarabine, doxorubicin, asparagenase, methotrexate, and prednisone. On Oct. 16, 1979, he was in apparent clinical remission. He and his siblings were HLA-crosstyped and he was listed for bone marrow transplantation.

On Oct. 22, 1979, when examined for his ophthalmologic complaint, his best corrected visual acuity was 6/4.5 (20/15) in the right eye ($-2.00 +1.25 \times 170$) and 6/90 (20/300) in the left eye ($-1.25 +0.25 \times 160$). Results of the external examination, motility test, and examination of the pupils, including the swinging light test, were normal. The slit-lamp examination showed a normal right anterior segment but heavy cellular infiltration of the left vitreous with only 1+ anterior chamber cells. There were no lens changes, rubeosis iridis, hypopyon, or hyphema. Gonioscopically, there were no abnormalities of the anterior chamber angle in either eye. The applanation pressure was 16 mm Hg in the right eye and 12 mm Hg in the left eye. Examination of the vitreous and retina in the right eye disclosed a normal vitreous body, retina, and optic disk without evidence of leukemic infiltration or other disorders. The left vitreous had dense cellular infiltration, concentrated mainly just anterior to the detached posterior hyaloid membrane. There was some clumping of cells and vitreous fibrils into opaque vitreous sheets (Fig. 1). We could not visualize the retina posterior to the equator. A-scan and B-scan ultrasonographic examinations demonstrated an attached retina without evidence of choroidal thickening. Results of cranial computed tomographic studies, including sections showing the optic nerves, were normal. The IV_4e white and I_4e white isopters on the Goldmann perimeter were normal when we tested the right eye, as was the IV_4e white when we tested the left eye.

We decided to obtain a vitreous specimen in order to define the nature of the cellular infiltrate and to be certain that there was no infectious endophthalmitis. The patient received general anesthesia. After making a conjunctival incision in the 2-o'clock meridian and a full-thickness sclerotomy, we placed a 20-gauge needle 4 mm posterior to the corneoscleral limbus and entered it into the center of the vitreous after light diathermization of the ciliary body. We aspirated approximately 0.5 ml of fluid without difficulty,

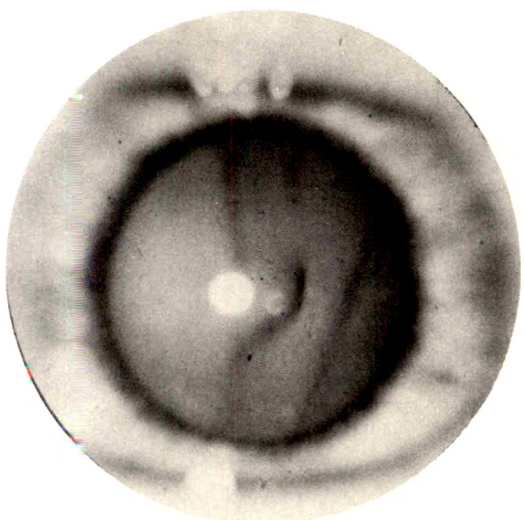


Fig. 1 (Swartz and Schumann). Clumping of cells and vitreous fibrils into sheets.

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using microscopic control. We immediately sent the aspirated fluid, which was clear, to the cytopathology laboratory and to be cultured for bacteria and fungi. We used a preplaced mattress 6-0 Vicryl suture to close the sclerotomy and closed the conjunctiva with 6-0 plain catgut. Indirect ophthalmoscopy showed the entrance site to be in the pars plana just posterior to the pars plicata.

A Papanicolaou stain disclosed numerous blast cells (approximately 8 to 12 μ in diameter) characterized by nuclear enlargement, prominent nuclear membrane indentations, coarse chromatin, multiple irregular nucleoli, and a scant rim of cytoplasm (Fig. 2). Wright's stain showed similar blast cells, immature lymphoid cells, and numerous smudge cells characteristic of acute lymphoblastic leukemia. The cytomorphologic analysis of the vitreous aspirate led us to reexamine the cerebrospinal fluid. An atraumatic lumbar puncture disclosed the presence of several blast cells (Fig. 3) similar to the cells from the vitreous. Thus, the vitreous infiltrate was the only sign of leukemic involvement of the central nervous system. The peripheral blood cells and bone marrow aspirate, examined while the patient was under general anesthesia, showed no evidence of leukemic relapse.

We instituted therapy promptly with five days of orally administered prednisone (100 mg/day), five days of subcutaneously administered cytarabine (100 mg/day), and six 100-mg intrathecal doses of cytarabine one week apart. Cranial radiotherapy was delivered with a proton beam linear accelerator through parallel apposed shaped lateral fields with a calculated midline dose of 200 rads at each of six sessions over seven days, for a total of 1,200 rads.

The patient's left vitreous began to clear rapidly

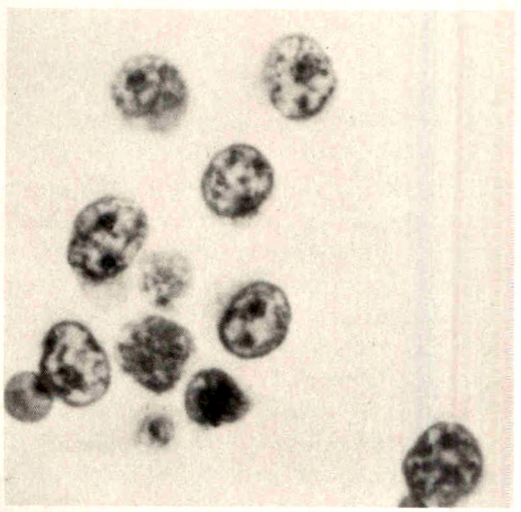


Fig. 2 (Swartz and Schumann). Lymphoblasts in the vitreous aspirate (Papanicolaou stain, oil immersion, × 1,000).

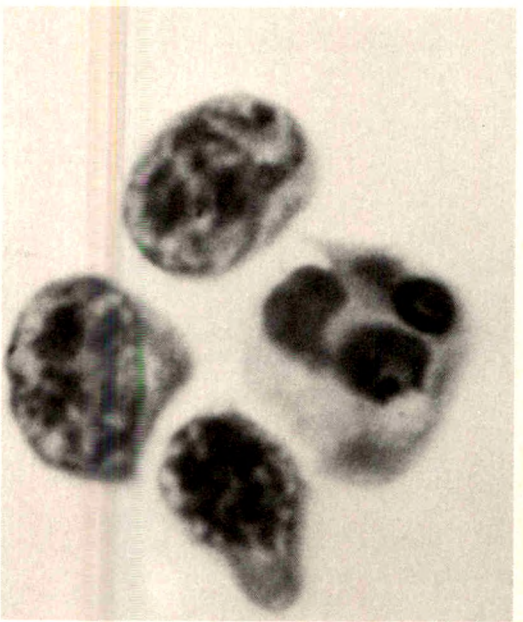


Fig. 3 (Swartz and Schumann). Lymphoblasts, similar to those from the vitreous specimen, are present in the cerebrospinal fluid (Papanicolaou stain, oil immersion, × 1,000).

during this treatment and, when the entire retina could be examined, there were no signs suggestive of choroidal or retinal infiltration. Biomicroscopic examination of the optic disk showed attenuation of the overlying vessels, especially the venules as they approached the nerve (Fig. 4). The vascular narrowing is especially noteworthy when compared to the results two weeks later (Fig. 5). The vascular compression was present at a time when the intraocular pressure was 14 mm Hg. Also present was subtle but definite sheathing of the retinal vessels overlying the optic disk (Fig. 5).

On Nov. 9, 1979, 14 days after the vitreous tap, visual acuity returned to 6/4.5 (20/15). There was no afferent pupillary defect, a normal lens, and remarkable clearing of the cellular content of the vitreous. The cells still present remained concentrated centrally just anterior to the posterior hyaloid membrane. Cerebrospinal fluid was devoid of blast cells. We considered the patient ready for the bone marrow transplantation.

Discussion

Cytopathologic confirmation of the acute lymphoblastic leukemic nature of the clinically observed vitreous cells provided the initial sign of central nervous system leukemic involvement, and elimi-

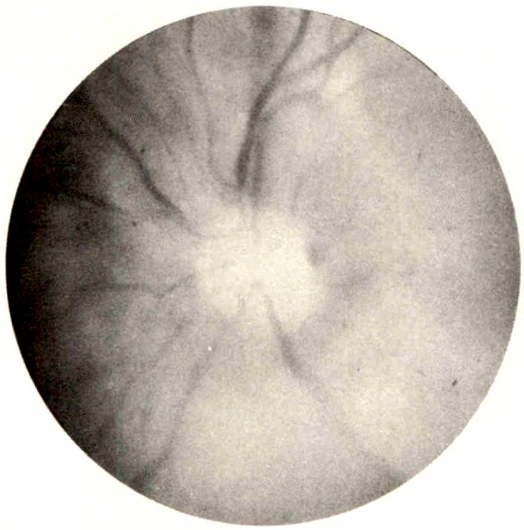


Fig. 4 (Swartz and Schumann). Six days after the initiation of radiotherapy, the vitreous has cleared enough to show the attenuation of the retinal vessels overlying the disk.

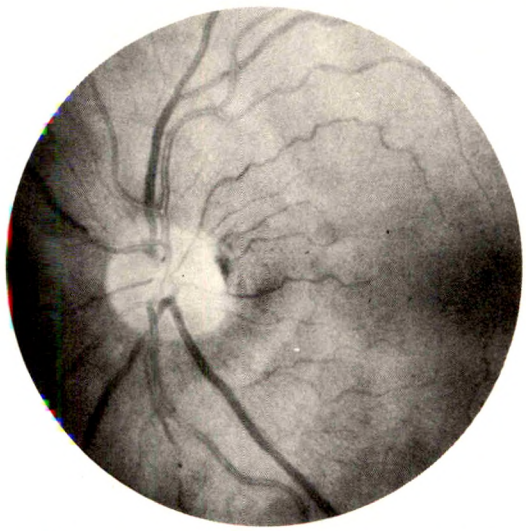


Fig. 5 (Swartz and Schumann). Thirteen days later, the vessels have regained their normal caliber although subtle sheathing remains. The sheathing can be seen best along the inferotemporal vein as it traverses the disk.

nated bacterial endophthalmitis as a possible diagnosis. Prompt institution of radiotherapy and chemotherapy produced rapid reduction of cells in the vitreous and clearing of cells from the cerebrospinal fluid.

Vitreous specimens obtained by trans pars plana aspiration or vitrectomy have been used to differentiate chronic uveitis from reticulum cell sarcoma (histiocytic lymphoma).[2-5] Postmortem examinations have shown that the hematologic neoplastic disorders known to infiltrate the vitreous include Burkitt's lymphoma,[6] multiple myeloma,[7] Hodgkin's disease,[8] and reticulum cell sarcoma.[2,9-19] These disorders, like acute lymphoblastic leukemia, tend to be radiosensitive.

Ocular involvement in acute leukemia has been reported in 15 of 17 patients in an autopsy series[7] and in 52 of 657 patients in a clinical series.[20] The ocular signs of acute leukemias include: (1) infiltration of the optic nerve, choroid, retina, iris, ciliary body, and anterior chamber[7,21-39]; (2) central serous retinopathy with numerous hyperfluorescent dots deep to the retina on fluorescein angiography[39-42]; (3) retinal pigment epithelium clumping [43]; (4) retinal vascular sheathing[7,44]; and (5) hemorrhage into the subconjunctival space, anterior chamber, retina, and vitreous.[7]

Acute leukemic ocular infiltration has a significant correlation with central nervous system involvement and survival. Of 29 children with ocular infiltration, 27 had central nervous system involvement before or during ocular involvement.[24] The high correlation of intraocular leukemic infiltrates and central nervous system involvement may reflect the proximity of the subarachnoid space to the posterior choroid and retina.[24]

In our case, the source of the leukemic cells was not clinically obvious. Possibly the lymphoblasts gained access to the vitreous via the optic nerve or its vasculature. Although we did not observe definite optic nerve infiltration, the apparent

compression of the optic disk vasculature and vascular sheathing are suggestive of such a process. On the other hand, Ridgeway, Jaffe, and Walton[24] have speculated that the eye, like the central nervous system, is a "pharmacologic sanctuary" relatively unaffected by systemic chemotherapy. If so, lymphoblasts might survive in intraocular sites and the central nervous system and ocular involvement could have been coincidental.

Summary

A 30-year-old man with acute lymphoblastic leukemia, null cell, L_2 type, developed a unilateral dense cellular vitreous infiltrate while in apparent remission. Cytopathologic examination of material aspirated from the vitreous established the diagnosis of lymphoblastic infiltration. Prompt radiotherapy and chemotherapy rapidly reduced the vitreous infiltration and cleared cells from the cerebrospiral fluid.

Intraocular involvement by the leukemic process was the initial sign of central nervous system involvement. Acute lymphoblastic leukemia should be added to the list of lymphoproliferative disorders capable of vitreous infiltration, and which can be identified by vitreous aspiration and cytopathologic examination.

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THE EFFECT OF EXTENDED-WEAR APHAKIC HYDROPHILIC CONTACT LENSES AFTER PENETRATING KERATOPLASTY

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Studies over the last several years have shown that with hydrophilic contact lenses of sufficiently high water content and extreme thinness, or both, there is enough trans-lens oxygen diffusion to support minimal corneal respiratory needs.[1-3] Because of these physical properties it is possible to fit contact lenses in aphakic eyes for extended wear, that is, contact lenses that can be worn for weeks to months without removal and beneath which the cornea remains clear and free of blood vessels. Indeed, extensive clinical trials with these contact lenses have shown little neovascularization of the cornea and a slight change in measurements of corneal thickness.[4] Because of the lack of clinical problems, the contact lenses have been judged successful and acceptable in a high percentage of cases.

Patients who are aphakic and who have undergone penetrating keratoplasty have visual needs similar to those of other patients with aphakia. The improved field of vision obtained with a contact lens and the lack of handling essential for many elderly people have made it an attractive means of correcting aphakia. As part of a clinical investigation of extended-wear contact lenses in patients, with aphakia I fitted a series of patients who had also undergone penetrating keratoplasty after a cataract extraction or as part of a combined procedure with extended-wear contact lenses. To my knowledge, there have been no reports on the effect of extended-wear contact lenses after penetrating keratoplasty.

SUBJECTS AND METHODS

During the course of a larger clinical trial involving aphakic patients with extended-wear contact lenses, I fitted nine patients who had undergone corneal transplantation five to 13 months previously. Three of these patients were fitted with 55% water content Hydrocurve II-55 contact lenses and the other six with 80% water content Sauflon PW contact lenses. Fitting criteria, that is, movement and stability of vision, were the same as for other patients with aphakia. After the initial fitting, I examined the patients 24 to 48 hours, one week, and two weeks later. Thereafter examinations were monthly. Two patients were also using 0.1% dexamethasone eyedrops, once daily.

All patients had undergone penetrating keratoplasty with an 8-mm donor button in a 7.5-mm host wound. I had used four interrupted 10-0 nylon sutures plus a running 10-0 nylon suture in eight of nine patients; one patient had 16 interrupted sutures. All knots were buried.

I fitted the extended-wear contact lenses in four patients while the sutures were still in place; in the remaining five patients I removed the sutures before fitting the contact lenses.

RESULTS

Eight of the nine patients fitted with extended-wear soft contact lenses showed neovascularization of the graft two to 24 weeks after fitting (Table). All of these

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TABLE

SUMMARY OF DATA

Case No.	Host Cornea	Interval Between Keratoplasty and Use of Lens (mos)	Interval Between Lens Use and Vascularization (wks)	Extent of Vascularization (mm)	Comments
1	Edema	10	6	3	Contact lens use continued with corticosteroids
2	Edema	17	4	4	At 1 yr, visual acuity was 6/15 (20/40); at present, visual acuity is 6/22 (20/70)
3	Edema	7	9	1	———
4	Edema	6	4	2	Graft rejection successfully treated with corticosteroids
5	Edema and vascularization	36	4	2	———
6	Edema and vascularization	4	9	0	At 2 mos, presumed *Candida* infection
7	Edema	9	2	3	Progessive vacularization with daily wear halted with corticosteroids
8	Edema	5	2	2	———
9	Edema	11	30	2	———

patients had edematous host corneas; two had had vascularization of the host cornea before contact lens fitting. The pattern of vascular ingrowth in the graft showed no predilection for any area but involved any point on the circumferences of the graft.

In the grafts, in which sutures remained, vascular ingrowth proceeded along suture paths.

Postoperative astigmatism, measured by keratometry, ranged from 1.5 diopters to 7.0 diopters. The patients obtained best corrected visual acuity with hydrophilic contact lens and spectacles overcorrection.

In two patients for whom there was no other practical way to correct vision, I left the contact lens in place and successfully suppressed further vascularization with topically applied 0.1% dexamethasone. In one patient vascularization continued to progress with daily wear over a six-month follow-up period.

Patient 4 had an acute graft rejection six weeks after contact lens fitting; within two weeks I noted vessels in the graft. I removed the contact lens, treated the eye intensively with topically applied 0.1% dexamethasone, and suppressed the graft rejection.

CASE REPORTS

Case 1—A 69-year-old woman, whose corrected visual acuity was counting fingers at five feet because of Fuchs' dystrophy and cataract, underwent a combined 8-mm penetrating keratoplasty and intracapsular cataract extraction. Her postoperative course was uncomplicated. I removed the sutures after ten months. Her corected visual acuity was 6/12 (20/40). I fitted a Hydrocurve II (55% water content) contact lens within two weeks of suture removal. I noted vascular ingrowth extending 3 mm into the graft two months later (Fig. 1). Neither daily-wear contact lenses nor spectacles were feasible for this patient, and so she continued to use an extended-wear contact lens. I prescribed 0.1% dexamethasone eyedrops to be used twice daily. I noted no increase in vascularization during a 15-month observation period. The graft has remained clear and her visual acuity is 6/12 (20/40).

Case 4—A 67-year-old woman, whose corrected visual acuity was 6/60 (20/200) because of Fuchs' dystrophy and cataract, underwent a combined 8-mm penetrating keratoplasty and planned extracapsular cataract extraction. Her postoperative course was uncomplicated for six months. Spectacle-corrected visual acuity was 6/15-2 (20/50-2). Because the patient had unilateral aphakia with decreasing visual acuity in the other eye, I fitted a Sauflon (80% water content) extended-wear contact lens. A continuous 10-0 nylon suture with a buried knot was in place and all medications had been discontinued. The contact lens was comfortable and provided good visual acuity. Four weeks later, I noted neovascularization extending 2 mm into the graft (Fig. 2). The eye, however, showed no inflammatory signs.

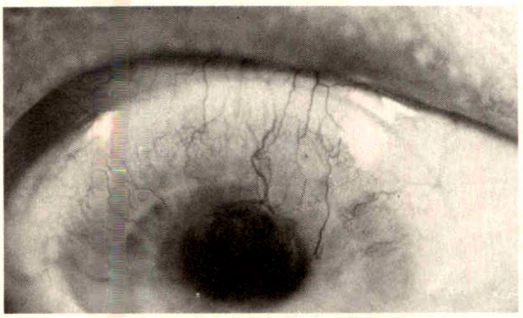


Fig. 1 (Lemp). Case 1. Vascularization of the graft under a moderately hydrophilic contact lens (55% water content).

Two weeks later the patient complained of redness and decreased visual acuity. Signs of a graft rejection were present (Fig. 3). Intensive treatment with topically applied corticosteroid and removal of the lens resulted in successful reversal of the reaction. During a four month follow-up period, this patient has maintained a visual acuity of 6/12-1 (20/40-1) with daily wear of the contact lens and the use of 0.1% dexamethasone eyedrops, once daily.

Case 5—A 69-year-old man with bilateral Fuchs'

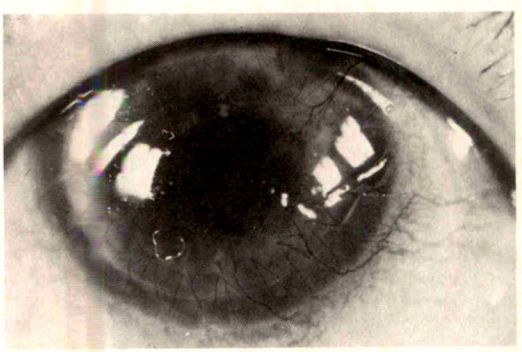


Fig. 2 (Lemp). Case 2. Vascularization of the graft under a highly hydrophilic contact lens (80% water content).

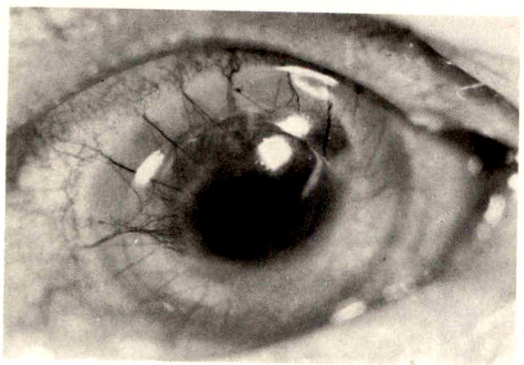


Fig. 3 (Lemp). Case 4. Acute graft rejection after six weeks of contact lens wear.

dystrophy and cataract had undergone a successful penetrating keratoplasty of the left eye. Postoperatively, however, the cataract worsened, making an intracapsular extraction necessary. There was a subsequent graft rejection, which was suppressed with topically applied corticosteroids. Eventually, the graft became edematous and there was a separation of Descemet's membrane and endothelium from the graft.

A combined 8-mm penetrating keratoplasty and cataract extraction of the right eye was complicated by a partial choroidal hemorrhage. This caused extensive peripheral synechiae and secondary glaucoma. A pars plana vitrectomy and trabeculectomy were successful in controlling the intraocular pressure and the graft remained clear. Corrected visual acuity was 6/15 (20/50).

Three months after keratoplaty, I fitted a Sauflon (80% water content) contact lens in the right eye. The patient remained comfortable and had stable visual acuity for two months. At that time I noted a decrease in visual acuity and a 3-mm superficial infiltrate in the graft. I removed the contact lens. Scrapings and culture for bacteria and fungus were negative. The patient, however, had a *Candida* skin infection. I treated him with nystatin eyedrops and the graft ulcer healed. Subsequent daily wear of the contact lens has been successful. Visual acuity has been good and no further infections have occurred.

Discussion

The effects of extended-wear hydrophilic contact lenses on corneal respiration have been judged clinically acceptable in aphakic patients.[4] We know that most of the oxygen necessary for corneal metabolism is supplied by way of the tear film.[5] Recent evidence suggests that there is minimal tear exchange beneath a hydrophilic contact lens and that most of the oxygen supplied to the cornea depends upon diffusion through the contact lens material.[6] It has also been shown that oxygen sufficient for the minimal needs of the corneal epithelium, is available beneath the highly hydrophilic soft contact lens.

Factors thought to induce neovascularization of the cornea include lack of physical compactness of the cornea (edema), hypoxia, and a vascular-stimulating factor.[7] A heat-labile, diffusible, noninflammatory substance probably liberated by damaged cells has been implicated in neovascularization of the cornea. Cellular hypoxia probably stimulates the release of such a factor.[8]

This series is too small to determine whether there is any significant difference in vascularization of grafts beneath a moderately and a highly hydrophilic contact lens. We know that the metabolism in the grafted cornea differs from that of a normal cornea[9]; possibly the oxygen requirements of a grafted cornea are greater than those of a cornea without a graft. Moreover, an extended-wear contact lens placed over a cornea probably decreases the rate of evaporation from the cornea while increasing corneal thickness and surface temperature, thus inducing a higher metabolic rate. An accumulation of metabolic products caused by limited tear film exchange beneath the contact lens can occur. Additionally, blood vessels in a peripheral host cornea or corneoscleral limbus, or both, may be more reactive after grafting.

All of these patients had swollen host corneas and two had vascularization of the host corneas before contact lens fitting, something that could contribute to neovascularization. Since vascular ingrowth proceeded along suture lines in those eye with sutures in place, a lack of tissue compactness may play a role in vascularization.

The low incidence of allograft rejection

in corneal transplantation has been attributed to a special immunologic privilege of the avascular cornea. Ingrowth of vessels in the graft compromises this privilege and is associated with a higher incidence of rejection (Case 4). The effect of topically applied corticosteroids in retarding the progression of further graft vascularization in the absence of clinical signs of inflammation is curious and requires further explanation (Cases 1 and 7). Additionally, one patient (Case 6) developed a presumed *Candida* corneal ulcer beneath an extended-wear lens. This responded to treatment with nystatin after contact lens removal. Contact lens wear was resumed on a daily-wear basis without further complications.

The appearance of significant neovascularization of the corneal graft in eight of nine patients fitted with extended-wear soft contact lenses in our series strongly suggests that these patients are at special risk in the fitting of extended-wear contact lenses. I suggest that alternative methods of correcting visual acuity be considered in patients with aphakia who have received corneal grafts.

Summary

I fitted nine aphakic patients who had undergone penetrating keratoplasty with extended-wear hydrophilic contact lenses (55% to 80% water content). All patients had edematous host corneas. Eight of nine developed vascularization of the graft after two to 30 weeks of contact lens wear. One patient developed a presumed *Candida* keratitis. The development of vascularization of the graft suggests that patients with grafted corneas are at special risk with extended-wear hydrophilic contact lenses.

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PREVALENCE OF CONJUNCTIVAL CHANGES IN WEARERS OF HARD CONTACT LENSES

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We previously described a syndrome in wearers of soft contact lenses and wearers of polymethylmethacrylate hard contact lenses that is characterized by excess mucus, ocular itching, diminished or destroyed contact lens tolerance, and giant papillae in the upper tarsal conjunctiva.[1] The syndrome developed after months to years of otherwise successful lens wear. Srinivasan and associates[2] reported the presence of giant papillae associated with the wearing of other types of methylmethacrylate ocular prostheses. We undertook a prospective study to determine the prevalence of upper tarsal papillary changes in a select group of successful wearers of hard contact lenses. Our interest stems from the possibility that giant papillary conjunctivitis is the extreme example of conjunctival changes related to contact-lens wear. We know that giant papillary conjunctivitis is caused by contact-lens wear. Our hypothesis is that there is a spectrum of conjunctival changes associated with contact-lens wear, and that giant papillary conjunctivitis is at the far end of the spectrum. We wanted to identify more of the spectrum.

Our purpose was to determine the incidence of elevated papillae in the upper tarsal conjunctiva of patients with polymethylmethacrylate contact lenses and of a control group of subjects who did not wear contact lenses. The presence of papillae 0.3 mm in diameter was arbitrarily selected as the dividing line between normal and abnormal.

SUBJECTS AND METHODS

Subject selection—To obtain the desired 200 patients with contact lenses, we examined 335 consecutive patients with hard contact lenses between July 11, 1977, and Nov. 10, 1978. The criteria for subject selection included the successful wearing of polymethylmethacrylate con-

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tact lenses at least eight hours per day for at least five years, with no more than one week's interruption, and no reported symptoms or difficulties for a minimum of five years (symptoms may have developed later) after the initial adaptation and the final lens fitting. In an attempt to lessen the likelihood that symptoms or palpebral changes, or both, resulted from less than optimal fit, we further required that there had been no changes in the design of the lenses over the five-year period. We excluded patients with systemic or ocular disease. The 200 subjects selected had come for examination because of vision problems (four cases), routine contact-lens evaluation without lens-related problems or ocular symptoms (160 cases), scratched or damaged contact lenses (eight cases), lost lenses requiring replacement (three cases), mucus, itching, or both (ten cases), and other contact lens or ocular symptoms (15 cases). We included eleven subjects with keratoconus and three with aphakia. Of the 135 subjects we excluded, 14 had systemic or ocular disease, 116 did not meet our other criteria, and five had one giant papilla each, varying in diameter from 1.0 to 2.0 mm.

The control group comprised 500 consecutive individuals examined between Sept. 10, 1977, and March 22, 1979 for ocular check-ups and possible contact-lens fittings. None had ever worn contact lenses.

Information collected on all subjects included age, duration of lens wear in years (if applicable), average daily wearing time of lenses in hours (if applicable), symptoms, refractive status, history of atopy, and presence or absence of elevated papillae larger than 0.3 mm in diameter over the tarsal conjunctiva. Although the examiners were aware of which subjects wore contact lenses, they were careful to examine each patient in the same way regardless of category.

Examination—We everted the upper eyelid and examined the tarsal conjunctival surface biomicroscopically under white light and ×16 magnification to detect papillary changes. Two drops of sterile 2% sodium fluorescein were instilled into the lower cul-de-sac of each eye. We instructed the subjects to blink completely six times to distribute the fluorescein over the tarsal conjunctival surface. We reinverted the upper eyelid and repeated the biomicroscopic examination with cobalt blue light. Figure 1 shows examples of photographs made by both methods. We used only the appearance of the conjunctiva over the tarsus to determine the presence of papillary changes. Classification as positive required more than one elevated papilla larger than 0.3 mm in diameter on one or more of the three zones[1] of the tarsal plate (Fig. 1). Measurements were made with the micrometer on the slit apparatus of a Haag-Streit 900 slit lamp. To determine whether papillae were elevated, we directed a narrow slit beam over the papillae and observed it with ×10 magnification. Elevated papillae caused a discontinuity of the slit beam from the surface of the surrounding conjunctiva.

We analyzed the data by χ^2 with Yates' correction.[3] $P \leq .05$ was considered significant.

Results

Of the 200 subjects with contact lenses, 21 (10.5%) had elevated papillae larger than 0.3 mm in diameter. Detection of papillae, particularly of those less than 0.75 mm in diameter, frequently required biomicroscopic examination of the fluorescein-stained tarsal conjunctiva with cobalt blue light. Of the control group of 500 subjects, only three (0.6%) showed elevated papillae larger than 0.3 mm in diameter ($P < .001$). Two of these subjects were subsequently found to have vernal conjunctivitis.

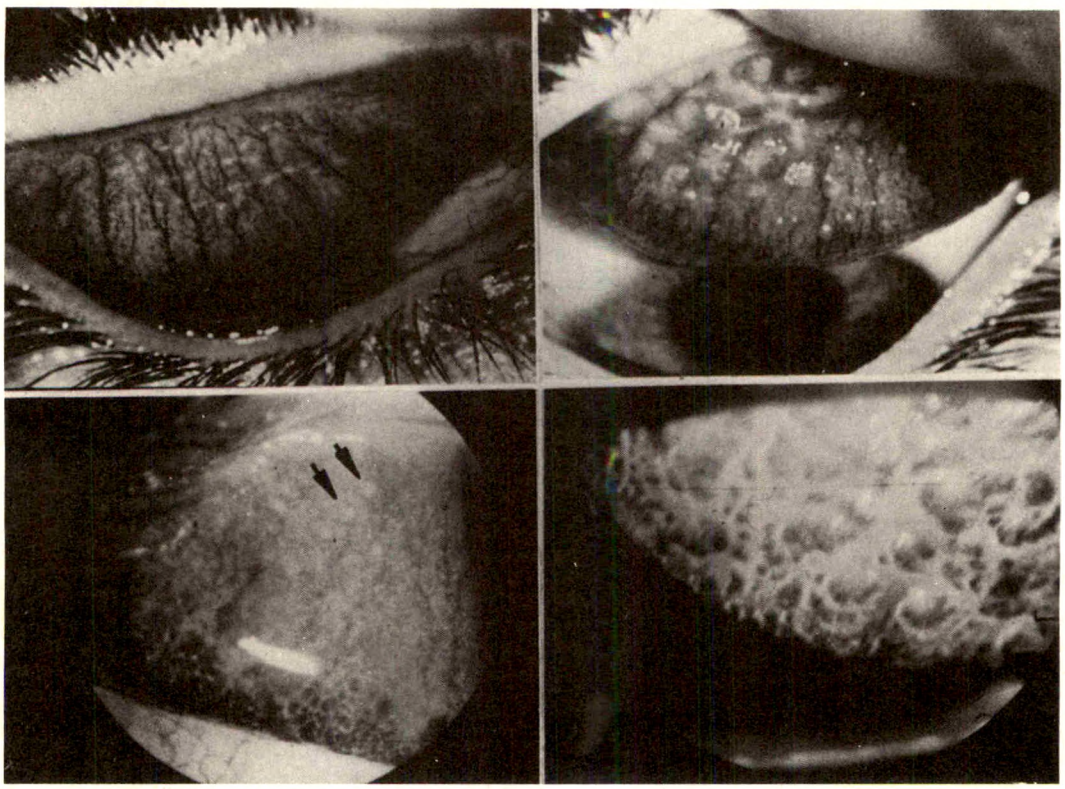


Fig. 1 (Korb and associates). Upper tarsal conjunctivae of subjects wearing hard contact lenses photographed with white light (top left and top right) and cobalt blue light after instillation of fluorescein (bottom left and bottom right). Conjunctivae have papillae 0.3 to 0.5 mm in diameter (top left and bottom left) and papillae 0.5 to more than 1.0 mm in diameter (top right and bottom right).

The papillary changes ranged from mild, without symptoms, to very severe, with almost total loss of lens tolerance. Of the 21 subjects with contact lenses who had papillary changes, 16 had bilateral and five had unilateral changes. The incidence of papillary changes did not vary significantly with refractive status (Fig. 2), duration of lens wear (Fig. 3) (which ranged from five to 28 years, with a mean of 13.5 years), average daily wearing time (Fig. 4), history of atopy (Fig. 5) ($P > .05$), or age (Fig. 6) ($P > .05$).

Although only ten of the subjects with contact lenses complained of mucus, itching, or both, upon questioning at the

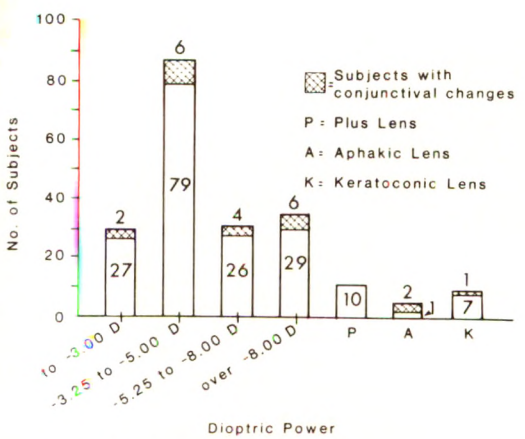


Fig. 2 (Korb and associates). Refractive status of subjects without apparent conjunctival changes and of those with elevated papillae more than 0.3 mm in diameter.

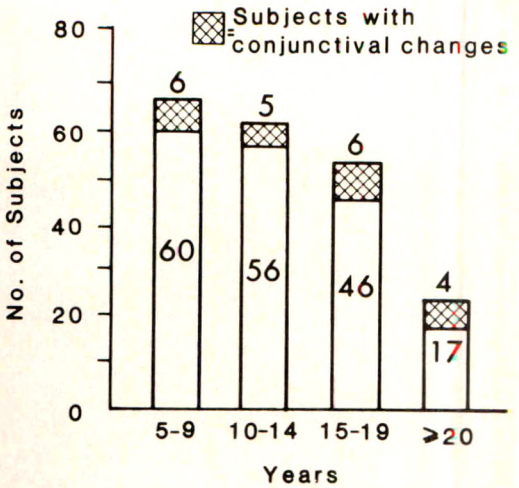


Fig. 3 (Korb and associates). Average duration of lens wear in subjects without apparent conjunctival changes and in those with elevated papillae more than 0.3 mm in diameter.

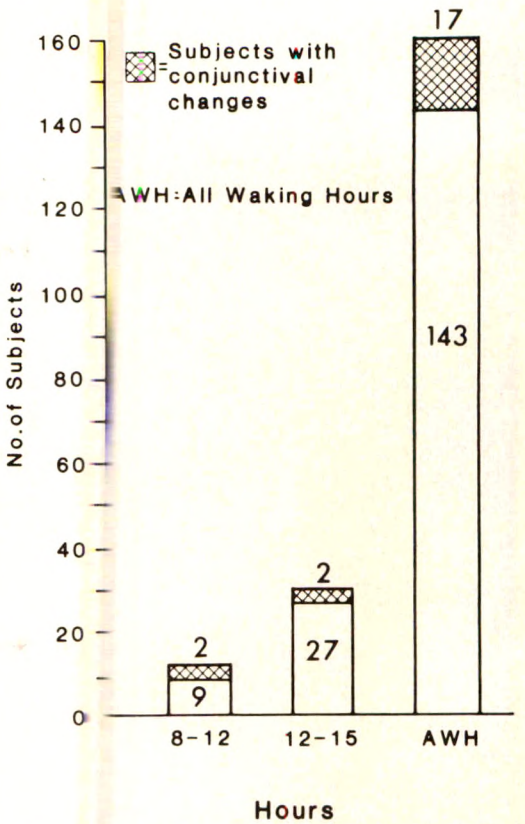


Fig. 4 (Korb and associates). Average daily wearing time in subjects without apparent conjunctival changes and in those with elevated papillae more than 0.3 mm in diameter.

time of examination 21 more admitted to such symptoms. Of these 31 symptomatic subjects, papillary changes were present in 16 (53%); the eyelids of the other 15 appeared to be normal by biomicroscopy. Of the ten subjects with a chief complaint of mucus, itching, or both, four had papillary changes. Mucus or itching symptoms occurred in 16 of 21 (76%) of all subjects with papillary changes, but in only 15 of 179 (8%) of those without papillary changes ($P \leq .001$) (Fig. 7).

Discussion

The prevalence of papillary changes in the conjunctivae of subjects with polymethylmethacrylate contact lenses was 10.5%. Since a criterion for subject selection was uninterrupted successful and asymptomatic contact-lens wear for a minimum of five years before examination, and since only three of the 500 subjects without contact lenses showed papillary changes similar to those associated with wearing polymethylmethacrylate contact lenses, such a frequent finding of papillary changes was not expected. Indeed, the prevalence would have been 13% if the five subjects with contact lenses who each had a single gigantic papilla 1.0 mm or more in diameter had been included.

This study also showed that when significant symptoms were present, papillary changes were also present 53% of the time, and that when papillary changes were present, symptoms were present 76% of the time. Nevertheless, the symptoms of mucus, itching, or both are often ignored by the patient as "part of wearing contact lenses," and, therefore, the patient does not complain or seek examination. Thus, of the 21 subjects with papillary changes, only four

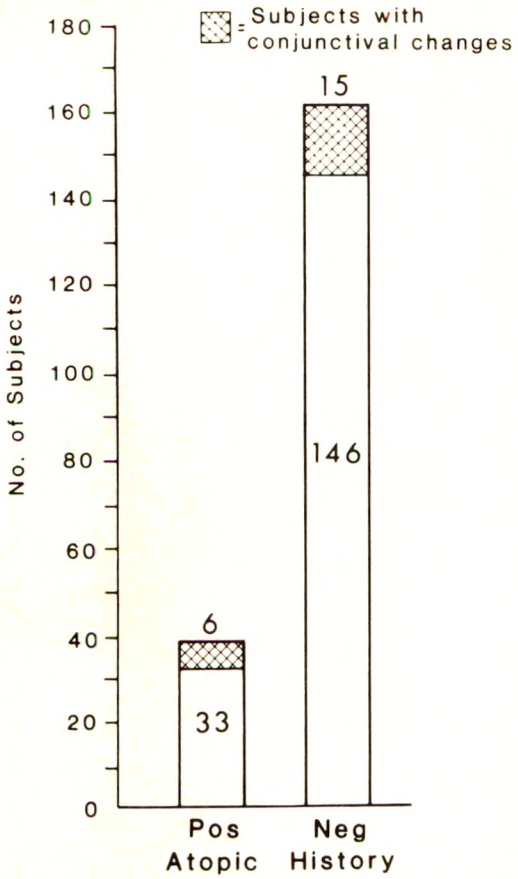


Fig. 5 (Korb and associates). History of atopy in subjects without apparent conjunctival changes and in those with elevated papillae more than 0.3 mm in diameter.

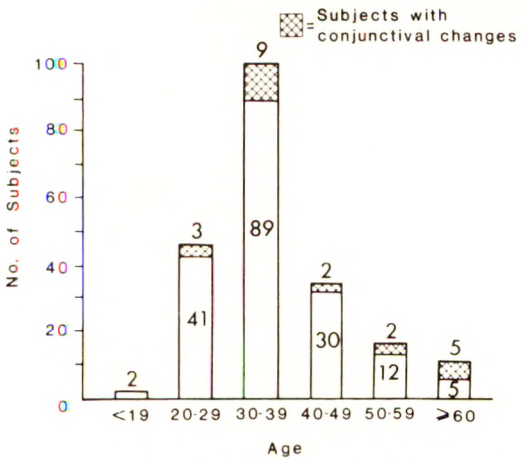


Fig. 6 (Korb and associates). Age distribution of subjects without apparent conjunctival changes and of those with elevated papillae more than 0.3 mm in diameter.

came for examination with the chief complaint of mucus, itching, or both, while 12 subjects admitted to these symptoms only when specifically asked.

Refractive status was included in this study because it was expected that a high-minus contact lens (over −8.00 diopters) might predispose the conjunctiva to papillary changes. The thickness of the high-minus lens presents the most extreme example of excess peripheral mass; thus, the degree of lens-conjunctiva contact is exaggerated. Further, the pressure exerted on the conjunctiva by the high-minus lens would appear to be increased,

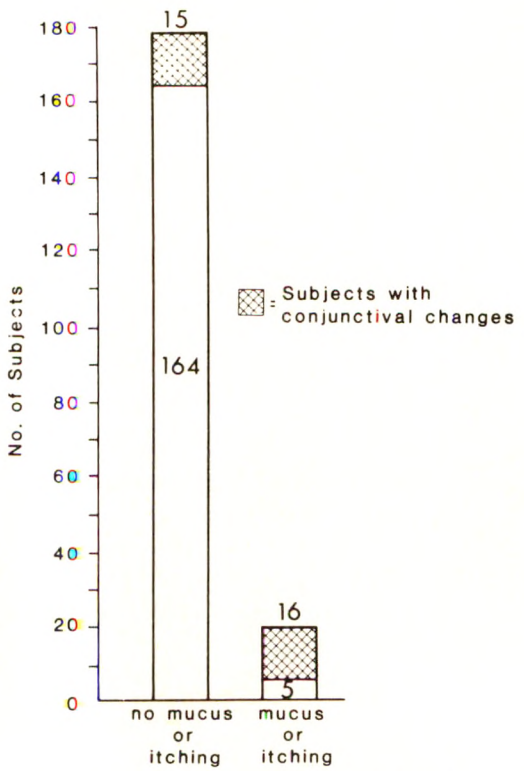


Fig. 7 (Korb and associates). Distribution of symptoms of mucus, itching, or both in subjects without apparent conjunctival changes and in those with elevated papillae more than 0.3 mm in diameter.

particularly during blinking. Consequently, we might expect a greater frequency of problems with such lenses, assuming that the cause of the papillary changes was related to mechanical trauma. In this study, 35 subjects wore polymethylmethacrylate contact lenses in the range of −8.00 to −25.00 diopters; however, there was no significant increase in the prevalence of papillary changes.

The term "giant papillary conjunctivitis" was initially used to describe a syndrome characterized by certain papillary changes. We pointed out[1] that the size of the papillae increased as the disease progressed, and that in the fully exacerbated state the papillae often reached or exceeded 1 mm in diameter. Our experience in this study has led us to conclude that contact-lens-associated conjunctival changes show a spectrum of papillary changes, including elevation of papillae and papillary diameters larger than 0.3 mm.

Summary

The prevalence of elevated papillae more than 0.3 mm in diameter was 10.5% in the conjunctivae of 200 subjects who had successfully worn polymethylmethacrylate hard contact lenses for eight hours or more daily for more than five years. Only three of 500 (0.6%) control subjects who had never worn contact lenses had these papillary changes. The prevalence of papillary changes for those with the so-called normal symptoms of mucus, itching, or both associated with wearing of hard contact lenses was 53% (16 of 21 subjects). The prevalence of these symptoms was 76% among subjects with polymethylmethacrylate contact lenses who had papillary changes and 8% among subjects with polymethylmethacrylate contact lenses who did not have papillary changes. We conclude that changes in the upper tarsal conjunctiva are associated with the wearing of hard contact lenses, occur in a significant percentage of patients wearing hard contact lenses for prolonged periods, and include a spectrum of papillary changes.

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ACUTE ISCHEMIC OPTIC NEUROPATHY IN SEVERE PREECLAMPSIA

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Retinal changes have been observed in 40% to 100% of cases of preeclampsia.[1-9] Despite this prevalence, true amaurosis as a complication of preeclampsia of pregnancy is an unusual occurrence, and only isolated case reports have been published. In most of these cases, loss of vision was secondary to compromise of the retinal arterial circulation. To our knowledge, no one has reported blindness as the result of impairment of the blood supply to the prelaminar portion of the optic nerve head.

CASE REPORT

A 32-year-old, gravida 4, para 1, abortus 2, woman, 32 weeks pregnant, experienced loss of vision in both eyes. She reported that on the previous evening she had developed a severe, throbbing, right-sided headache, which had persisted throughout the night. Upon arising in the morning, she noted that her vision was blurred. Within 30 minutes visual loss progressed until she could barely perceive light. She had had no previous visual symptoms of any kind but had experienced mild preeclampsia during her pregnancy. Her medical history indicated preexisting untreated hypertension.

At the time of initial examination, visual acuity was no light perception in each eye. Ophthalmoscopic examination disclosed chalky-white optic disks with no vessels evident within the substance of the nerve heads. Overlying the optic disks were subtle changes in the vitreous suggestive of a feathery opacification of the posterior aspect of Cloquet's canal (Fig. 1). The retinal arterioles were mildly attenuated but there were no focal spasms. Other findings were unremarkable.

General examination disclosed obesity, hypertension (170/110 mm Hg), a uterus of 34-week gestational size, trace pedal edema, and 4+ proteinuria.

With the onset of seizures, a caesarean section was performed and a live male infant was delivered. The mother received diazoxide because of sustained increase of the blood pressure. Subsequently, the pressure decreased and a transient flushing of the optic disks with lessening of the vasoconstriction in the disk vessels was observed. Results of an electroencephalogram and computed tomography of the head were normal.

On the first day after delivery, her visual acuity was finger counting at 4 feet in the right eye and 2 feet in the left eye. Results of an ophthalmoscopic examination were essentially unchanged from those at admission.

By the third day after delivery, visual acuity had improved to 6/7.5 (20/25) in each eye. There was only a slight return of color to the optic disks, and the retinal vessels remained mildly and diffusely constricted.

Three weeks after delivery, her visual acuity was 6/6 (20/20) in each eye and there was a partial return of color to the optic disks with persistent slight arteriolar narrowing. We also noted retinal striae in the papillomacular area for the first time (Fig. 2). Two months after delivery, her visual field and color perception were normal in both eyes.

DISCUSSION

Retinal changes occurring in preeclampsia were first described by von Graefe in 1855.[10] Most of these changes involve the retinal arterial vasculature, with spasms being the most common finding. Duke-Elder[10] divided the retinal changes into three stages: Stage 1, Arteriolar spasms; Stage 2, Organic changes in vessels; Stage 3, Edema, hemorrhage, and damage to extravascular tissues. Other ocular findings include retinal detachment[10] and spasms of the conjunctival arteries.[11]

Transient blindness has been reported in 1% to 3% of patients with eclampsia.[4,12] With early detection and current methods of treatment of preeclampsia, the present incidence is probably much

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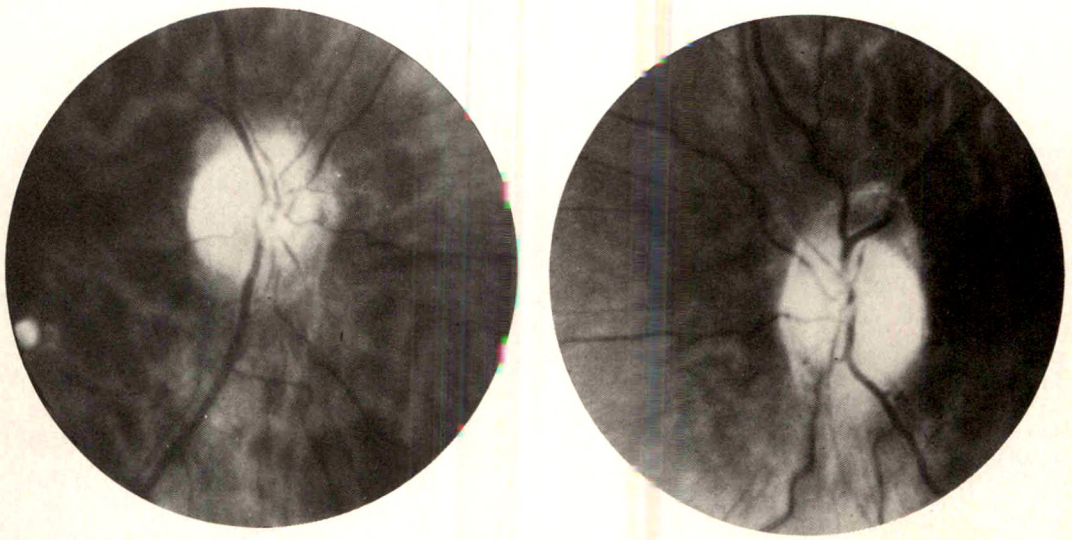


Fig. 1 (Beck and associates). Fundi of right eye (left) and left eye (right) at first examination.

lower. Only a few case reports of blindness lasting longer than a few minutes have been published. All of these cases were associated with marked spasm of the retinal vessels.

Gandhi, Ghosh, and Pillari[13] described a 20-year-old woman with preeclampsia whose blindness resulted from severe spasms of the retinal arterioles with marked retinal edema. Within five days visual acuity returned to normal. Matthew, Rajani, and Gulati[14] reported three cases of visual loss secondary to preeclampsia. In two of these patients the most prominent ocular finding was retinal vasospasm; the results of the ophthalmoscopic examination were not noted in the third patient. Radford and Dow [15] report-

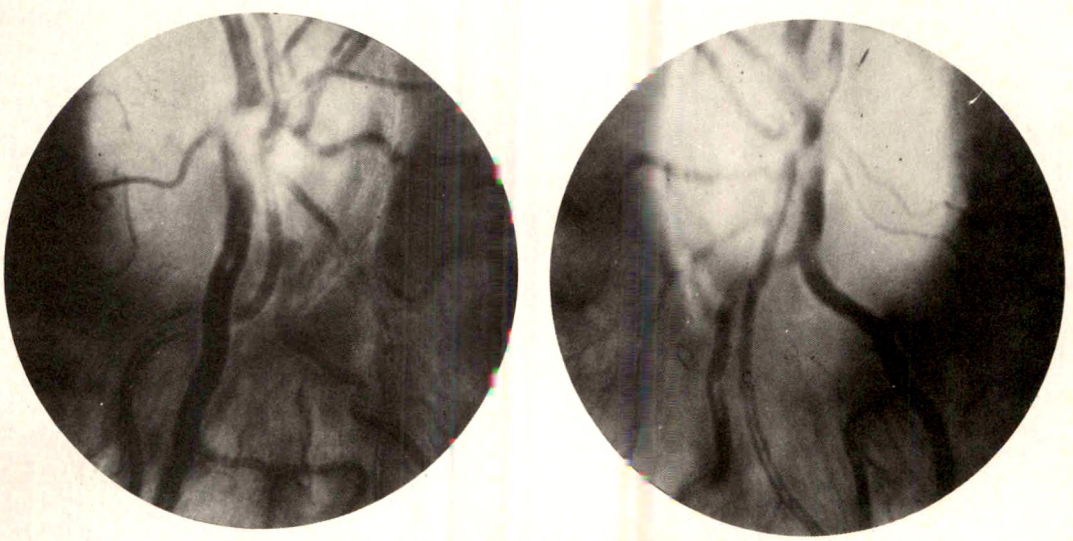


Fig. 2 (Beck and associates). Fundi of right eye (left) and left eye (right) three weeks after delivery.

ed a case of transitory blindness occurring in an eclamptic patient as the result of marked spasm of the retinal vessels. Carpenter, Kava, and Plotkin[16] described a 22-year-old woman with preeclampsia who developed permanent blindness in association with severe compromise of the central retinal artery circulation. Treatment with intravenously administered papaverine, anticoagulants, and a stellate ganglion block did not alter the course. Sommerville-Large[5] reported the case of a 26-year-old woman with preeclampsia who developed total blindness in both eyes with spasm of the retinal arterioles, marked edema of the retina and disks, scattered hemorrhages and exudates, and small retinal detachments in both eyes. Bocev[17] reported two cases of complete visual loss lasting several days in patients with preeclampsia whose fundi showed spasm of the retinal arterioles. Ingiulla[18] described four other cases of preeclampsia in which amaurosis resulted primarily from spasm of the retinal vessels. In one of these cases retrobulbar injection of procaine HCl (Novocain) was effective in restoring visual acuity, whereas in two of the others intravenously administered procaine HCl had a beneficial effect. In each case, visual acuity completely returned to normal.

In all of the preceding reports visual loss could be explained on the basis of severe impairment of the vascular circulation of the retina. Skenderovic and Pestelek[19] and Kelly[20] each described a case of transitory blindness occurring in the presence of optic disk pallor and retinal vascular changes. Both of these cases were presented from the obstetrical standpoint, however, with only cursory reference to ophthalmologic findings; thus, no conclusions about the origin of the amaurosis can be drawn. Cases of blindness occurring with normal fundi have also been reported in association with preeclampsia.[19,21] In some of these cases loss of vision has been attributed to occipital lobe ischemia or hysteria.[4,12,16,22] Dieckman[4] suggested that an occipital lobe disturbance is the usual origin of amaurosis occurring in preeclampsia. In our patient, results of computed tomography of the brain were normal, making this an unlikely possibility.

To our knowledge, no documented reports exist in which retinal vascular changes were minimal and profound pallor of the optic disk strongly supported acute ischemic optic neuropathy as the primary cause of blindness in a patient with preeclampsia. The only similar reported case, to our knowledge, was a patient with acute intermittent porphyria.[23] In patients with porphyria, spasms have been observed in the retinal vessels and, on occasion, generalized angiospastic activity throughout the body has been noted.[24,25] As in preeclampsia, the vasospasms may be secondary to a toxic or biochemical defect, although no specific mechanism has been identified.[23] In this reported case of porphyria, blindness apparently resulted from ischemic optic neuropathy in a fashion similar to our case.

The pathophysiology of selective ischemia of the optic nerve head in preeclampsia is speculative. The blood supplies of the retina and optic disk have been shown experimentally to be distinct. Hayreh,[26] Ernest and Potts,[27] and others have demonstrated in monkeys and humans that the prelaminar optic nerve head receives its blood supply from the posterior ciliary circulation and not from the central retinal artery or its branches. Thus, there is an anatomic basis for selective ischemia of the optic disk. However, the branches of the posterior ciliary artery from the peripapillary choroid and the circle of Zinn, which supply the nerve head, are currently thought to be devoid of musculature within their walls. Thus, vasospasm is an unlikely mecha-

nism for hypoxia in this region. If the short posterior ciliary arteries themselves were in vasospasm, changes in the choroid and outer retinal layers would be expected; yet these were not observed clinically.

Conversely, Ernest and Potts[27] have shown in rhesus monkeys that the intravascular pressure in the posterior ciliary circulation to the optic disk is lower than the intravascular pressure in the central retinal artery or its branches. Thus, generalized vasoconstriction in the posterior ciliary and retinal circulations might cause decreased circulation to the prelaminar portion of the optic nerve because of the effective lowering of perfusion pressure in this region by intraocular pressure, while the retinal circulation remained relatively intact. Additionally, a decrease in posterior ciliary circulation to the choroid may have been clinically unapparent. This appears to be the most logical physiologic basis for the events observed in this patient.

Although the rapid onset of optic disk pallor in association with severe visual loss may result from acute inflammation, our patient had no inflammatory signs of the optic nerve head. Furthermore, vasospasm is the hallmark of preeclampsia, and the acute infiltration of nerve tissue with inflammatory cells in this clinical setting is extremely unlikely. Thus, we believe selective ischemia of the optic nerve head was the most likely cause of this patient's visual loss and may, indeed, be a more common cause of blindness in preeclamptic patients than has been previously recognized. In some patients, the presence of such changes in the optic nerve may be obscured by the more spectacular and widely reported spasms of the central retinal artery.

We were unable to determine precisely the length of time during which our patient was totally blind, but this episode apparently lasted several hours. Complete recovery following such an episode is difficult to explain, given our knowledge of the reaction of the central nervous tissue to hypoxia, yet a review of published reports discloses several cases of similar recovery after several hours or even days of complete blindness in association with preeclampsia.

The transient flushing of the optic nerve heads observed after delivery is interesting. The role of diazoxide in this vasodilation and in restoration of vision cannot be evaluated on the basis of this single case.

Summary

A 32-year-old woman developed bilateral blindness along with severe preeclampsia of pregnancy. Ophthalmoscopic examination disclosed chalky-white optic disks with only minimal changes in the retinal vasculature. After caesarean section, the preeclampsia improved, and complete return of visual acuity occurred during a three-day period.

Blindness has rarely been reported as a complication of preeclampsia. In almost all cases it has been associated with severe compromise of the retinal vasculature. Conversely, the ophthalmoscopic findings in our case strongly support acute ischemic optic neuropathy as the cause of blindness.

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SARCOIDOSIS WITH NEOVASCULARIZATION OF THE OPTIC NERVE HEAD

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Sarcoidosis is a multisystem disease process with characteristic histopathologic features of noncaseating granulomas or naked epithelioid granulomas. These granulomas are surrounded by only a scattering of lymphocytes. The granulomas can be located in any tissue of the body. Involvement of the eye and its adnexa has been reported in 7% to 64% of patients with sarcoidosis,[1,2] but most reports describe an ocular involvement in about 25%.[3-7]

Iridocyclitis is the most common ocular manifestation of sarcoidosis. The lacrimal gland is involved in about 7% of patients with sarcoidosis,[1] producing a firm and enlarging lesion in the lacrimal fossa. Involvement of the lacrimal gland may produce keratitis sicca, from insufficiency or abnormality of tears. Noncaseating granulomas of sarcoidosis account for about 5% of lacrimal gland tumors.[8] Subcutaneous nodules of the eyelids may occur in sarcoidosis. The orbit can be involved primarily[9] or by extension from the paranasal sinuses.[10] The conjunctiva and episclera are often involved in sarcoidosis, and biopsy of this area is now recommended for diagnosis of uncertain cases.[11] Flat confluent follicles of the palpebral conjunctiva are more likely to show the characteristic pathologic features of sarcoidosis. Involvement of the retina was first documented in 1939 by Walsh,[12] who noted yellow-white perivascular retinal lesions, which were later termed "candle-wax drippings."[13]

Features of sarcoidosis of the ocular fundus[14,15] are chorioretinal spots, retinal periphlebitis, macular edema, and optic nerve edema. There may also be chorioretinal granulomas, chorioretinitis, optic nerve atrophy, and preretinal infiltrates. Asdourian, Goldberg, and Busse[16] described peripheral retinal neovascularization in a "sea-fan" configuration.

We studied a patient with generalized sarcoidosis, with neovascular fronds on the optic nerve head in both eyes. There was an associated vitreous hemorrhage bilaterally. With corticosteroid therapy, the neovascular process resolved dramatically.

CASE REPORT

A 25-year-old man with a four-month history of progressive weight loss, anorexia, tiredness, and occasional blurred vision was examined by his physician. Before the onset of symptoms, the patient had been a heavy-equipment operator and also engaged in sports. He denied cardiovascular, gastrointestinal, genitourinary, or neurologic symptoms, and he had no history of ocular problems. Examination showed a generalized lymphadenopathy and bilateral hilar adenopathy on chest X-ray. Pathologic study of a scalene lymph-node biopsy specimen showed a lesion compatible with sarcoidosis, although lymphoma could not be ruled out. The physician referred the patient here for further examination.

At admission the patient appeared healthy and was afebrile. There was a generalized lymphadenopathy, with palpable nontender cervical, supraclavicular, axillary, epitrochear, and inguinal lymph nodes. Examination of the lungs, heart, and abdomen gave normal results. The patient was referred to us for ophthalmologic consultation because of abnormalities noted in the ocular fundus.

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Visual acuity was 6/6 (20/20) in both eyes. Intraocular pressure by applanation tonometry was 14 mm Hg in both eyes. Visual fields were normal. We noted no fullness in the lacrimal fossa and no subcutaneous nodules. The conjunctiva was not injected, but a few follicles were present (they were not confluent or broad-based). The cornea, iris, and anterior-chamber angle appeared essentially normal, and there were no anterior or posterior synechiae. The vitreous had a mild cellular reaction. No Landers nodules were present.

We found neovascular fronds, which extended approximately 5 to 6 mm into the vitreous, on the optic nerve heads in both eyes. The neovascular tissue showed budding and small hemorrhages of the terminal capillaries. The right eye had a horizontally layered hemorrhage, contiguous with the detached vitreous base and the neovascular frond from the disk (Fig. 1). The other retinal vasculature showed no abnormalities on direct and indirect ophthalmoscopic examination. We noted no perivascular sheathing or candle-wax drippings, and there were no localized retinal hemorrhages or exudates.

Fluorescein angiography was done before beginning corticosteroid therapy and in serial studies thereafter, with a total follow-up of six months. The angiograms done before systemic corticosteroid therapy showed some abnormalities. The retinal vascular pattern was obscured and the fluorescence was blocked by scattered overlying vitreous hemorrhage. The arterial phase of angiograms showed the symmetrical filling of the retinal arteries at ten seconds after injection of the dye (Fig. 2). In the late phases, some fluorescein staining was visible along the vein wall, both above an below the macula, in both eyes. The smaller venules showed staining that progressed to leakage in the late phase, especially around the macula. The small arterioles appeared intact, with no staining or leakage. The retinal capillaries appeared normal throughout the study, with no evident areas of capillary nonperfusion.

The most striking abnormality was in the area of the optic nerve head. Here there was an irregular network of vessels overlying and partially obscuring the retinal circulation. These new-formed vessels filled in the arterial stage of the angiogram and leaked profusely in the late stage. The neovascular pattern was similar in both eyes. The vessels on the optic nerve heads were larger than capillaries, about one half the diameter of normal retinal arteries. The vitreous hemorrhage, and thus the areas of blocked fluorescence, appeared to be associated with the neovascular membrane. The details of the optic nerve itself were obscured by the neovascular network, but the nerve head did not appear to be elevated. In summary, the major angiographic findings were documentation of the vitreous hemorrhage, a widespread periphlebitis, absence of capillary closure, and a vivid neovascular membrane on both optic nerve heads.

Laboratory studies showed a microcytic anemia with a hematocrit of 35.2% and hemoglobin level of 11.6 g/100 ml. The white blood cell count was 6,400/mm^3 with a normal differential cell count. Other pertinent findings were: total protein, 7.4 g/100 ml (normal, 6.4 to 8.2 g/100 ml), albumin,

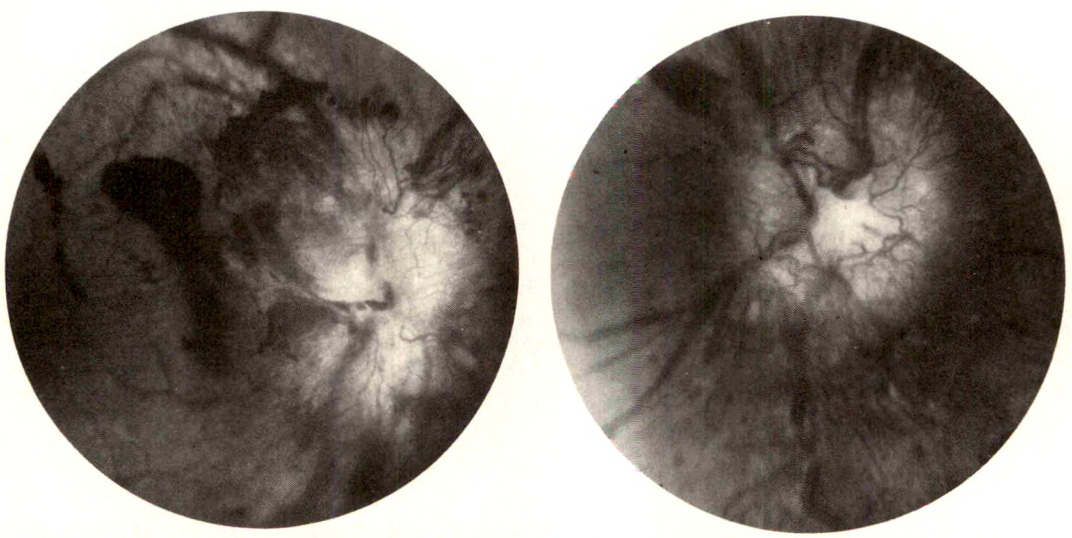


Fig. 1 (Doxanas, Kelley, and Prout). Optic disks in the right (left) and left (right) eyes. There is neovascularization of the optic nerve before corticosteroid therapy and budding of terminal capillaries. A layer of vitreous hemorrhage obscures some retinal details. The elevation of the neovascular process is evident by the different focus plane of the retinal vessels.

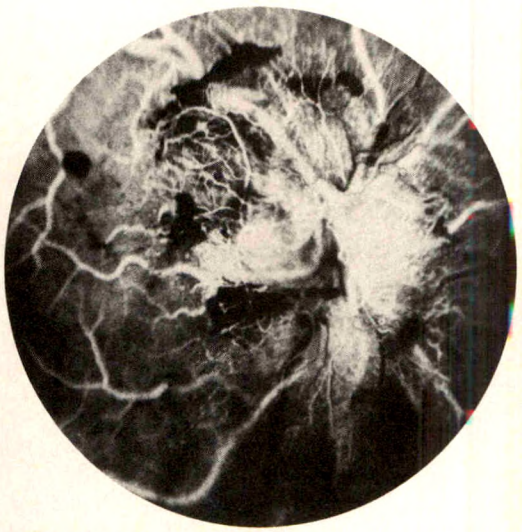


Fig. 2 (Doxanas, Kelley, and Prout). Fluorescein angiogram of the right eye shows an irregular network of vessels that obscures the retina. Leakage of the neovascular process is already evident in early stages of the angiogram, and leakage became more prominent in later stages.

3.8 g/100 ml (normal, 3.7 to 5.2 g/100 ml); serum globulin, 3.6 g/100 ml (normal, 2.0 to 3.6 g/100 ml); albumin-globulin ratio, 1.06 (normal, 1.15 to 2.07). Additional laboratory tests, including collagen vascular screening, sickle-cell test, hemoglobin, electrophoresis, and erythrocyte sedimentation rate, gave normal results. Radiologic evaluation confirmed the bilateral hilar adenopathy. Pulmonary function tests showed a normal vital capacity and normal forced-expiratory volume. Bone marrow aspirate and right epitrochlear node biopsy specimens revealed non-caseating granuloma. Staining results for acid-fast bacilli and fungi were negative.

We believed the patient had systemic sarcoidosis involving the lungs, lymph nodes, eyes, and bone marrow, and we began giving him prednisone, 80 mg/day. After starting therapy, he noted a marked symptomatic improvement. As the patient improved during the next three months, the prednisone was tapered off to a maintenance dosage of 10 mg/day. While he received corticosteroid therapy, the patient's visual acuity remained 6/6 (20/20) in both eyes. The neovascularization of the optic disk resolved dramatically, with no residual disk abnormalities (Fig. 3).

Follow-up fluorescein angiograms showed a dramatic change when compared to the angiograms made before corticosteroid therapy. These changes occurred during a ten-week interval. Unlike the pretreatment angiograms, the veins now showed no late staining (Fig. 4). The small venules showed one or two areas of slight hyperfluorescence near the macula. The most striking change was the nearly total absence of the preexisting neovascular membrane. There was no early-stage fluorescein filling of the neovascular membrane and no late leakage. Both optic nerves showed a slight hyperfluorescence, but no evidence of neovascular tissue.

Discussion

Optic nerve sarcoidosis can have four different (but overlapping) manifesta-

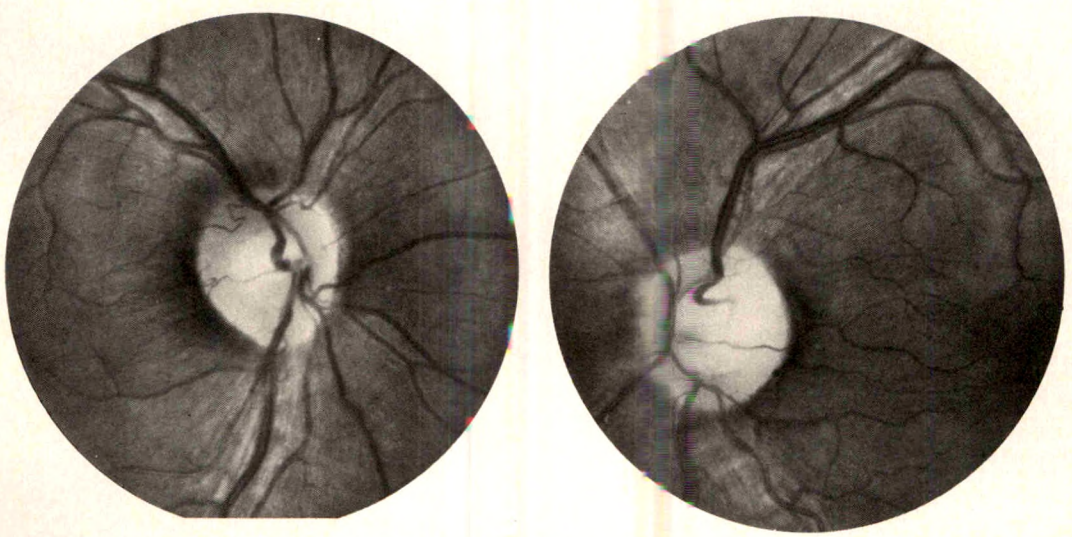


Fig. 3 (Doxanas, Kelley, and Prout). Optic nerve head after corticosteroid therapy in the right (left) and left (right) eyes. There is a regression of the neovascular membrane. No residual abnormalities are evident.

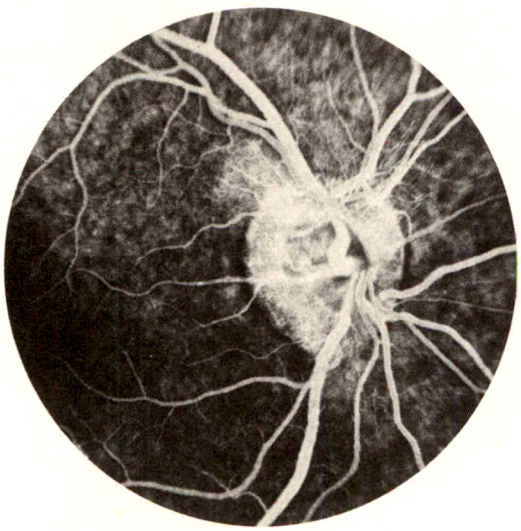


Fig. 4 (Doxanas, Kelley, and Prout). The neovascular process is no longer present and there is no leakage of fluorescein 3½ minutes after injection. At later stages of the angiogram, some hyperfluorescence was present at the optic nerve head.

tions: optic nerve neuritis, papilledema, optic nerve atrophy, and granulomas of the optic nerve.[17-23] Optic nerve edema in sarcoidosis can be caused by diffuse intracranial involvement with or without increased infiltration of the optic nerve with sarcoid granulomas. Neovascularization of the ocular nerve head has not previously been documented.

It is usual in sarcoidosis to obtain dramatic resolution of lesions with prednisone therapy. Some processes, such as macular edema, respond to prednisone therapy more slowly.[24] Prednisone does not, of course, reverse optic nerve atrophy.

In the present case, that of a 25-year-old man with systemic symptoms and generalized lymphadenopathy, the neovascular fronds extending from the ocular disk were an unusual feature of sarcoidosis. Although we noted no perivasculitis on direct ophthalmoscopy, fluorescein angiography disclosed evidence of vasculitis. There were small areas of fluorescein staining and leakage along terminal vessels near the macula. Prednisone therapy produced a prompt and dramatic resolution of this neovascular frond and the slight vasculitis. After three months of prednisone therapy there was no ophthalmoscopic or angiographic evidence of the neovascular process, and the vitreous hemorrhage had entirely cleared.

This case demonstrates several interesting features. Although neovascular fronds in sarcoidosis have occurred in the peripheral retina,[14] we could find no published reports of neovascularization of the optic nerve head associated with sarcoidosis. The only other description of neovascularization was that of Asdourian, Goldberg, and Busse.[16] They observed peripheral fundus neovascularization in a sea-fan configuration in three patients with sarcoidosis with no evidence of sickle-cell disease. Therapy in the three reported cases was specified in only one; the patient was treated with argon laser photocoagulation. Although treatment ablated the neovascular process, a neovascular tuft returned in six months, producing a vitreous hemorrhage. After the vitreous hemorrhage resolved, they found a total retinal detachment. The patient was scheduled to undergo retinal reattachment surgery; no corticosteroid therapy was used.

Jampol, Woodlin, and McLean[20] described an atypical example of optic nerve sarcoidosis. Their patient had markedly enlarged optic nerve heads, which protruded into the vitreous. However, stereoscopic photographs documented the absence of neovascularization. They noted that the dramatic response to corticosteroids in their case suggested a sarcoid granuloma directly affecting the optic nerve head. They said granulomas of the optic nerve may be a more common manifestation of sarcoidosis than has been recognized.

The second curious feature of our case

is the dramatic resolution of the neovascular frond after the start of corticosteroid therapy. The dramatic response of sarcoidosis to corticosteroid therapy, especially retinal perivasculitis and optic nerve granulomas, is a consistent feature of the disease. Retinal neovascularization is not commonly thought to respond to corticosteroids. However, in our case of neovascularization of the disk, the response to corticosteroid therapy may be a feature of neovascular processes. This association, based on a single case report, is speculation.

Summary

A 25-year-old man with systemic sarcoidosis had neovascularization of the optic nerve heads and associated bilateral vitreous hemorrhages. Oral administration of prednisone produced a prompt resolution of these unusual neovascular fronds and cleared the vitreous hemorrhages.

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OPTIC DISK VASCULITIS

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Occlusion of the central retinal vein is readily recognized ophthalmoscopically by a swollen optic disk, distended, tortuous retinal veins, and widespread retinal hemorrhages.[1,2] In older patients, without glaucoma or hematologic hyperviscosity syndromes, this condition is usually attributed to concomitant arteriosclerotic arterial disease.[3-6] In young healthy patients without evidence of systemic arterial disease, inflammation of the retinal or papillary vessels, or both, has been speculated to be the cause,[3,5-9] although infrequently confirmed histopathologically.[10,11] We describe the clinical and histopathologic findings of a 44-year-old woman who initially had diffuse retinal vascular abnormalities associated with optic disk edema, and subsequent optic atrophy.

CASE REPORT

A 44-year-old woman had gradually progressive, painless loss of vision of her right eye for six months before admission here on Aug. 8, 1977. Two months previously, visual acuity of the eye was 6/60 (20/200) and a swollen disk was noted. A ten-day course of orally administered corticosteroids had produced no change. The patient had not used oral contraceptive medications.

On admission, neuro-ophthalmic examination showed visual acuities of L.E.: 6/4.5 (20/15) and R.E.: hand movements. The right eye was normal externally. Ocular motility was normal. The pupils were equal, and a marked afferent pupillary defect was present in the right eye. Visual fields showed only two islands of vision remaining in the superior and inferior fields of the right eye. Biomicroscopy showed minimal rubeosis iridis without cells or flare in the anterior chamber in the right eye, and intraocular pressure (IOP) was R.E.: 11 mm Hg and L.E.: 14 mm Hg. Ophthalmoscopy of the right eye (Fig. 1) showed a markedly atrophic optic disk. Extensive retinal hemorrhages were present inferiorly. There was marked sheathing of the branches of the central retinal artery and vein. Caliber variation was noted in both vessels and prominent beading occurred in the superior temporal vein. Fluorescein angiogram (Fig. 2) showed filling of the central retinal artery but no filling of the inferior nasal arteriole, branches of the inferior temporal arteriole, and the peripapillary choroid. There was slow filling of the superior branches of the central retinal vein but no filling of the inferior branches. Ophthalmodynamometry was 70/35 bilaterally, with a brachial blood pressure of 130/72 mm Hg. No carotid or ocular bruits were audible.

General physical examination showed only obesity and scattered pulmonary wheezes attributed to chronic bronchitis related to smoking two packs of cigarettes daily. Neurologic examination was unremarkable, except for symmetric hyporeflexia.

Laboratory tests showed normal values for hematocrit, white blood cell count and differential, erythrocyte sedimentation rate, and serum concentrations of glucose, urea nitrogen, calcium, phosphorus, bilirubin, alkaline phosphatase, lactic acid dehydrogenase, total protein, and albumin. Lipoprotein phenotyping indicated Type IV lipoproteinemia with a triglyceride level of 288 mg/100 ml (normal, 10-160 mg/100 ml) and cholesterol value of 213 mg/100 ml (normal, 150-280 mg/100 ml). Serology tests were negative for rheumatoid factor, cryoglobulins, fluorescent antibody profile, and the VDRL test. Roentgenograms of the skull and chest were normal, as were electroencephalogram and electrocardiogram. Films of bilateral carotid and left vertebral arteriography that had been performed elsewhere in July 1977 were reviewed and were normal. The right ophthalmic artery was visible and appeared normal.

The patient received an additional course of orally administered corticosteroids, beginning with 100 mg of prednisone daily for two days, with gradual reduction of dosage over eight weeks. There was no improvement in either the function of the right eye or its ophthalmoscopic appearance on Aug. 29, 1977 (Fig. 3). In mid-September, she developed secondary glaucoma, and in October the eye had no light perception and an IOP of 65 mm Hg. The right eye

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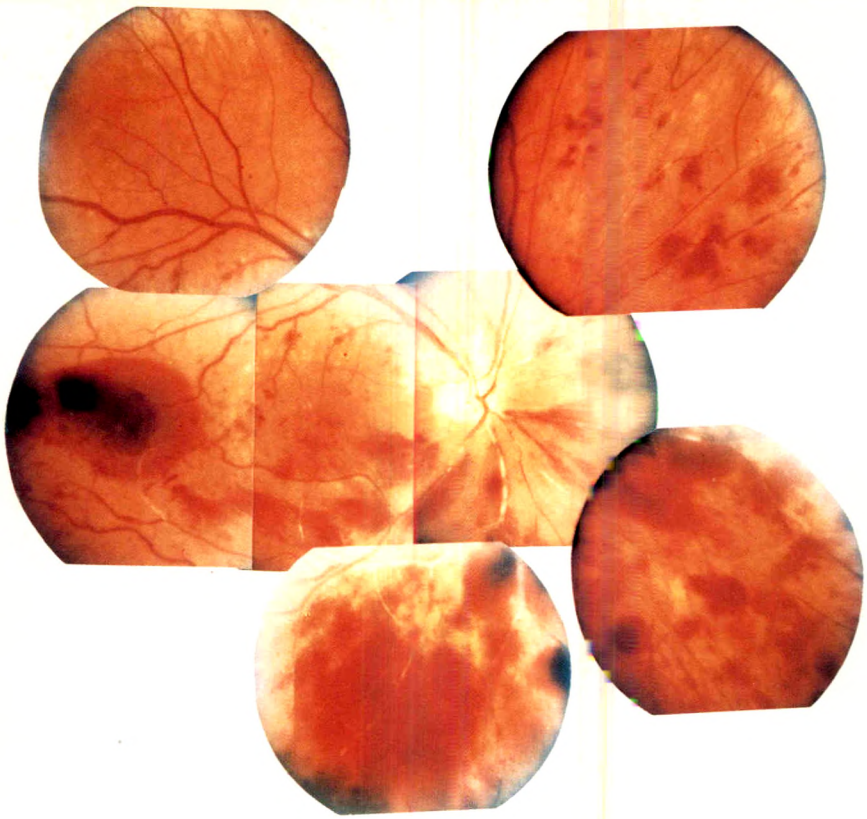


Fig. 1 (Appen, de Venecia, and Ferwerda). Montage of fundus appearance of the right eye on Aug. 10, 1977, showing optic atrophy, diffuse retinal hemorrhages, sheathing of the retinal arterioles, and distention of the retinal veins.

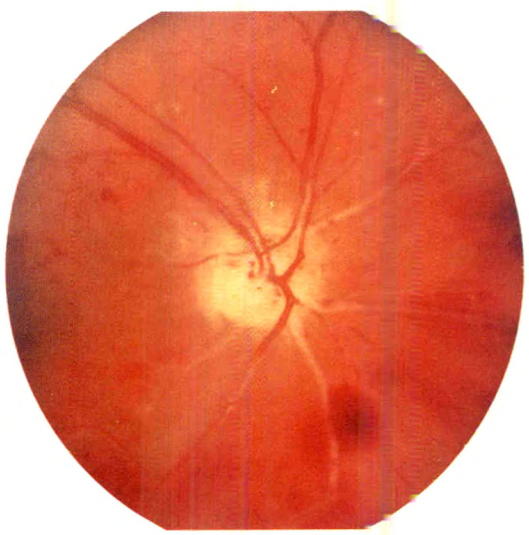


Fig 3 (Appen, de Venecia, and Ferwerda). The right fundus on Aug. 29, 1977, six weeks

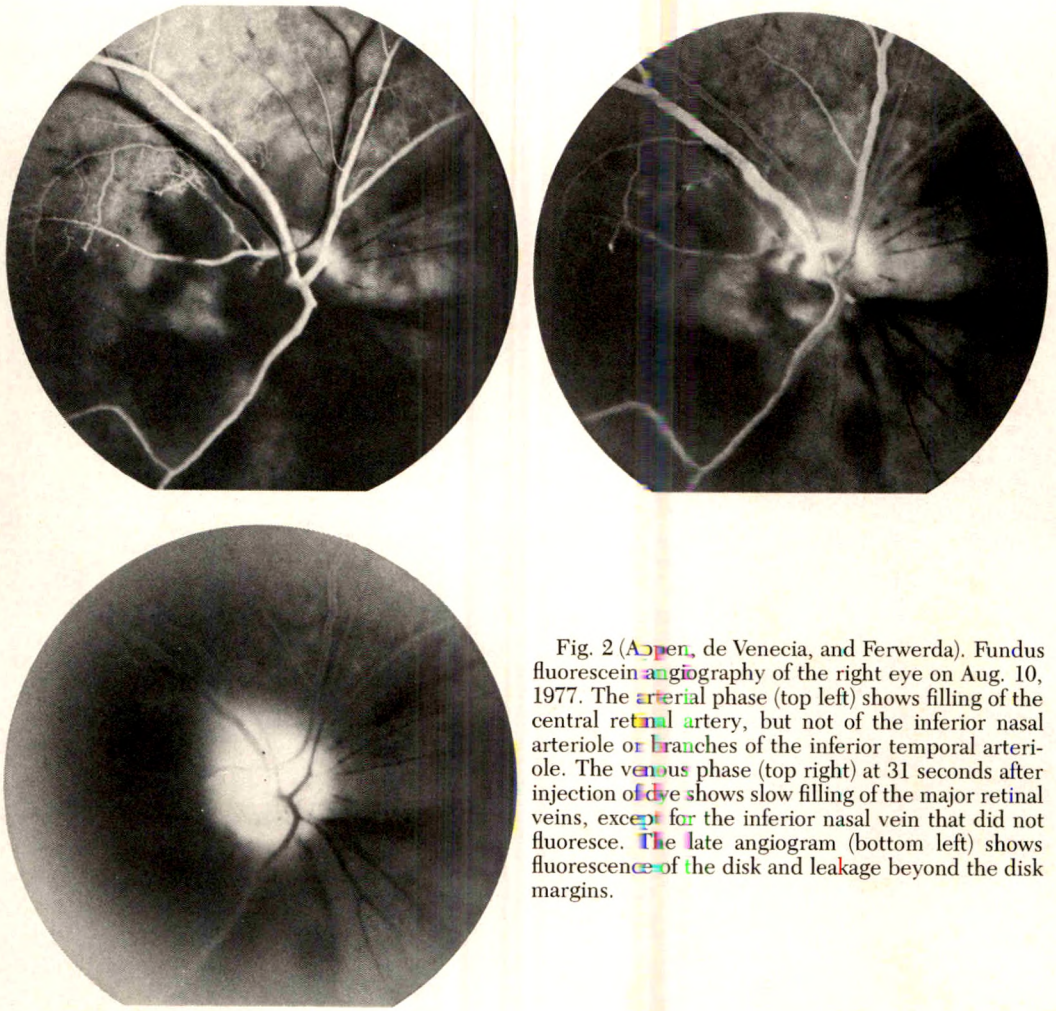


Fig. 2 (Appen, de Venecia, and Ferwerda). Fundus fluorescein angiography of the right eye on Aug. 10, 1977. The arterial phase (top left) shows filling of the central retinal artery, but not of the inferior nasal arteriole or branches of the inferior temporal arteriole. The venous phase (top right) at 31 seconds after injection of dye shows slow filling of the major retinal veins, except for the inferior nasal vein that did not fluoresce. The late angiogram (bottom left) shows fluorescence of the disk and leakage beyond the disk margins.

was enucleated on Oct. 15, 1977, six weeks after the most recent fundus photograph. Twenty-one months later, she remained well with no abnormality of her left eye.

Results

The normal-sized eye contained a 7-mm stump of optic nerve and was fixed in 10% buffered formalin solution. The optic nerve was cut 4 mm from the globe, and the portion separate from the eye was serially sectioned transversely. The globe was sectioned vertically, and the portion containing the optic nerve and the peripapillary area was serially sectioned at 8 μ. Alternate sections were stained with PAS reagent, hematoxylin and eosin, and Lendrum stains.

The anterior chamber angle was occluded by peripheral anterior synechiae. There was a dense neovascular membrane on the anterior iris surface that was especially prominent near the pupil, extending peripherally into the angle. There was no inflammation involving the iris or ciliary body, and the lens and vitreous were unremarkable.

The retina was artifactually detached, and extravasated blood involved all lay-

ers posterior to the equator superiorly. The optic nerve showed cupping, posterior bowing of the lamina cribrosa, and infarction of the distal portion of the nerve (Fig. 4, left). The optic nerve, which was 4 mm posterior to the globe, contained the central retinal artery and vein (Fig. 5, top left and right). The central retinal vein showed mild cuffing by a mononuclear infiltrate, and the central retinal artery was patent. The optic nerve pial vessels showed extensive phlebitis and obliteration of the lumens of several arterioles (Fig. 5, bottom left). The material within the arteriolar lumen stained positively with PAS and Lendrum for fibrin. The central retinal vein in the retrolaminar optic nerve was practically obliterated by a marked mononuclear inflammatory infiltrate (Fig. 4, right). There was thickening of the walls of the central retinal artery, which contained an intact internal elastic lamina. The markedly sheathed retinal veins noted clinically showed on serial sections obliteration of the vessel by an amorphous material that contained some fibroblasts and macrophages (Fig. 6).

Discussion

Lonn and Hoyt[3] and Ellenberger and Messner[9] used the term "papillophlebitis" to describe the syndrome in which young healthy persons sustain a unilater-

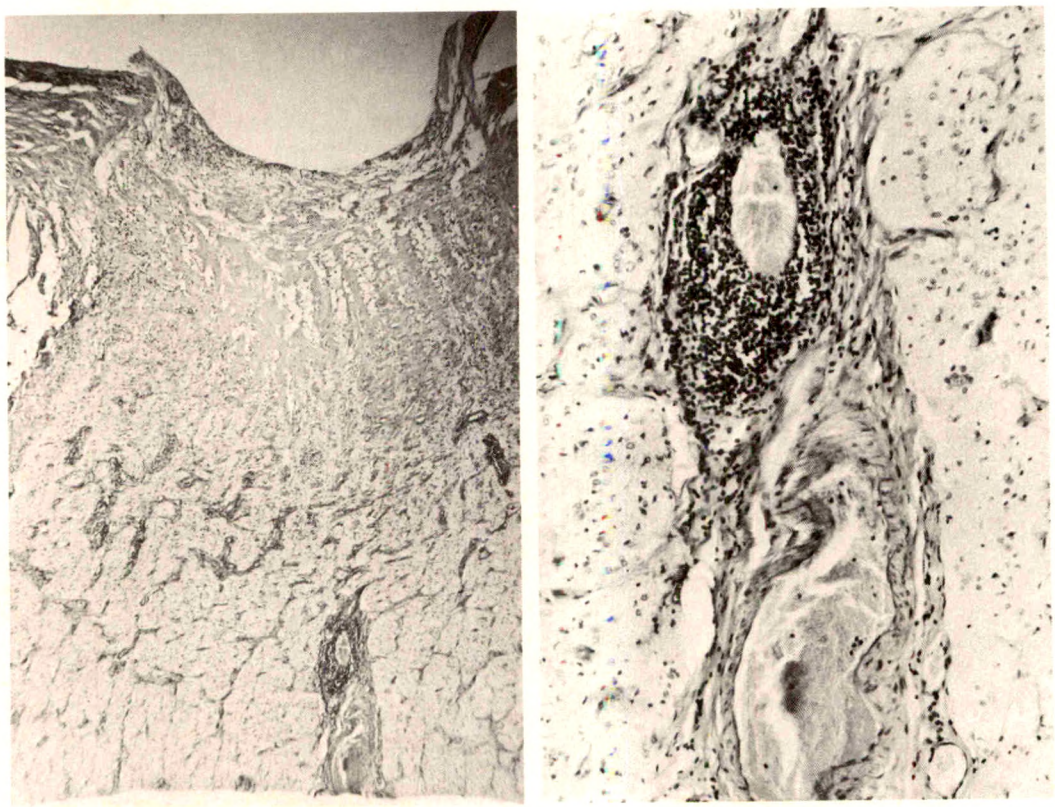


Fig. 4 (Appen, de Venecia, and Ferwerda). Left, There is cupping and infarction of the optic nerve head (hematoxylin and eosin, ×4). Right, Higher power view of the central retinal artery and vein seen in Figure 4, left. The artery is patent, and a dense lymphocytic inflammatory infiltrate surrounds the vein (hematoxylin and eosin, ×21).





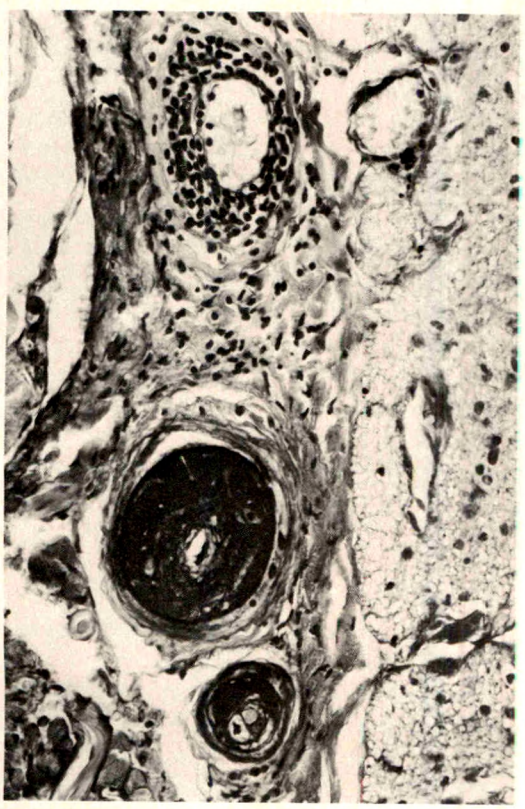


Fig. 5 (Appen, de Venecia, and Ferwerda). Top left, Cross section of the more posterior portion of the optic nerve containing the central retinal artery and vein in its center (hematoxylin and eosin, ×5). Top right, Higher power view of the central retinal artery and vein shows them to be patent with a minimal lymphocytic infiltrate about the vein (hematoxylin and eosin, ×25). Bottom left, Higher power view of the small vessels at the periphery of the optic nerve (See arrow in Figure 5, top left) shows phlebitis and obliteration of the lumen of the arterioles (hematoxylin and eosin, ×53).



Fig. 6 (Appen, de Venecia, and Ferwerda). Microscopic appearance of the sheathed inferior temporal retinal vessels noted ophthalmoscopically near the optic disk margin. The arterial branch (A) has a patent lumen, whereas that of the vein (V) is obliterated by the amorphous fibrinous material (hematoxylin and eosin, ×16).

al, almost asymptomatic, central retinal venous occlusive disease with edema of the optic disk, with gradual resolution in six to 18 months. Similar patients have been described with different diagnostic labels including retinal vasculitis,[5,7] optic disk vasculitis,[8] and retinal and papillary vasculitis.[10] Cogan[10] emphasized that there is a spectrum of severity of involvement in this group of conditions and suggests that the groups described by others[1,3,5,7-9] fall into the "benign" or mild classification. Other patients have moderate to marked loss of vision and abnormality of the retinal vasculature and frequent development of neovascular glaucoma. Cogan described the histologic findings in four eyes of such patients as being similar to those of our patient.

To test the possibility that the vasculitis seen in our patient was an effect of the four to six weeks of marked glaucoma, we reviewed the sections of ten consecutive eyes processed by our ocular pathology laboratory for which neovascular glaucoma was the primary histologic diagnosis. Two of the ten eyes showed a mild accumulation of inflammatory cells in the episcleral tissue of the globe or around the tiny pial vessels at the periphery of the optic nerve, but none of them had the concentrated inflammatory infiltrate around the central retinal vessels that was present in this patient. We conclude, therefore, that the vasculitis of our patient was a primary process causing the vascular disturbance, and was not a response to the glaucoma.

Recently, Stowe, Zakov, and Albert[12] reported a mild perivascular inflammatory infiltrate in the optic nerve of a 40-year-old woman who sustained a central retinal vascular occlusion while taking anovulatory agents orally. They concluded that the vasculitis was part of a prolonged inflammatory response to the thrombotic debris in the occluded vessels. In our patient this was not applicable because of the intensity of the infiltrate, the patency of the central retinal vein, and the shorter duration of the disorder.

We speculate that the initial lesion in our patient was a diffuse phlebitis that caused obstruction of multiple retinal and pial veins to the optic nerve. This stagnation of blood flow may have produced thrombus formation in accompanying arterioles and the resultant ischemic infarction of the optic nerve head. We agree with the concept of a spectrum of optic nerve vasculitis, and suggest that documentation of the optic nerve phlebitis in our patient supports the accuracy of the suffix "itis" in the terms "papillophlebitis" or "papillary and optic disk vasculitis" in the more benign variants. The ultimate cause of the condition remains obscure, but does not necessarily appear to be associated with a systemic vasculitis. However, the histologic findings indicate that the appropriate therapy, when warranted, would be an anti-inflammatory agent, such as corticosteroids.

Summary

A 44-year-old woman developed progressive loss of vision associated initially with a swollen optic disk, and later with optic atrophy and a diffuse retinal vasculopathy, which caused extensive retinal hemorrhage. Histopathologic examina-

tion showed hemorrhagic infarction of the retina, as well as infarction of the anterior optic nerve. In the optic nerve, the central retinal vessels showed extensive phlebitis and occlusion of many small arterioles.

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BIOMETRY OF 7,500 CATARACTOUS EYES

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Previous studies by Sorsby[1] and others have provided estimates of the normal ranges of some of the clinical measurements of the eye's refractive status. These studies, however, were on patients without cataracts and with known refractions, so that correlations were possible. Oguchi and Van Balen[2] reported preoperative and postoperative measurements of 42 patients with intraocular lenses. They found steeper corneas after implant surgery and a 0.02-mm mean shortening in axial length (range, -0.7 to +0.9 mm); they also noted that shallow chambers deepened and deep chambers shallowed after intraocular lens surgery. Leonard,[3] in a series of 26 eyes, noted a 0.2-mm (0.6-diopter) mean decrease in axial length after intraocular lens surgery in 15 of 26 eyes. Yu and associates[4] used ultrasound to measure axial length in the eyes of 1,789 normal Chinese young adults (18 to 25 years old). They found a mean length of 23.74 mm (±1.24) and a average difference between fellow eyes of 0.42 mm.

My analysis of the biometric measurements of 7,500 eyes provides important new information and suggests that a revision of some commonly accepted figures and formulae is needed.

MATERIAL AND METHODS

I collected data on patients examined for cataract surgery with intraocular lens implantation between 1974 and 1979. Each patient had some form of cataractous change in one or both eyes. I measured axial length with various A-scan ophthalmic ultrasound devices (Kretz 7200MA, Sonometrics DBR-300, Xenotec, and Britt/DOC) and took keratometric readings. I measured anterior chamber depth with a pachymeter in 60% of the eyes and with A-scan ultrasound in the remaining 40%.

I examined exactly 7,500 eyes; from these, I selected a random series of 3,600 (1,800 patients) for a separate comparison of fellow eyes. An independent biostatistician performed the data analysis.

RESULTS

Of the 7,500 eyes, 6,950 were phakic, 400 were aphakic, and 150 were pseudophakic (Table 1). The mean age of the patients was 72 years (±10), with a range of 0.1 to 97 years (Fig. 1). The group with aphakic eyes was older than the groups with phakic or pseudophakic eyes by two years ($P<.002$ by Student's *t*-test).

The mean axial length (Table 1) of the 7,500 eyes was 23.65 mm (±1.35). Figure 2 shows the distribution. A comparison of the axial lengths of phakic (23.65 mm [±1.35]), aphakic (23.64 mm [±1.44]), and pseudophakic (23.64 mm [±1.45]) eyes shows no statistically significant difference. These data indicate that there is no significant shortening of the eye after cataract surgery, a finding which disagrees with those of other investigators used by Binkhorst[5] in his formula for lens calculation.

The mean average corneal power for this series was 43.81 diopters (±1.60). It is important to compare eyes that were operated upon with those that were not, since Binkhorst's formula takes into ac-

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TABLE 1

ANALYSIS OF 7,500 EYES

	Eyes						Total of the Three Groups (N=7,500)	
	Phakic (N=6,950)		Aphakic (N=400)		Pseudophakic (N=150)			
Measure	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
Age in years	72	±10	70	±10	72	±10	72	±10
Axial length (mm)	23.65	±1.35	23.64	±1.44	23.64	±1.45	23.65	±1.35
Keratometric reading (diopters)	43.81	±1.60	43.65	±1.60	43.78	±1.60	43.81	±1.60
Anterior chamber depth (mm)	3.24	±0.44	3.76	±0.48	3.32	±0.43	—	—
Astigmatism (diopters)	1.00	±1.00	1.50	±1.25	1.65	±1.25	—	—
Emmetropic IOL power (diopters)*	17.00	±4.50	18.00	±4.00	17.00	±4.00	17.00	±4.50
Central endothelial cell count (cells/mm^2)	2,538	±503	1,792	±680	1,737	±767	2,470	±570

*Prepupillary style.

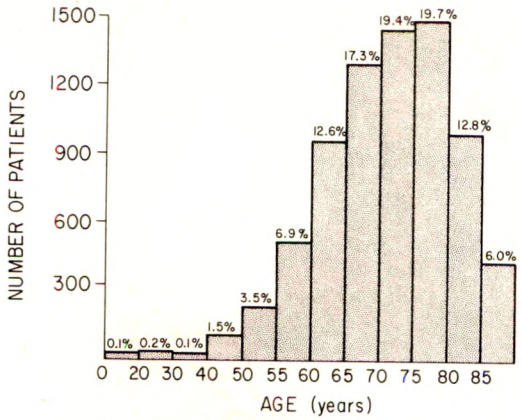


Fig. 1 (Hoffer). Age distribution.

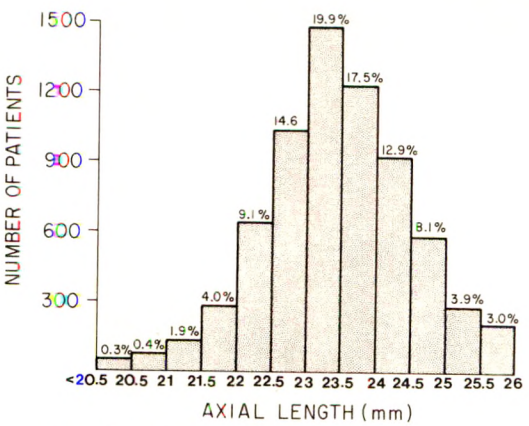


Fig. 2 (Hoffer). Axial length distribution.

count a flattening of the cornea after cataract surgery based on Floyd's[6] report of 42 eyes. Binkhorst used 1.3333 rather than the accepted 1.3375 as the refractive index of the cornea. Phakic eyes had an average keratometric reading of 43.81 diopters (±1.60), aphakic eyes, 43.65 diopters (±1.60), and pseudophakic eyes, 43.78 diopters (±1.60) (Fig. 3). Patients with aphakia do show a statistically significant flattening (P<.006) but it amounts to only 0.16 diopter. A comparison of the phakic eyes to the combined aphakic and pseudophakic groups is less significant (P<.01) and the flattening amounts to only 0.13 diopter. Most importantly, there is no significant flattening of the cornea in pseudophakic eyes compared to phakic eyes. These data indicate that a definite, but minor, flattening of the cornea occurs in aphakic eyes, but further studies are needed to document a more significant flattening in pseudophakic eyes after surgery.

Figure 4 shows the distribution of anterior chamber depth for the entire series. The mean anterior chamber depth in the phakic eyes was 3.24 mm (±0.44). The depth in aphakic eyes, as expected, was significantly greater (P<.002) at 3.67 mm (±0.48), as was the depth

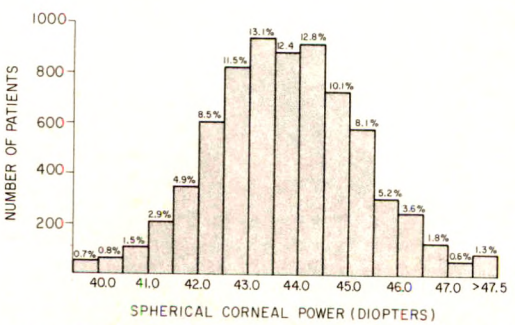


Fig. 3 (Hoffer). Average keratometric distribution for the entire series.

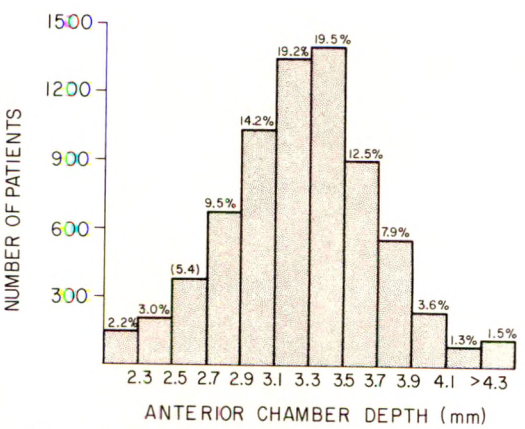


Fig. 4 (Hoffer). Anterior chamber depth distribution.

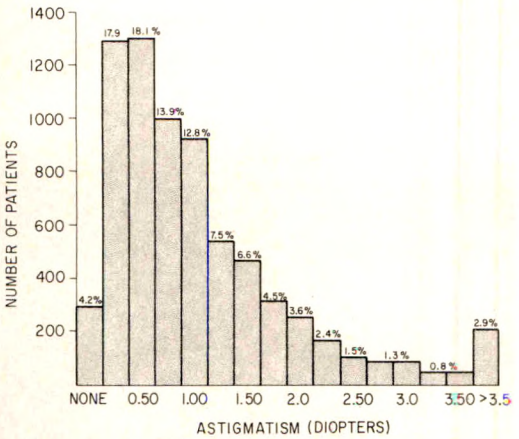


Fig. 5 (Hoffer). Astigmatism distribution.

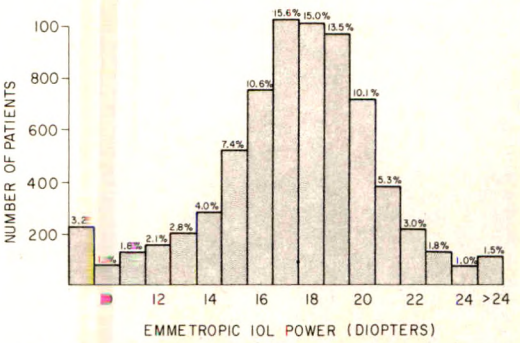


Fig. 6 (Hoffer). Distribution of prepupillary intraocular lens powers for emmetropia.

in pseudophakic eyes, 3.32 mm (±0.43) rather than the 3.50 mm usually quoted. I did not, however, define lens type in analyzing the pseudophakic eyes.

Figure 5 shows the distribution of corneal astigmatism in all 7,500 eyes. The mean astigmatism of the phakic eyes was 1.0 diopter (±1.0). The aphakic group showed an increase in astigmatism that was statistically significant ($P<.002$) but amounted to only 0.5 diopter. There was a significant increase in astigmatism in pseudophakic compared to aphakic eyes, but it amounted to only 0.13 diopter. These data do not substantiate the expected increase in astigmatism in aphakic and pseudophakic eyes.

The mean intraocular lens power, using my data and Collenbrander's[7] formula, was 17.0 diopters (±4.5). Figure 6 shows the bell-shaped curve distribution of powers for emmetropia. Of the lens powers that can be used clinically (eliminating powers under 14.0 diopters), 64% are between 17.0 and 20.0 diopters, 22% are less than 17.0 diopters, and 14% over 20.0 diopters (Fig. 7).

I performed random endothelial cell counts on 1,021 of the 6,950 phakic eyes with a specular microscope (Heyer-Schulz HS-CEM-3). The mean phakic central count was 2,538 cells/mm^2 (±503), which is in agreement with previous studies.[8] Figure 8 shows this distribution. There is no statistical significance in the

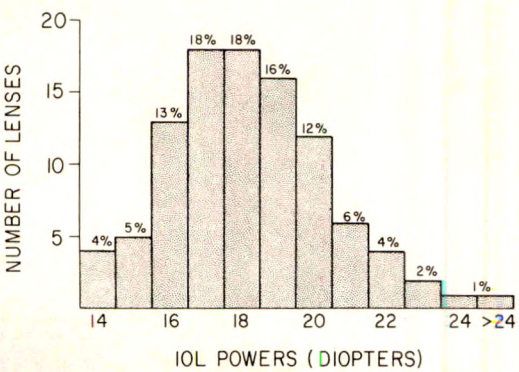


Fig. 7 (Hoffer). Percentages of prepupillary lens powers for clinical use.

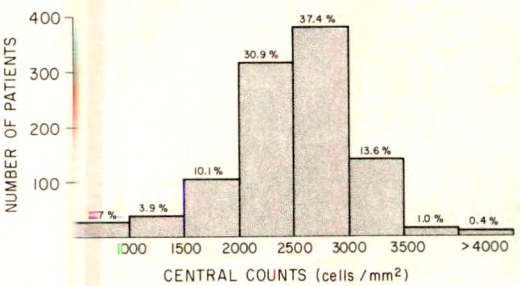


Fig. 8 (Hoffer). Central endothelial cell count distribution (cells/mm^2).

mean postoperative cell counts of aphakic (1,792 cells/mm^2 [±680]) and pseudophakic (1,737 cells/mm^2 [±767]) eyes (Table 1).

It is possible to compare other biometric variables for eyes defined as having axial myopia, emmetropia, or hyperopia (Table 2). I defined axial emmetropia as 23.65 mm with a tolerance of ±0.75 diopter, that is, 23.4 to 23.9 mm. I defined axial myopia as more than 23.9 mm and axial hyperopia as less than 23.4 mm. Of this series, 47% had hyperopia, 33% myopia, and 20% emmetropia. Because of cataract formation, correlation with refractive error was not possible. Age analyses (Table 2) showed that the group with hyperopia was older than the group with emmetropia by one year, while the group with emmetropia was older than the group with myopia by two years. Both figures are significant to P<.002. Myopic eyes may be prone to cataract formation at an earlier age.

Anterior chamber depth, based on axial length analysis, showed that shorter eyes have shallower anterior chambers, 3.11 mm (±0.45), and that longer eyes have deeper anterior chambers, 3.46 mm (±0.41). Emmetropic eyes were halfway between the other two groups, 3.28 mm (±0.42). These differences were significant to P<.002. It is disturbing that the range of anterior chamber depths based on axial length is small. It can be seen from Table 2 that the minimum depth is less in hyperopia (1.0 mm) but equal in emmetropia and myopia (2.0 mm), while

TABLE 2

ANALYSIS BASED ON AXIAL LENGTH

Measure	Eyes		
	Hyperopic	Emmetropic	Myopic
Axial length by ultrasound (mm)	<23.40	23.40-23.90	>23.90
No.	3,600	1,400	2,500
%	47	20	33
Age in years			
Mean	73	72	70
S.D.	±10	±10	±10
Anterior chamber depth (mm)			
Mean	3.11	3.28	3.46
S.D.	±0.45	±0.42	±0.41
Range	1.0-6.0	2.0-6.0	2.0-7.0
Average keratometric reading (diopters)			
Mean	44.43	43.58	43.13
S.D.	±1.56	±1.23	±1.52
Astigmatism (diopters)			
Mean	1.00	0.92	1.00
S.D.	±1.06	±0.88	±1.00
Range	9.50	6.30	9.50
Emmetropic IOL power (diopters)*			
Mean	19.00	17.00	14.50
S.D.	±2.7	±2.0	±5.6
Central endothelial cell count (cells/mm^2)			
Mean	2,423	2,485	2,525
S.D.	±627	±482	±530

*Prepupillary style.

the maximum is higher in myopia (7.0 mm) but equal in emmetropia and hyperopia (6.0 mm). This does not provide great security in using refractive error (for example, axial length) to estimate anterior chamber depth, leaving it a rather unpredictable variable.

Analysis of the average keratometric values for these groups demonstrates the so-called emmetropizing relationship between axial length and keratometric reading (Table 2). Hyperopic eyes had stronger or steeper corneas (44.43 diopters [±1.56]) than emmetropic eyes (43.58 diopters [±1.23]), a difference of 0.85 diopter. It is interesting to note that the difference in mean keratometric values between emmetopic and hyperopic eyes is twice as much as that between emmetropic and myopic eyes (0.45 diopters).

It is obvious that there is an inverse relationship between axial length and emmetropic prepupillary intraocular lens power. Patients with hyperopia required, on the average, 19.0-diopter (±2.7) lenses to achieve emmetropia; patients with emmetropia required 17.0-diopter (±2.0) lenses; those with myopia required 14.5-diopter (±5.6) lenses. The standard deviation is significantly larger in the group with myopia, indicating a greater potential for error when using clinical history to assess myopic eyes. Some individuals with myopia, for example, required 20-diopter lenses.

An analysis, based on axial length, of astigmatism (Table 2) shows that emmetropic eyes (0.92 diopter [±0.88]) have less mean astigmatism than myopic (1.0 diopters [±1.0]) or hyperopic (1.0 diopter [1.06]) eyes, a difference of 0.08 diopter ($P<.005$). Hyperopic eyes were equal to myopic eyes in regard to astigmatism. The maximum range of astigmatism was 6.3 diopters for emmetropic eyes and 9.5 diopters for both myopic and hyperopic eyes.

I also analyzed endothelial cell counts on the basis of axial length. There is a direct relationship between axial length and central endothelial cell counts. Hyperopic eyes with the the shortest axial lengths had the lowest mean cell count (2,423 cells/mm^2 [±627]) compared to emmetropic eyes which demonstrated a mean of 2,485 cells/mm^2 (±482). This is significant to $P<.002$, even though the difference amounted to only 62 cells/mm^2 (2.5%). The emmetropic mean count was lower than that seen in myopic eyes (2,525 cells/mm^2 [±530]) by 40 cells/mm^2 (1.6% less). The myopic mean cell count was 6.5% higher than the hyperopic mean count. These correlations were all significant to $P<.002$.

I compared the biometric values of both eyes for 1,800 randomly selected patients in this series (Table 3) to determine the need to measure the axial length of the fellow eye in order to

TABLE 3

DIFFERENCES BETWEEN FELLOW EYES*

Measure	Mean Difference	S.D.	Range
Axial length by ultrasound (mm)	0.34	±0.70	0-11.02
Average keratometric value (diopters)	0.87	±0.83	0-9.65
Anterior chamber depth (mm)	0.23	±0.27	0-2.94

*No. =3,600 (1,800 patients).

calculate intraocular lens power for one eye and to determine the consistency of these measurements between fellow eyes. The mean of the difference in axial length between fellow eyes was 0.34 mm, which is very similar to the 0.42 mm found by Yu and associates.[4] However, the standard deviation of 0.7 mm indicates that there is no predictable trend in axial-length symmetry between fellow eyes. The minimum difference was 0 mm and the maximum difference was 11.02 mm.

Comparison of the average keratometric values for fellow eyes disclosed a mean difference of 0.87 diopter (±0.83). This indicates that 66% of the patients showed a difference in average keratometric values of 0 to 1.7 diopters, while 34% showed a greater difference. The minimum difference was 0 diopters, while the maximum was 8.65 diopters. The anterior chamber differences revealed an average of 0.23 mm (±0.27), with a minimum of 0 mm and a maximum of 2.94 mm. Sixty-six percent showed anterior chamber depth differences of 0 to 0.5 mm.

There was no central tendency or predictability in any of the variables of axial length, average keratometric value, or anterior chamber depth between fellow eyes of these 1,800 patients. Therefore, it is imperative to obtain measurements of both eyes when selecting intraocular lens power for the first eye.

It should be noted that analysis of the data showed no statistically significant relationship between steepness of the cornea (average keratometric value) and astigmatism, anterior chamber depth, central endothelial cell count, or age, or between age and astigmatism or anterior chamber depth.

Analysis of the graphic distribution of the data for each factor is summarized in Table 4.

TABLE 4

SUMMARY OF CLINICAL FINDINGS

Clinical Finding	% of Patients*
Age ≥ 70 years	58
Anterior chamber depth (mm)	
<3.3	54
3.5	32
>3.7	14
Keratometric value (diopters)	
±2.5 of mean	90
>2.50 more hyperopic than the mean	3
Emmetropic	12
Astigmatism (diopters)	
None	4
<0.5	40
0.5 to 2.0	50
>2.0	10
>3.5	3
Endothelial cell count (cells/mm^2)	
<2,000	17
2,000 to 3,000	68
>3,000	15

*Percentages are rounded off to nearest whole number.

DISCUSSION

In my use of oculometry for intraocular lens power calculations, I had assumed that the "emmetropic" axial length was 23.5 mm, the "emmetropic" average keratometric value was 43.5 mm, and the normal anterior chamber depth was 3.5 mm. Since the axial length, the average keratometric value, and the position and power of the crystalline lens interdependently produce the refractive status of the eye, how does one define an emmetropic axial length or average keratometric value? Is it one found in an emmetropic eye, one defined by schematic eye formulation, or the mean of the measurements on a large series of eyes such as this study provides? We do not have the answers to these questions.

It must be kept in mind that measuring axial length by ultrasound does not necessarily measure the true axial length, but is probably more accurate than histological measurements.

This study shows that several accepted values should be changed. The preoperative anterior chamber depth is 3.24 mm rather than 3.5 mm. There is no significant change in axial length or corneal curvation after cataract surgery. Formulas based on other assumptions should be reevaluated. Mean astigmatism changes little after cataract surgery. We found little difference in the postoperative endothelial cell counts of cataract patients with and without intraocular lenses. The aphakic eyes showed a 29% and pseudophakic eyes a 32% mean cell loss compared to the phakic eyes. The mean emmetropia-producing prepupillary intraocular lens power was 17.0 diopters rather than 18.0 diopters as is commonly held.

These data can be used to restructure the Gulstrand model of the eye and lead to more accurate theoretic formulae for intraocular lens power and other important calculations.

Summary

This biometric analysis of 7,500 eyes of cataract patients gave a mean axial length of 23.65 mm, a mean average keratometric value of 43.81 diopters, a mean preoperative anterior chamber depth of 3.24 mm, and a mean central endothelial cell count of 2,470 cells/mm^2. There is a statistically significant but clinically insignificant 0.16-diopter flattening of the cornea after cataract surgery without an intraocular lens, but none with an intraocular lens. Pseudophakic eyes do not show a clinically significant increase in corneal flattening over aphakic eyes. Anterior chamber depth increases from 3.24 mm to 3.32 mm (±0.08) in pseudophakic eyes and to 3.67 mm (±0.43) in aphakic eyes. Astigmatism averaged 1.0 diopter in phakic eyes preoperatively, showing a mean increase of only 0.5 diopter in aphakic eyes and 0.65 diopter in pseudophakic eyes.

One third of the eyes in this series had axial myopia while slightly less than half had axial hyperopia; the remaining 20% were in the emmetropic range. Preoperative anterior chamber depths were lowest in eyes with short axial lengths and increased with axial length. However, deep (5 mm) and shallow (2 mm) anterior chamber depths were encountered in all three groups. Shorter eyes had steeper corneas than emmetropic eyes by less than 1.0 diopter while myopic eyes had weaker corneas than emmetropic eyes by about 0.5 diopter. Astigmatism was essentially the same in all three groups (1.0 diopter) except that emmetropic eyes showed a small (0.08 diopter) but statistically significant decrease in cylinder from the other groups, as well as a smaller range (6.3 vs. 9.5 diopters). There is a very small but extremely significant increase in endothelial cell count from hyperopic to myopic eyes. There is little correlation between fellow eyes for axial length, average keratometric value, or anterior chamber depth, indicating the need for bilateral examination in calculating intraocular lens power. All other possible correlations of these data were not statistically significant.

Acknowledgement

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OPHTHALMIC MINIATURE

Great pains should be taken over the paper's summary, which should make use of the whole of the journal's ration of space (one-fifth or one-sixth of the length of the text, as the case may be), and its composition is the severest test of an author's literary skill, particularly in days when "précis writing" has been dropped from the syllabus in most schools for fear of stifling the scholars' creative afflatus. The writing of a summary tests the author's powers of apprehension and sense of proportion—the feeling for what is really important and what can be left out. A summary must be complete in its own limits. It may well start with a statement of the hypothesis under investigation and end with its evaluation. Nothing is more abjectly feeble than to write some such sentence as "The relevance of these findings to the etiology of Bright's disease is discussed." If it has been discussed, the discussion should be summarized, too. If not, say nothing. The preparation of abstracts is a public service a young scientist should sometimes volunteer to do. Even if his work is overseen by an experienced editor before it goes to press, abstracting can be good practice in writing.

P. B. Medawar, *Advice to a Young Scientist*
New York, Harper and Row, 1979

AUTOMATED CLINICAL VISUAL ACUITY TESTING

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The ability to read a letter chart is the most widely accepted measure of the visual effects of ocular disease and its treatment. Unfortunately, variations in daily testing procedure and restrictions in the number of letters available on conventional test charts limit the accuracy of clinical measurements and, therefore, the confidence which an ophthalmologist can have in changes recorded in visual acuity from one office visit to the next. Variation in daily testing procedure can be eliminated by automating the visual acuity testing procedure as has been done with projected visual acuity targets and early minicomputers.[1] Limitations in the number of letters available of a given size can be avoided by using a device such as the BVAT (Mentor) in which a microprocessor generates visual acuity targets on a television monitor. Both of these problems are readily eliminated by using an inexpensive, general-purpose microcomputer to display visual acuity targets on a television monitor, control a staircase testing procedure, and provide a printed record of mean visual acuity and its standard deviation.

MATERIAL AND METHODS

A general-purpose microcomputer (Apple II) presents visual acuity targets (illiterate E) on a television monitor placed either 10 or 20 feet from the patient. The computer also monitors the responses the patient enters through the small hand-held response box shown in Figure 1. The visual acuity target is displayed continuously until the patient responds by pressing a button on the response box. The response box has four large buttons, each corresponding to one of the four directions in which the target E is displayed randomly on the television monitor (up, down, left, or right). Press-

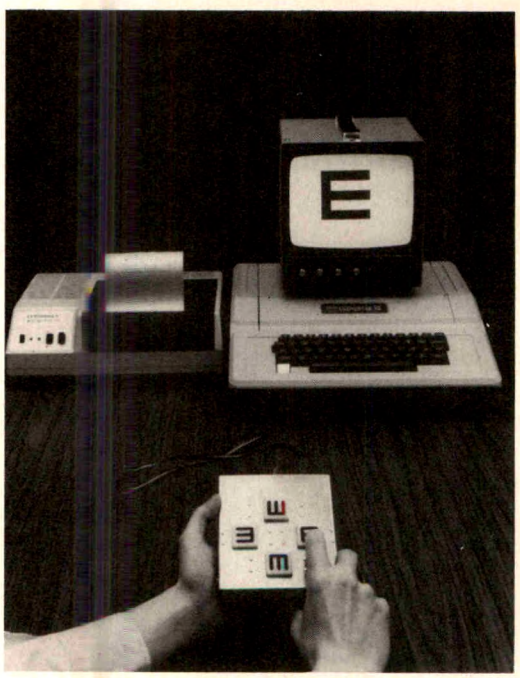


Fig. 1 (Timberlake, Mainster, and Schepens). Equipment required for microcomputer-automated visual acuity testing. In addition to a general-purpose microcomputer, monitor, and printer, a hand-held response box is used by the patient to enter responses to the visual acuity targets displayed on the television screen.

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ing the correct button generates a short, high-frequency tone. Pressing the incorrect button generates a slightly longer, low-frequency tone. The audible tones provide feedback concerning the correctness of the patient's response and produce greater stability in the patient's criterion for deciding which button to press.[2] After each response by the patient, the television screen blanks and then a new visual acuity target appears.

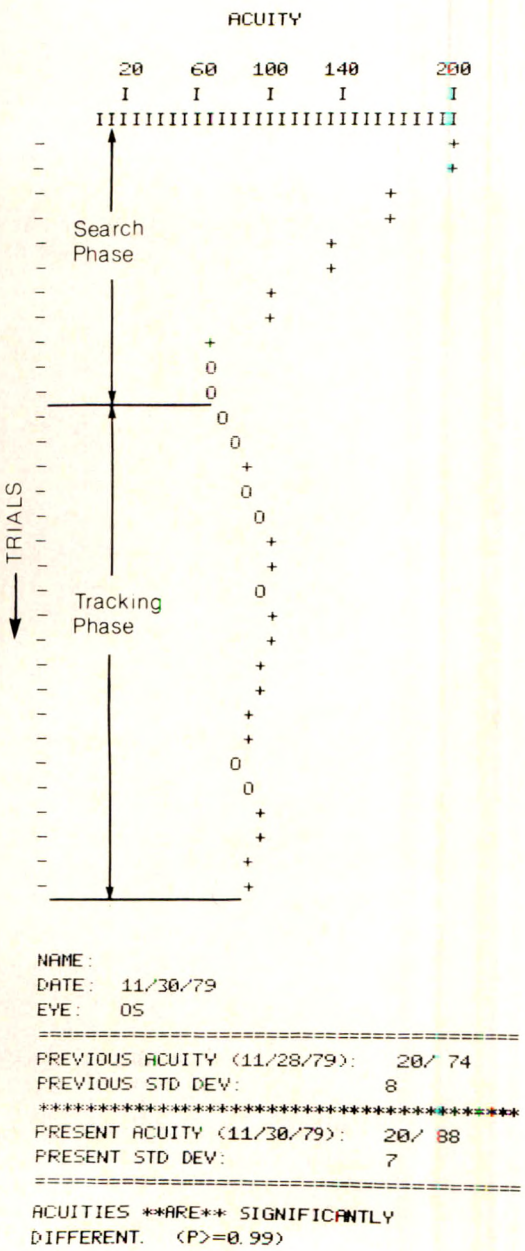
The computer also controls a four-option, forced-choice, staircase procedure.[2] This psychophysical method was selected because of its efficiency and accuracy.[3] The procedure consists of two phases: (1) a brief "search" phase which begins with a large target (6/120 or 6/60 [20/400 or 20/200]) and quickly reduces target size to determine a patient's approximate visual acuity; and (2) a lengthier "tracking" phase which begins after the first two successive incorrect responses and determines the patient's mean visual acuity and its standard deviation. The algorithm used in the search phase is the following: a single correct or incorrect response does not cause a change in target size; two successive correct responses reduce letter segment size by five units; two successive incorrect responses increase segment size by one unit and initiate the tracking phase. One unit is 0.33 minutes of visual angle at 20 feet. The orientation of successive targets is selected randomly by the computer. The algorithm used in the tracking phase is the following: a single correct response does not change target size; a single incorrect response increases letter segment size by one unit; two successive correct responses decrease target size by one unit. Once initiated, the tracking phase consists of a series of 20 randomly oriented targets whose size is selected by the preceding algorithm. The targets used in the automated test procedure range from 6/60 to 6/2.1 (20/200 to 20/7) for a 20-foot test distance or from 6/120 to 6/4.2 (20/400 to 20/14) for a ten-foot test distance. At the conclusion of the test, the computer prints a complete record of the patient's responses and the mean visual acuity and standard deviation computed from the 20 trials of the tracking phase (Fig. 2). Performance of the test requires three to four minutes for each eye. Personnel are needed only to give standard, 20-second instructions to the patient, to enter data for each patient (20 seconds), and to start the test (five seconds).

We tested ten consecutive patients being examined for retinal disease with the computerized procedure and with both standard Snellen and illiterate E test charts. The charts were illuminated from the front by fluorescent lights. The background luminance of the test charts was 107 mL and letter contrast was 93%. The television monitor background luminance and letter contrast were also set at 107 mL and 93%, respectively. Visual acuity target background luminance from ambient room illumination was less than 8 mL, in accordance with the recommendation of the National Academy of Sciences-National Research Council Committee on Vision that surround luminance not exceed one-half of chart luminance.[4]

The patients tested ranged in age from 7 to 74 years (Table). We tested both eyes of each patient, first with the visual acuity charts and then with the automated procedure. The television monitor was located next to the test charts, but only the visual acuity target in use was illuminated. All testing was conducted at 20 feet (6.1 m).

Results

Since there was excellent agreement between Snellen and E chart visual acui-



NAME:
DATE: 11/30/79
EYE: OS

PREVIOUS ACUITY (11/28/79): 20/ 74
PREVIOUS STD DEV: 8

PRESENT ACUITY (11/30/79): 20/ 88
PRESENT STD DEV: 7

ACUITIES **ARE** SIGNIFICANTLY DIFFERENT. (P>=0.99)

Fig. 2 (Timberlake, Mainster, and Schepens), Record of a patient tested 24 hours after photocoagulation treatment (name is blanked out). The record is printed automatically at the end of the test. The graph at the top of the record shows the responses that the patient made to each visual acuity target presentation. A plus sign indicates that the patient correctly identified the letter orientation; a zero indicates an incorrect response. Mean visual acuity and standard deviation are printed at the bottom of the record along with the results of a Student's *t*-test performed automatically at the end of the test. The *t*-test shows that there is a 99% probability that the patient's decline in visual acuity from 11/28/79 to 11/30/79 is significant. Previous mean visual acuity and standard deviation are entered from the patient's medical record (chart or computer file) at the beginning of the test along with other relevant data.

ties, only the more directly comparable E chart results appear in the Table for comparison with the results of computerized testing. The Table shows good correlation between automated and E chart visual acuities, but it also demonstrates that the 30 target sizes used in the computerized method permit a more precise determination of visual acuity than that obtainable with the eight target sizes on the E chart. This is reflected in the better visual acuities found in auto-

TABLE

VISUAL ACUITIES DETERMINED BY E CHART TESTING AND BY MICROCOMPUTER-CONTROLLED AUTOMATED TESTING FOR TEN CONSECUTIVE PATIENTS BEING EXAMINED FOR RETINAL DISEASE

Patient No.	Right Eye			Left Eye		
	E Chart	Computer, Mean±SD		E Chart	Computer, Mean±SD	
1	6/6 (20/20)	6/6.9±3.6	(20/23±12)	6/60 (20/200)	6/37.6±2.3	(20/125±8)
2	6/9 (20/30)	6/8.0±1.9	(20/27±6)	6/6 (20/20)	6/6.3±1.6	(20/21±5)
3	6/30 (20/100)	6/34.3±4.6	(20/114±15)	6/30 (20/100)	6/25.6±2.6	(20/85±9)
4	6/9 (20/30)	6/6.9±1.9	(20/23±6)	6/15 (20/50)	6/8.3±1.3	(20/28±4)
5	6/15 (20/50)	6/6.9±2.0	(20/23±7)	6/15 (20/50)	6/12.6±2.6	(20/42±9)
6	6/6 (20/20)	6/5.0±1.3	(20/17±4)	6/9 (20/30)	6/9.3±1.3	(20/31±4)
7	6/21 (20/70)	6/26.6±3.0	(20/89±10)	6/15 (20/50)	6/13.6±1.6	(20/45±5)
8	6/15 (20/50)	6/13.9±1.3	(20/46±4)	6/6 (20/20)	6/7.9±1.6	(20/26±5)
9	6/30 (20/100)	6/19.6±3.0	(20/65±10)	6/30 (20/100)	6/24.9±1.6	(20/83±5)
10	6/30 (20/100)	6/38.6±3.6	(20/129±12)	6/9 (20/30)	6/6.9±1.3	(20/23±4)

mated testing for 13 of the 20 eyes (65%) in the Table. The better visual acuities were presumably missed in E chart testing because of larger increments between adjacent letter sizes.

The Table also shows the standard deviations of the computerized visual acuity measurements that are computed automatically at the conclusion of a test and printed on the patient's record (Fig. 2). The standard deviation is an indication of the variability of a particular visual acuity measurement, and it has the same units as the denominator of the Snellen fraction (meters or feet). Statistically, there is a 68% chance that the "true" visual acuity lies within ± 1 SD of the mean visual acuity. Thus, for the right eye of Patient 5, there is a 68% chance that "true" visual acuity lies between 6/5 and 6/9 (20/16 and 20/30), that is, between 6/6.9−2 (20/23−7) and 6/6.9+2 (20/23+7). This uncertainty about "true" visual acuity is present regardless of whether visual acuity is measured automatically or with a chart. The advantage of the computerized method is that the standard deviation of a visual acuity measurement is determined automatically, and since the uncertainty of the measurement is thus known, it is possible to determine if changes in visual acuity from one office visit to the next are statistically significant.

In addition to computing mean visual acuity and standard deviation for each test, the computer also uses the data for each patient entered at the beginning of the test (personal data, as desired; and previous mean visual acuity and standard deviation, if available) and Student's *t*-test to determine automatically if a change in visual acuity is statistically significant. For example, we tested the patient whose record is given in Figure 2 before photocoagulation treatment and on the day after. Before photocoagulation, his visual acuity was 6/22.3 (SD±2.3) (20/74 [SD±8]). On the day after photocoagulation treatment his mean visual acuity was 6/26.3 (SD±2.0) (20/88 [SD±7]). While this change in visual acuity was small, computer analysis performed automatically at the time of

the second test demonstrated a 99% probability that the measured difference was significant (t =5.75, df =38).

Discussion

While more sophisticated tests of visual function have been developed, the ability to read a letter chart remains the most widely used measure of the effects of disease on visual performance. Unfortunately, this is a statistical measurement, difficult to perform reliably under clinical conditions because chart testing (1) limits the number of letter sizes and therefore the precision of the test, (2) severely limits the number of letters of a given size and therefore precludes statistical sampling, and (3) is influenced by a variety of additional factors such as the examiner's instructions, speed, and even attitude. These considerations limit the confidence an ophthalmologist can have in variations in a patient's visual acuity from one office visit to the next, but these difficulties can be overcome with this automated testing procedure.

The cost of the additional information is the expense of the microcomputer and the additional time required for the computer to perform the test. Since inexpensive microcomputers have become available in the last five years,[5] the cost of using automated testing in the small clinic is no longer prohibitively high. Since office staff are required only to instruct the patient briefly at the beginning of the test and to begin the test, the additional burden on paramedical personnel is limited. The fundamental advantage of automated testing is that the clinician can know immediately if variations in visual acuity represent statistically significant changes in visual performance and can therefore accurately follow the visual course of a patient's ocular disease and therapy.

Summary

We developed a method for testing visual acuity using a general-purpose microcomputer which displays visual acuity targets on a television monitor, controls a staircase psychophysical testing procedure, and provides a printed record of mean visual acuity and standard deviation. This automated procedure can be used to increase the precision of clinical visual acuity testing and to determine if changes in a patient's visual acuity are statistically significant.

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OSTEOGENIC SARCOMA OF THE HUMERUS AFTER COBALT PLAQUE TREATMENT FOR RETINOBLASTOMA

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Osteogenic sarcoma is common in patients who have been previously treated for bilateral retinoblastoma.[1-7] Of patients who survive bilateral retinoblastoma, 15 to 20% develop second, nonocular, tumors from one to 42 years after the successful treatment of their ocular tumors. These second tumors are fatal in 85% of patients; thus, more patients with bilateral retinoblastoma now die of the nonocular tumor than succumb to retinoblastoma itself. Of these patients, 71% develop tumors in the field of radiation; the most common of these tumors is osteogenic sarcoma. Osteogenic sarcoma of the skull is 2,000 times more frequent in these patients than in the rest of the population.[8]

Although the mean latent period from irradiation of bilateral retinoblastoma to the development of second tumors is 11.4 years, we have seen such tumors within two years in the field of radiation with doses as low as 3,500 rads.[8]

Of patients who developed tumors after radiation therapy for bilateral retinoblastoma, 19% developed tumors at sites distant from the field of radiation; the average latent period was 11.1 years. Such tumors have been seen within one year of radiation treatment with doses as low as 3,500 rads. Again, osteogenic sarcoma is the most common of these tumors and even in sites more remote from the radiation beam it occurs 500 times more frequently than in the rest of the population.[8]

Second, nonocular, tumors also occur in patients who have been treated for bilateral retinoblastoma without irradiation.[8,9] In our series of 22 survivors of bilateral retinoblastoma treated with bilateral enucleation without any radiation, three (14%) have developed second, nonocular, tumors. One of these tumors was rhabdomyosarcoma of the temple. Another patient developed an osteogenic sarcoma of the femur ten years after the retinoblastoma.

We now report on one case of a patient who developed an osteogenic sarcoma of the humerus after treatment with enucleation of one eye and a cobalt plaque to the other.

CASE REPORT

We examined a 2 11/12-year-old boy with esotropia of four months' duration and diagnosed bilateral retinoblastoma. There was no family history of retinoblastoma.

We enucleated the left eye and confirmed the diagnosis of retinoblastoma pathologically. There was no extention of tumor in the optic nerve or choroid (Fig. 1).

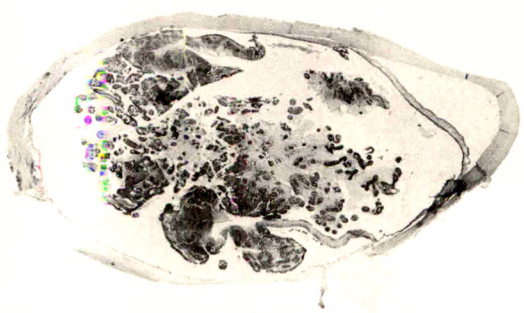


Fig. 1 (Abramson, Ellsworth, and Kitchin). Microscopic section of enucleated eye (hematoxylin and eosin, ×3).

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Examination of the right eye while the boy was under anesthesia disclosed a large solitary tumor at the ora serrata centered at 2 o'clock. It measured 7.5 mm in diameter and was elevated 5 to 6 mm. The lesion extended to an ora tooth at 2 o'clock. While the boy was under general anesthesia, we attached a 10-mm round cobalt plaque to the sclera after localizing the tumor with diathermy. The plaque was removed six days later, at which time a dose of 4,000 rads had been delivered at 6 mm.

One month after the plaque was removed, an examination disclosed that the tumor was smaller. Six months later the tumor measured 3 to 4 mm across and only 1 mm in elevation with calcification in the tumor.

We applied transconjunctival cryopexy to the tumor remnant 3½ years after insertion of the plaque.

Six years after the insertion of the plaque, the tumor was completely gone and had been replaced by an extensive chorioretinal scar with calcification.

Nine years and seven months after the insertion of the plaque there was heavily calcified tumor that had not changed. The optic disk and macula were normal and his vision was normal.

Three months later he complained of pain in the upper right extremity. The pathologic diagnosis was ostegenic sarcoma of the right upper humerus (Fig. 2). The humerus was resected and replaced with a Silastic humeral prosthesis. The patient began chemotherapy with doxorubicin, methotrexate and citrovorium factor. Within three months we noted multiple pulmonary metastases.

Discussion

The association between bilateral retinoblastoma and the development of second, nonocular, tumors has been emphasized in recent years.[1-9] We now recognize that, despite unilateral retinoblastoma being more common than bilateral retinoblastoma, the second tumors that appear in survivors of bilateral retinoblastoma occur whether or not they have been treated with external beam irradiation, no irradiation, or, as in our case, a local radioactive applicator. Of all patients who survive any treatment for bilateral retinoblastoma, 15 to 20% will develop these second tumors at a mean of ten to 11 years after treatment of retinoblastoma. Despite the above, the exact role of the radiation in inducing these tumors is not clear.

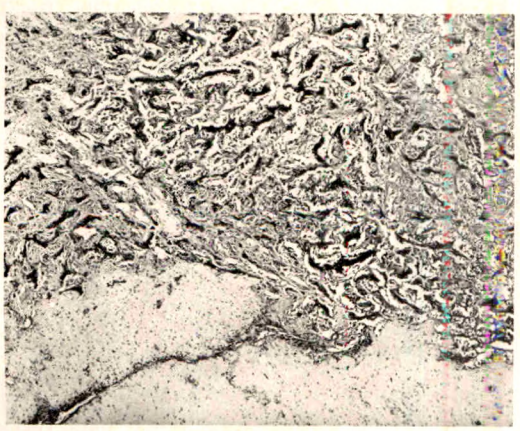


Fig. 2 (Abramson, Ellsworth, and Kitchin). Microscopic section of osteogenic sarcoma of the humerus (hematoxylin and eosin, ×100.

The argument implicating the radiation in the genesis of these tumors is as follows: The majority of second tumors occur in the field of the radiation portal (71%) following a long latent period (11 years) and frequently after high doses of radiation (15,000 to 20,000 rads). The most common type of tumor in the field of irradiation is a sarcoma, which we know occurs as a result of irradiation.

The argument suggesting that radiation plays little if any role in the genesis of these tumors is as follows: The second tumor clearly occurs in the patients with bilateral retinoblastoma who harbor the germinal mutation. They have not occurred in patients with unilateral retinoblastoma treated with irradiation.

The second tumors in the field of radiation may develop after short latent periods one year from radiation after low doses of radiation (3,500 rads) or even with no radiation.

Second tumors occur at sites distant from the radiation portal after high doses, low doses, or no irradiation. Although the mean latent period for the development of tumor at distant sites is similiar to that of tumors in the field of irradiation, some tumors at distant sites are seen within a year or two of radiation. It would be difficult to implicate radiation in some

patients, such as those with Wilms' tumor. The observation that some patients develop multiple tumors at different sites[8] emphasizes the basic cancer propensity of these patients. We have been struck by the occurrence of osteogenic sarcoma in family members who appeared to harbor the gene for retinoblastoma but who did not develop the disease.

Summary

A 13-year-old boy developed an osteogenic sarcoma of the right humerus ten years after treatment for bilateral retinoblastoma. The bilateral retinoblastoma had been managed with enucleation of one eye and successful treatment of the solitary tumor in the other eye with a single application of a local 10-mm round cobalt plaque.

Patients with bilateral retinoblastoma have a significant (15 to 20%) chance of developing a second, nonocular, neoplasm. These neoplasms have occurred from one to 42 years after the treatment for retinoblastoma. The most common of these tumors is an osteogenic sarcoma; it may occur after external beam irradiation, local cobalt plaque, or no irradiation. The tumors may be in the skull or at distant sites whether or not these patients receive radiation.

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TARSAL BUCKLING AFTER SUTURELESS FASANELLA-SERVAT PROCEDURES

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The Fasanella-Servat procedure, although popular,[1] can have a number of complications. The procedure causes loss of tissue containing the basic secretors of tears and the goblet cells required for mucin production.[2] One other common problem is postoperative corneal abrasion from the tarsal suture.[3-5]

Marcovits[6] described a sutureless Fasanella-Servat procedure in which an immediate postoperative wound dehiscence, which caused worsening of the blepharoptosis, was remedied by suturing the tarsal wound. We merely crushed and excised the tarsus with good results in approximately 25 patients. Recently, however, we experienced a complication that has not previously been reported to the best of our knowledge, and which may be unique to this procedure. Dramatic tarsal buckling occurred in both eyes of a patient, apparently after dehiscence of a crushed and sutureless tarsal wound.

CASE REPORT

A 62-year-old healthy woman underwent an uncomplicated sutureless Fasanella-Servat procedure on the left eye. Ten days postoperatively the eyelid was grossly distorted by what appeared to be a large chalazion. After six weeks without improvement, an incision disclosed a large nodule of scar tissue located at the superior tarsal border. Local excision could not change the contour and a full-thickness block resection of the upper eyelid was necessary for successful repair. The same patient then had a sutureless Fasanella-Servat procedure performed on her right eye. Three days postoperatively the appearance of the eyelid was excellent. Ten days after surgery, however, the eyelid was severely distorted (Figs. 1 and 2). Eversion of the eyelid disclosed a buckled tarsus (Fig. 3). After four weeks of observation, we performed a surgical revision. We found that the tarsal-conjunctival border had been replaced by a dense, horizontally oriented cicatrix. The tarsus was so buckled that it could not be unfurled by incision and undermining. We resected large, twisted, triangular-shaped blocks of tarsus almost to the lash line to cause flattening. We then sutured the conjunctival border to the tarsal remnants with a pull-out nylon suture and inserted a soft contact lens. Healing was uneventful and the final eyelid position was good (Fig. 4).



Fig. 1 (Weiss and Shorr). Bilateral sutureless procedures. Ten days postoperatively, the right eyelid has fallen. The left eye shows evidence of full-thickness resection required after it developed a distorting, cicatricial nodule.



Fig. 2 (Weiss and Shorr). Right eyelid shows an S-shaped contour caused by buckling of the underlying tarsus.

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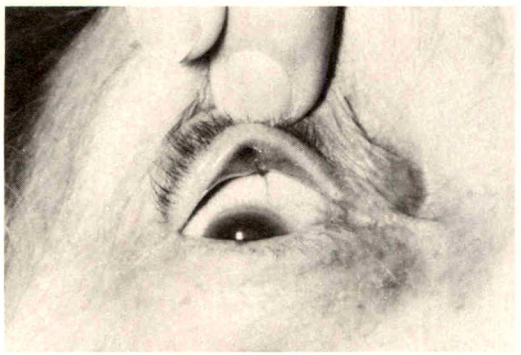


Fig. 3 (Weiss and Shorr). Eversion of right upper eyelid demonstrates a buckled tarsus; the superior border is shortened in a horizontal plane. Probably because of crushing, dehiscence, and scarring, simple incision did not unfold the tissue. Note that the amount of tarsus remaining should normally be adequate for eyelid stability.

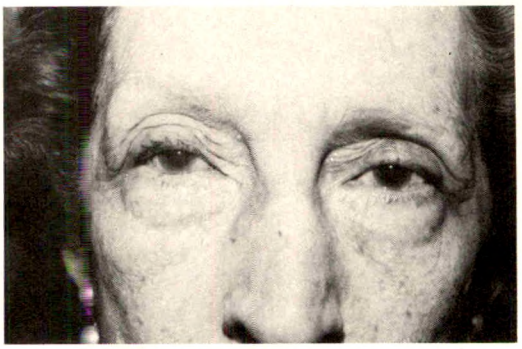


Fig. 4 (Weiss and Shorr). Final results (right eyelid) after resection of buckled tarsus, undermining of conjunctiva, and resuturing of wound.

Discussion

The Fasanella-Servat procedure is not the only blepharoptosis procedure that requires resection of the tarsus of the upper eyelid. McCord's procedure uses a combined tarsoaponeurectomy.[7] Mustardé described removal of split-level skin and orbicularis muscle anteriorly combined with a large tarsal resection posteriorly.[8] Baylis and Shorr recommended anterior tarsectomy for eyelid contour abnormalities.[9] In these cases, however, sutured tarsal incisions were used. Transmarginal rotation for upper eyelid entropion (Ballen procedure) and free tarsal grafting also leave gaping, unsutured tarsal wounds.[10,11] We have rarely encountered instances of postoperative eyelid distortion with free tarsal grafting. Any distortions are easily remedied by undermining the tarsal incision. Although the tarsus gapes as a result of all these procedures, the Ballen procedure and free tarsal grafting differ from the others in that they leave the incisions cleanly cut and uncrushed. If crushed tissue and wound separation are both present, as may happen in a sutureless Fasanella-Servat procedure, healing can lead to a circular or horizontally oriented scar. The result can be internal buckling of the tarsus with an external S-shaped eyelid. We did not recognize the cause of our patient's first eyelid deformity, but successfully treated it with a full-thickness resection of the eyelid. We treated her second eyelid with extensive undermining and internal, piecemeal tarsal resection. Despite our success, we anticipate great difficulties in reoperating on a tarsus that has been vertically constricted by a previous Fasanella-Servat procedure and is horizontally distorted by randomly directed fibers of scar tissue. Although sutureless Fasanella-Servat procedures are simple to perform, we believe that the problem of suture abrasion is better handled by use of pull-out nylon or protective soft contact lenses.

Summary

A patient who underwent bilateral, sutureless Fasanella-Servat procedures experienced wound dehiscence that resulted in a distorted eyelid contour which required substantial wound and eyelid revision. Other methods of handling the problem of suture abrasion exist that may prevent tarsal buckling after a Fasanella-Servat procedure.

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SECONDARY SYPHILIS WITH IRIS PAPULES

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Syphilis is a well-established cause of uveal disease and is thought to be responsible for several distinct types of intraocular inflammation. The less common manifestations of the disease may be transient and are easily missed. Their early recognition and confirmation by serologic testing may allow the ophthalmologist to diagnose at least one form of uveal inflammation that is amenable to standard antimicrobial therapy.

The following case is a clear example of one rarely recognized sign of secondary syphilis. We studied this patient by iris angiography, which gave us fresh insight into the pathogenesis of the lesion.

CASE REPORT

When examined in July 1978, a 30-year-old man had a nonpruritic, generalized papular rash of one day's duration. There was no involvement of his palms, soles, or mucous membranes, and no history of a chancre. Another physician made a tentative diagnosis of measles but drew blood for a VDRL test. The rash lasted only two days and the patient received no antibiotic therapy.

The patient returned three months later because of bilateral red eyes and photophobia. He denied a history of syphilis or collagen disease. He had had several episodes of acute back pain related to the lifting of heavy objects. There was no significant history of previous ocular disease and nothing sigificant in the family history.

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The young man was in no distress and had no evidence of a rash. His best corrected visual acuity was 6/6 (20/20) in both eyes. The left pupil was normal, but the right pupil reacted poorly to light and was irregular. The right eye had a ciliary flush. The corneal epithelium and stroma were clear, but there were multiple fine keratic precipitates on the endothelium. The anterior chamber was deep and showed +3 cells and +2 flare. There was a posterior synechia at the 5 o'clock position. Brown pigment was finely dispersed over the anterior lens capsule. Applanation tonometry showed a pressure of 22 mm Hg.

The left eye had a ciliary flush, clear cornea, and 12 medium-sized keratic precipitates on the endothelium. The anterior chamber was deep and showed +1 cells and flare. There were no posterior synechiae. The intraocular pressure was 20 mm Hg by applanation. There was a +1 cellular reaction in the vitreous of both eyes, and ophthalmoscopic examination showed a normal disk, macula, and vessels in both eyes.

We made a diagnosis of acute, bilateral, nongranulomatous iridocyclitis and treated the patient with topically administered prednisolone acetate 1% every two hours and scopalomine 0.25% three times a day.

The inflammation gradually resolved, and the medications were tapered off, but an exacerbation occurred after a month. At that time, the corticosteroid drops had been reduced to four a day. Frequent doses were reinstituted, and the inflammation again gradually subsided. In the course of a uveitis work-up then undertaken, an FTA-Abs test was positive and the VDRL titer was 1:32. We then obtained the previous medical records and found that on July 26, 1978, the patient had had a positive FTA-Abs test and a VDRL titer of 1:128. We now made a diagnosis of secondary syphilis with associated iridocyclitis, and on Dec. 18, 1978, began treating the patient with an intramuscular injection of 2.4 million units of penicillin G benzathine (Bicillin) which was repeated at weekly intervals for three more weeks. The topically given corticosteroid drops were continued.

Laboratory tests were negative for antinuclear antibody, latex fixation, and HLA-B27 and HLA-B8. Serum glucose, uric acid, and total protein levels were all within normal limits, as were the patient's complete blood cell count and erythrocyte sedimentation rate.

The patient had yet another recurrence of iritis in both eyes two days after his last course of penicillin G benzathine in February 1979. At that time, diffuse patches of dilated superficial vessels appeared, several on the right iris and one on the left, which looked like the published descriptions of "iris roseolae."[1] Five days later two superficial early

papules were noted in the midstroma of the right iris at the 2 and 5 o'clock positions (Fig. 1). Three weeks after their first appearance, the roseolae had disappeared from the left iris and only the papules remained on the right iris.

The patient's serum was tested for immune complex levels on March 9, and these were slightly increased. When tested by the Raji cell radio immuno assay, the level was 40 μg/ml. Normal levels for this laboratory are less than 20 μg/ml. At that time, the VDRL titer was down to 1:2.

On March 9, we also examined the right iris by angiography. In the early, arterial phase of filling, it showed leakage of fluorescein at the pupillary border in the region of the iris papules (Fig. 2). In the late, venous phase, there was diffuse leakage of fluorescein from the vessels at the pupillary margin, but even greater leakage from the 2 to 5 o'clock region of the iris where the papules were located (Fig. 3). By April 17, there was no evidence of iris papules and the eye was quiet. By May 9 the patient had been gradually tapered off all medications.

On June 7, a follow-up iris angiogram of the right eye was obtained. During the arterial phase, fluorescein again leaked from the 2 to 5 o'clock region of the iris where the papules had been (Fig. 4). Later photographs revealed generalized, profuse leakage of dye from the capillary plexus of the pupillary margin (Fig. 5).

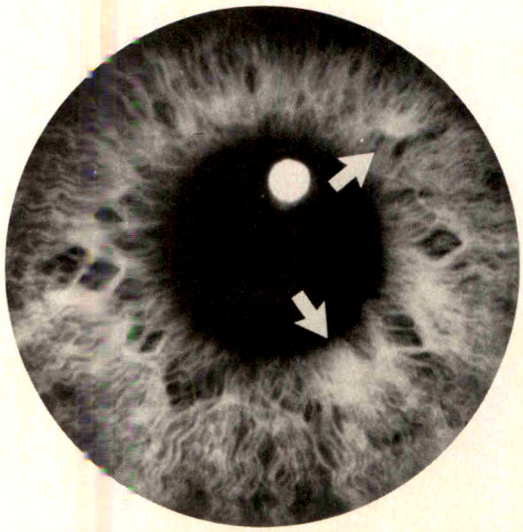


Fig. 1 (Schwartz and O'Connor). Patient with iris papules (arrows) in the right eye, two days after last course of penicillin.

Discussion

At the turn of the century, estimates of the incidence of syphilitic iritis ranged from 6 to 28%. At that time, the causes of many forms of iritis were not clear. Much later, in 1958, Schlaegel[2] estimated that 3% of uveitis cases were syphilitic, and in 1961 Perkins[3] considered a syphilitic

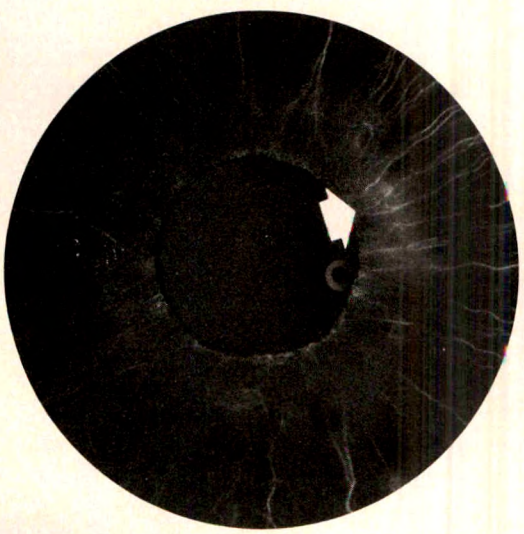


Fig. 2 (Schwartz and O'Connor). Fluorescein angiogram of iris of the right eye, March 9, 1979. Arterial phase shows leakage of fluorescein in region of iris papules (arrows).

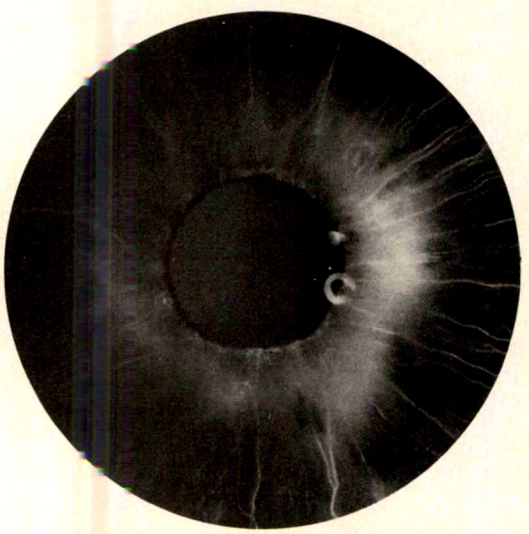


Fig. 3 (Schwartz and O'Connor). Same study as Figure 2, right eye. Late venous phase shows diffuse leakage of fluorescein from vessels of pupillary margin and from region of papules.

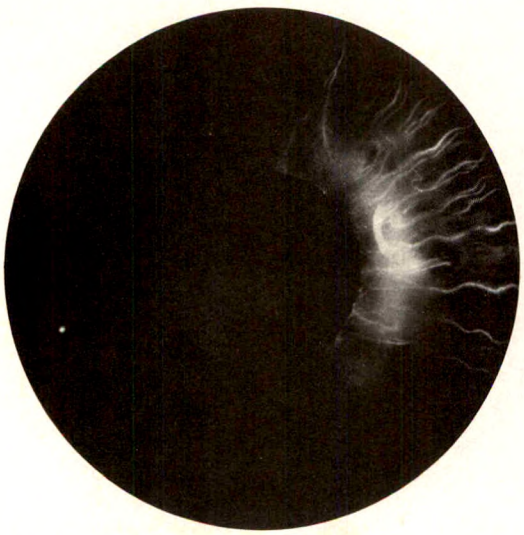


Fig. 4 (Schwartz and O'Connor). Fluorescein angiogram of right eye three months later. Arterial phase shows persisting leakage from region of papules.

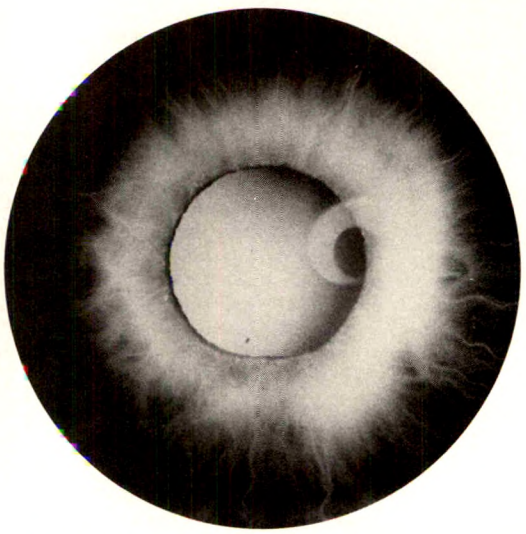


Fig. 5 (Schwartz and O'Connor). Same study as Figure 4, right eye. Late venous phase shows leakage of fluorescein from region of papules and vessels of iris sphincter.

cause in only 1% to 2% of his cases of uveitis in London.

Although the incidence of primary and secondary syphilis in the adult population has recently been increasing,[4] development of the late ocular signs of the disease is probably decreasing because of (1) better education of the public about the disease, (2) its earlier detection, and (3) more efficient antimicrobial treatment in the early stages. Meanwhile, there has been no increase in congenital syphilis,[4] probably because of the constant surveillance routinely maintained during the prenatal and postnatal periods.

According to Duke-Elder,[1] roseolae of the iris are rare but may be the first manifestations of syphilis of the eye. They may also be among the first signs of a generalized infection. In "syphilis d'emblée," which has been described by Wuepper[5] as a form of the disease that occurs without chancre, the first sign is an iridocyclitis or "red eye." There may also be minimal signs of secondary syphilis, such as a rash. Although often unnoticed by the patient, such signs may be diagnostically helpful.

In acquired syphilis, there may be four separate ocular manifestations[6]: (1) early, transient roseolae, (2) iridocyclitis, (3) papillitis, and (4) diffuse chorioretinitis. Either early or late, the inflammatory reaction may produce nodular lesions that result in highly vascularized papules in the early stage and in poorly vascularized gummata in the late stage. The latter are usually followed by necrosis.

The iritis of secondary syphilis may be of three types,[1] all of them probably variations of the same underlying process:

1. Roseolae occur in the initial stages of secondary syphilis (as early as the sixth week after the primary lesions) and may be associated with a macular skin eruption. They are essentially a hyperemic change in the already existent superficial vascular loops of the iris and produce a bright red spot of engorgement, usually

in the middle third of the iris. They are transient as a rule (lasting several days) and disappear without a trace. Late or recurrent roseolae may appear up to two years after the original infection. They form a localized patch on the iris and are frequently associated with other inflammatory signs and sequelae.

2. Papules develop from the roseolae and consist of round or pyramidal swellings whose size ranges from that of a small seed to that of a pea. Their color varies from yellow to red-brown. They occur at the pupillary or ciliary borders of the iris and may be single or multiple. Papules usually appear during the course of an iritis, may persist for days or weeks, and ultimately leave a depigmented, atrophic spot on the iris. Early papules may be superficial or deep and may be associated with broad-based posterior synechiae. Late papules may necrose or, rarely, bleed.

3. Gummata, most of which occur in the ciliary body or in the angle, are associated with severe syphilis.

The presence of immune complexes may be of importance in the causes of some of the lesions of both secondary and tertiary syphilis. Circulating immune complexes detected by Clq-binding and complement reduction were recently found in patients with secondary syphilis.[7] In patients with secondary syphilis a significant reduction in Clq-binding activity was also shown after treatment with penicillin. The reduction is rapid and returns the Clq-binding activity to nearly normal levels. Because our patient was adequately treated with penicillin before blood was drawn for the determination of immune-complex levels, we do not know what the levels were before treatment.

Studies in laboratory animals have shown that circulating complexes of antigen and antibody, or immune complexes, can be deposited in tissues and induce pathologic lesions, especially vasculitis and glomerulonephritis.[8] Immune complexes also may induce several disease states, among them the extravalvular manifestations of subacute bacterial endocarditis.[9] We speculate that there is an analogy between the pathogenesis of iris roseolae and papules, on the one hand, and the pathogenesis of the extravalvular signs of subacute bacterial endocarditis on the other, because both may perhaps depend on the deposition of circulating immune complexes.

The iris papules were slow to clear after penicillin therapy. This is in contrast to the rapid clearing of skin[10] and renal[11] lesions after treatment. The healing of the skin lesions parallels the decline in the serum antibody levels of secondary syphilis,[12] and the subsidence of the nephropathy parallels the disappearance of circulating immune complexes.[13] The vascular anomaly represented by the iris lesions could have persisted long after the decline of the level of circulating immune complexes. If occlusive vasculitis, secondary to immune complex depostion, was the forerunner of the formation of neovascular tufts in the iris, the neovascular tufts need not disappear as soon as the immune complexes disappear. The vascular tufts would persist as long as the basic circulatory disorder persists.

Iris angiography is still considered an experimental technique for the study of the pathophysiology of the anterior segment of the eye. With this procedure, we can record the dynamic events of vascular perfusion and obtain photographic documentation of therapeutic results. Angiographically detectable fluorescein leakage into the anterior chamber is considered a sign of disease related to the tightness and efficiency of the intercellular junctions of the vascular endothelium. (Although the iris angiogram in normal

young people usually shows no areas of leakage, pupillary leakage can sometimes be seen in older patients who show no evidence of disease.[14])

The first angiogram in our patient showed, in the early arterial phase, leakage of fluorescein at the pupillary margin and mid-iris in the region of the papules. This leakage persisted into the late phases and spread to include the entire capillary plexus of the pupillary margin. This indicates an effect of inflammation on the vessels in the region of both the roseolae and the papules of the eye. A second angiogram was made three months after the first, when the eye was free from any biomicroscopically detectable inflammatory signs. Because the leakage persisted from the same locations on the iris, we concluded that damage to the affected iris vessels had persisted.

Summary

A 30-year-old man developed bilateral iridocyclitis with iris roseolae and papules three months after the rash of secondary syphilis. The inflammation resolved after treatment with penicillin, intramuscularly, topically given corticosteroids, and cycloplegics. The serum immune-complex levels were slightly increased. Iris angiography showed leakage of fluorescein in the region of the papules and roseolae and from the vessels of the pupillary margin. When the eye was clinically uninflamed, a follow-up angiogram showed leakage of fluorescein from the same areas, suggesting that the inflammatory effect on the iris vessels persisted in the region of the roseolae, the papules, and the sphincter vessels.

Acknowledgments

John C. Cavender, M.D. reviewed the fluorescein angiograms, and Devron Char, M.D., performed the immune-complex assay.

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LACTATE DEHYDROGENASE IN TEARS

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There has been considerable interest in and conflicting information about enzymes in human tears since Fleming first described one (lysozyme) more than 50 years ago.[1] Recently, enzyme levels in tears have been used in the diagnosis of such diseases as keratoconjunctivitis sicca[2-4]; absent or decreased hexosaminodase A indicates Tay-Sachs disease.[5,6]

Lactate dehydrogenase, a ubiquitous intracellular enzyme which forms part of the glycolytic pathway, has been used for many years to diagnose cellular necrosis (in myocardial infarction or hepatic inflammation, for example), and has proved to be useful in the diagnosis of retinoblastoma, since 92% of patients with this disease have significant elevations of this enzyme in their aqueous humor.[7]

There has been confusion about the levels of lactate dehydrogenase in normal tears and, to the best of our knowledge, there have been no reports of lactate dehydrogenase isoenzyme patterns in tears from eyes with retinoblastoma. In 1974, van Haeringen and Glasius[8] reported that normal human tear fluid contains high levels of lactate dehydrogenase, ten to 20 times more than serum does. They also reported that the isoenzyme patterns for tears are entirely different from those of serum and concluded that "the lacrimal cells synthesize lactate dehydrogenase rather than collect it from the blood." More recently, however, these same investigators showed that the amount of lactate dehydrogenase in tears is related to the method of collection. When tears are collected on filter paper (Schirmer technique), the levels are ten to 20 times higher than those of serum, but when tears are collected by micropipettes no detectable lactate dehydrogenase appears.[9]

It was our purpose to measure lactate dehydrogenase levels in tears collected by micropipettes in order to clarify the contribution of artifact to the values obtained. We hoped that we would find high levels of lactate dehydrogenase in the tears of patients with retinoblastoma. If this happened, we would have a new diagnostic tool for substantiating a diagnosis of retinoblastoma without having to resort to an anterior chamber paracentesis.

SUBJECTS AND METHODS

We collected tear samples for lactate dehydrogenase determination via capillary action from the lacrimal lake, using hand-drawn capillary tubes. We took samples from 28 normal volunteers and from 17 patients with retinoblastoma. Samples were collected with and without the use of anesthesia. We analyzed lactate dehydrogenase levels and isoenzymes patterns by techniques described previously.[10] All determinations were performed on fresh samples stored at room temperature for a few hours.

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Results

We detected no lactate dehydrogenase in the tears of 14 of 20 normal, unanesthetized subjects. Two subjects had lactate dehydrogenase levels of 13 IU/liter and one each had values of 19, 20, 22, and 23 IU/liter. Of the eight normal subjects who received general anesthesia, six had low levels of lactate dehydrogenase (11, 12, 14, 15, 16, and 20 IU/liter). Only two had significant values (92 and 120 IU/liter); one of these had a "traumatized" eyelid that the anesthetist had rubbed accidentally.

This observation led us to measure lactate dehydrogenase levels before and after trauma. We performed one measurement, and then gently everted the eyelid and wiped the palpebral conjunctiva with a cotton applicator. We then took a second measurement. In each case, the level of lactate dehydrogenase increased to five to ten times the original level, from a mean of 12 to a mean of 90 IU/liter (Table 1).

Other studies have shown that normal aqueous humors and those with retinoblastoma have higher levels of lactate dehydrogenase 5 than of lactate dehydrogenase 1, the reverse of normal serum.[11] We found this to be true of tears as well (Table 2).

Of ten children known to have active tumors, six had low lactate dehydrogenase levels in their tears (five with 0, one with 13, and one with 16 IU/liter). Four of the children, however, did have high levels (49, 123, 158, and 581 IU/liter).

TABLE 1

LACTATE DEHYDROGENASE LEVELS BEFORE AND AFTER TRAUMA

Subjects	Lactate Dehydrogenase Levels (IU/Liter)	
	Before Trauma	After Trauma*
1	0	40.5
2	0	82.8
3	0	50.0
4	19	175.0
5	13	58.8
6	20	124.0
7	23	97.0
8	13	82.0
9	22	101.0

*Trauma consisted of everting the upper eyelid and wiping it gently with a cotton applicator.

Discussion

The lacrimal lake holds only 0.1 to 0.4 μl, and a minimum of 1 μl is needed for lactate dehydrogenase analysis, so we were forced to stimulate tear production to get adequate volumes. In cases where an enzyme is actively produced by the lacrimal gland, it is possible to stimulate tear production and leave the level of the enzyme in the tears unchanged. This is not true, however, for lactate dehydrogenase, because any technique that stimulates tear production will also dilute the basic lactate dehydrogenase in an unpredictable fashion. If lactate dehydrogenase can be detected in normal subjects, it is caused by the trauma of the detection technique.

Lactate dehydrogenase isoenzyme patterns do not appear to be helpful in diagnosing retinoblastoma. Even when the isoenzymes are present in measurable amounts, the patterns are similar to those of normal aqueous humor and may represent corneal or conjunctival isoenzymes, or both.

The level of lactate dehydrogenase in tears also seems to be of little use in diagnosing retinoblastoma. While it is possible that some patients with retinoblastoma do have high levels of lactate dehydrogenase, we cannot be certain that these patients have not rubbed their eyes or traumatized the conjunctival epithelial cells in some other way, thus creating falsely high values.

Our present techniques of collection and analysis are inadequate. If more

TABLE 2

ISOENZYME PATTERNS IN SEVEN PATIENTS WITH RETINOBLASTOMA

	Lactate Dehydrogenase Levels (IU/Liter)				
Patients*	LDH 1	LDH 2	LDH 3	LDH 4	LDH 5
1	0.07	4.9	13.0	35.0	43.0
2	3.0	14.0	25.0	30.0	28.0
3	2.7	9.0	21.0	31.5	26.0
4	2.2	6.4	15.9	32.7	40.7
5	1.2	9.4	13.3	37.1	31.7
6	8.2	12.9	15.9	24.3	36.4
7	4.2	7.3	13.5	36.2	30.6

*Samples were obtained while patients were anesthetized.

sensitive detection techniques which would permit analysis of the lacrimal lake alone were developed, levels of lactate dehydrogenase in tears might yet become clinically useful in the detection of retinoblastoma.

SUMMARY

Lactate dehydrogenase levels in tears were measured in normal subjects and in patients with retinoblastoma. When specimens were collected without trauma in normal subjects, there were usually no detectable levels of lactate dehydrogenase. When the eyelids were rubbed (probably liberating epithelial cells) lactate dehydrogenase levels were detectable and were five to ten times those of normal aqueous humor. When lactate dehydrogenase isoenzymes in such specimens were analyzed, the level of lactate dehydrogenase 5 was always higher than that of lactate dehydrogenase 1 (similar to normal aqueous humor).

While occasional patients with retinoblastoma do have elevated levels of lactate dehydrogenase in tears, it is uncertain if this is due to epithelial destruction or retinoblastoma itself. Lactate dehydrogenase in tears does not appear to be a useful test for the diagnosis of retinoblastoma given present techniques for collection and analysis.

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CEFOXITIN LEVELS IN HUMAN AQUEOUS HUMOR

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Cefoxitin is a new semisynthetic cephamycin with enhanced resistance to the action of gram-negative betalactamases.[1-3] Thus, it is effective against many gram-negative organisms such as indole-positive *Proteus* species and *Serratia* strains that are ordinarily resistant to cephalosporins. Additionally, the minimum inhibitory concentration of cefoxitin is lower than that of cephalothin against many cephalothin-sensitive gram-negative organisms.[3,4] Recent studies have suggested that cefoxitin penetrates the meninges better than other cephalosporins and that probenecid enhances this penetration.[5,6] For these reasons we undertook a study of cefoxitin penetration into human aqueous humor with and without probenecid.

SUBJECTS AND METHODS

Patients were selected for study who were to undergo elective cataract extraction and who had not received any antibiotic for one month before surgery. Patients were excluded if they stated they were allergic to penicillins or to cephalosporins. Informed written consent was obtained in all cases. In Group 1 (25 patients) 2 g of cefoxitin was dissolved into 10 ml of sterile water and was given intravenously over a three-minute period at time intervals scheduled to vary from 30 minutes to six hours before surgery. In Group 2 (23 patients) the timing and doses of cefoxitin were identical to those of Group 1, but, additionally, 500 mg of probenecid was given orally 30 minutes before the administration of cefoxitin. Patients were prepared for surgery with retrobulbar injections of 2% lidocaine (Xylocaine) and topical application of 0.5% tetracaine (Pontocaine) with one drop of silver protein (Argyrol). The eye was then washed with hexachlorophene (pHisoHex) and the cul-de-sacs were irrigated with normal saline. The residual silver protein was wiped out with a saline-moistened cotton swab.

Just before cataract extraction, a 2-mm nonpenetrating incision was made in the corneoscleral limbus and the anterior chamber was entered with a 27-gauge needle attached to a 2.5-ml plastic syringe. The aqueous humor was aspirated, placed in a sterile vial, and immediately frozen at −70°C until assayed. Between 0.1 and 0.2 ml of aqueous humor were obtained by this method. Simultaneous venous samples of blood were also obtained, spun, and the serum frozen at −20°C until the time of assay.

Antibiotic assays on serum and aqueous humor samples were performed with the disk plate method using *Escherichia coli* as the test organism.[7] One hundred milliliters of nutrient agar (Difco) was inoculated with 4 ml of a fresh suspension of the test organisms that had been standardized to give a 0.3 optical density at 660 nm. Five milliliters of this suspension was poured over 10-ml nutrient agar base layers in 100-mm petri dishes and al-

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lowed to cool. Four disks (⅜ inches) were then placed on each plate and 40 λ of sample or standard was pipetted onto each disk. Solutions of cefoxitin were prepared with pooled human serum and pH 6.0 phosphate buffer for the serum and aqueous humor standards, respectively. Final concentrations for both the serum and aqueous humor standards were 3.2, 1.6, 0.8, 0.4, and 0.2 μg/ml. Serum samples were diluted with pooled human sera so that the expected final concentrations would be within this range. Aqueous humor samples were assayed undiluted. Prepared plates were incubated at 37°C and zones of inhibition were read the next day.

Results

The serum levels after administration of 2 g of cefoxitin intravenously averaged 72.5, 48, 30.8, 17, and 6.7 μg/ml at 30 minutes and one, two, four, and six hours, respectively (Table 1). At 30 minutes, the average aqueous humor level was 1.54 μg/ml (Table 1). At one hour aqueous humor levels reached a peak of 3.16 μg/ml with a range from 3.0 to 3.4 μg/ml. Average levels decreased to 2.46, 1.22, and 0.82 μg/ml at two, four, and six hours, respectively. With preceding administration of probenecid, serum levels were higher (Table 2) with an average peak level of 79.4 μg/ml at one hour and average levels of 56.2, 33.8, and

TABLE 1

CEFOXITIN LEVELS IN SERUM AND AQUEOUS HUMOR AFTER ADMINISTRATION OF 2 g INTRAVENOUSLY

Case No.	Time after Injection	Level (μg/ml)		Average Levels (μg/ml)	
		Serum	Aqueous Humor	Serum	Aqueous Humor
1	23 min	70	1.5		
2	24 min	67.5	1.5	Approximately 30 min	
3	25 min	75	1.5	72.5	1.54
4	25 min	70	1.6		
5	25 min	75	1.6		
6	58 min	65	3		
7	1 hr 4 min	45	3.2	Approximately 1 hr	
8	1 hr 10 min	42.5	3.2	48	3.16
9	1 hr 10 min	40	3		
10	1 hr 16 min	47.5	3.4		
11	1 hr 55 min	30	2.4		
12	2 hrs 7 min	32	2.4	Approximately 2 hrs	
13	2 hrs 20 min	32	2.5	30.8	2.46
14	2 hrs 21 min	32	2.8		
15	2 hrs 30 min	28	2.2		
16	3 hrs 40 min	19	1.6		
17	4 hrs 13 min	16	1.2	Approximately 4 hrs	
18	4 hrs 13 min	15	0.7	17	1.22
19	4 hrs 20 min	11	0.8		
20	4 hrs 25 min	24	1.8		
21	5 hrs 43 min	9	1.0		
22	6 hrs	7	0.8	Approximately 6 hrs	
23	6 hrs	7.5	1.3	6.7	0.82
24	6 hrs 8 min	5	0.6		
25	6 hrs 25 min	5	0.4		

TABLE 2

CEFOXITIN LEVELS IN SERUM AND AQUEOUS HUMOR AFTER ADMINISTRATION OF 2 g INTRAVENOUSLY WITH PROBENECID

Case No.	Time after Injection	Level (μg/ml) Serum	Level (μg/ml) Aqueous Humor	Average Levels (μg/ml) Serum	Average Levels (μg/ml) Aqueous Humor
1	24 min	78	2.2		
2	24 min	80	1.8		
3	25 min	75	1.7		Approximately 30 min
4	26 min	75	2.2	76.6	1.9
5	40 min	75	1.6		
6	50 min	75	3.2		
7	50 min	90	2.8		Approximately 1 hr
8	1 hr 1 min	87.5	2.5	79.4	2.68
9	1 hr 16 min	65	2.2		
10	1 hr 38 min	70	3.4		
11	1 hr 49 min	56	3.2		Approximately 2 hrs
12	1 hr 50 min	45	1.8	56.2	3.0
13	2 hrs 16 min	60	3.4		
14	2 hrs 32 min	50	3.2		
15	3 hrs 50 min	42	3.2		
16	3 hrs 53 min	30	2.2		Approximately 4 hrs
17	3 hrs 57 min	34	3.2	33.8	2.76
18	4 hrs	28	2.2		
19	4 hrs 2 min	35	3.0		
20	5 hrs 45 min	28	2.0		
21	5 hrs 58 min	16	1.8		Approximately 6 hrs
22	6 hrs	7.5	1.3	16.4	1.82
23	6 hrs 17 min	14	2.2		

16.4 μg/ml at two, four, and six hours, respectively. Aqueous humor levels of 1.9, 2.68, 3.0, 2.76, and 1.82 μg/ml were obtained in these patients at 30 minutes and one, two, four, and six hours, respectively.

As graphically shown in the Figure, the addition of probenecid prolonged significant aqueous humor levels, but did not produce a higher peak level of cefoxitin. These findings are compatible with the inhibition by probenecid of the organic anion pump that removes anions such as penicillins or cephalosporins from aqueous humor.

No adverse reactions occurred from the administration of cefoxitin to these patients and no cases of endophthalmitis were observed.

DISCUSSION

Bacterial endophthalmitis occurs rarely, but is a devastating complication of cataract surgery.[8] It occurs after some filtering bleb procedures for glaucoma,[9] after trauma to the eye,[10] and rarely results from bacteremia.[11] The organisms most commonly responsible for this infection are *Staphylococcus aureus*, *Pseudomonas aeruginosa*, *P. mirabilis*, indole-positive *Proteus* species, *E. coli*, *Hemophilus influenzae*, and *Klebsiella* species.[8,10,12] Recently, an increase in the

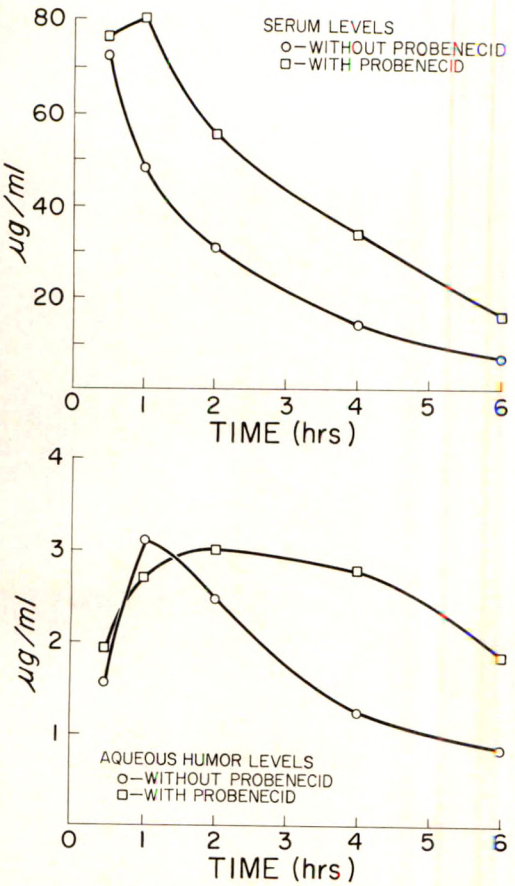


Figure (Axelrod and Kochman). Comparison of serum (top) and aqueous humor (bottom) concentrations after administration of 2 g of cefoxitin intravenously, with and without probenecid.

number of cases caused by *S. epidermidis* has also been reported.[12] The median minimum inhibitory concentration of cefoxitin against these organisms is presented in Table 3.[1,2,4,13,14] The minimum inhibitory concentrations against *Pseudomonas* are omitted because these organisms are resistant to cefoxitin. Cumulative susceptibility of bacterial eye pathogens to the attainable peak aqueous humor levels is also presented in Table 3. From 93 to 100% of gram-positive isolates would be susceptible, but only 32 to 78% of gram-negative organisms. Thus, although cefoxitin has an enhanced spectrum against gram-negative organisms, aqueous humor levels are still not high enough to be certain a specific organism would be eradicated. The addition of probenecid does not prevent this problem because higher aqueous humor levels were not achieved. Possibly, higher cefoxitin levels would be attained in the inflamed eye, but no cases of endophthalmitis were studied.

We compared cefoxitin penetration into human aqueous humor with that of other available parenteral cephalosporins. Data with respect to cephalothin, cephaloridine, and cefazolin are sparse, but show that peak aqueous levels of 3.3 μg/ml after cephalothin[15] and of 4.0 μg/ml after cephaloridine[16] have been achieved in isolated patients. Studies of five patients after administration of cefazolin intravenously have shown aqueous humor levels of 0 to 0.6 μg/ml.[15] A comparison of the cefoxitin data with a previous study of cefamandole penetration into human aqueous humor in 35 patients[17] is presented in Table 4. Average peak cefamandole levels of 1.26, 1.0, and 1.57 μg/ml were found at 30 minutes and one and two hours. These are much lower than the average peak levels of 1.54, 3.16, and 2.46 μg/ml of cefoxitin found in aqueous humor at the same time intervals. Because the average minimum inhibitory concentrations of cefoxitin against both gram-positive and gram-negative organisms tend to be higher than that of cefamandole,[4] the clinical significance of the higher cefoxitin levels cannot be determined with certainty. From the available data it appears that both cefamandole and cefoxitin might be effective as part of the therapeutic regimen for the treatment of bacterial endophthalmitis caused by gram-positive organisms. Their role in the therapy of serious gram-negative eye infections cannot be determined until more data are

TABLE 3

SUSCEPTIBILITY OF COMMON PATHOGENS TO CEFOXITIN IN VARIOUS STUDIES

Organism	Minimum Inhibitory Concentration, μg/ml				
	Median (range)		Cumulative Susceptibility to 3 μg/ml		
	Norrby, Brorsson, and Seeberg[13]	Eickoff and Ehret[4]	Heseltine and associates[14]	Wallick and Hendlin[1]	Neu[2]
Gram-positive					
S. aureus	1.0	3.2	2 (≤1-4)	93%	87%
S. epidermidis	—	3.2	—	—	100%
Streptococcus pneumoniae	1.0	0.4	≤1 (≤1-2)	100%	90%
St. pyogenes, Group A	—	0.4	2 (≤1-8)	100%	100%
Gram-negative					
E. coli	2.0	2.8	4 (≤1-16)	60%	36%
H. influenzae	1.3	2.1	32 (16-32)	—	—
Klebsiella species	—	—	32 (16-32)	34%	77%
P. mirabilis	0.75	4.2	—	78%	38%
Proteus species, indole positive	1.0	1.4	—	32%	2%

TABLE 4

COMPARISON OF CEFAMANDOLE AND CEFOXITIN SERUM AND AQUEOUS HUMOR LEVELS AFTER ADMINISTRATION OF 2 g INTRAVENOUSLY

Time (hrs)	Cefamandole			Cefoxitin		
	No. of Patients Studied	Average Concentration (range) μg/ml		No. of Patients Studied	Average Concentration (range) μg/ml	
		Serum	Aqueous Humor		Serum	Aqueous Humor
½	8	100.2 (50-1.56)	1.00 (0.37-1.56)	5	72.5 (67.5-75)	1.54 (1.5-1.6)
1	8	81.3 (38-116)	1.57 (0.66-2.6)	5	48 (40-65)	3.16 (3.0-3.4)
2	5	27.4 (15-36)	0.73 (0.12-1.8)	5	30.8 (28-32)	2.46 (2.2-2.8)
4	8	8.3 (0.4-16)	0.34 (0-0.92)	5	17.0 (11-24)	1.22 (0.7-1.8)
6	7	5.0 (0.9-10.6)		5	6.7 (5-9)	0.82 (0.4-1.3)

available with respect to penetration in the inflamed eye.

SUMMARY

We administered 2 g of cefoxitin to 25 patients before cataract surgery. An additional 23 patients received 500 mg of probenecid 30 minutes before the 2-g doses of cefoxitin. Average aqueous humor levels of 1.54, 3.16, 2.46, 1.22, and 0.82 μg/ml were achieved at 30 minutes and one, two, four, and six hours, respectively, after the 2-g dose. The addition of probenecid prolonged significant aqueous humor levels. Therapeutic levels against common gram-positive pathogens were consistently achieved at two hours after injection without probenecid and from two to four hours after injection with probenecid. Therapeutic levels effective against Enterobacteriaceae were inconsistent.

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EVALUATION OF THE CAUSES AND ACCURACY OF PHARMACOLOGIC LOCALIZATION IN HORNER'S SYNDROME

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The clinical features of Horner's syndrome are familiar to most physicians, but the causes are less well known. What are the incidences of the various causes of Horner's syndrome? In particular, how frequently is an undetected malignancy the underlying cause? How accurate are the pharmacologic tests used to identify the site of the interrupted neuron? In an attempt to answer these questions, we reviewed 450 cases of Horner's syndrome.

Horner's syndrome derives from an interruption of the sympathetic pathway at any point along its course between the hypothalamus and the orbit. This pathway is generally considered to consist of three neurons (Fig. 1). The first neuron (central) arises from the posterolateral region of the hypothalamus and descends through the brainstem and then through the lateral column of the cervical cord to terminate in the ciliospinal center of Budge between C-8 and T-1. The second neuron (intermediate) originates at this point and immediately leaves the central nervous system through the ventral roots of the lower cervical or first thoracic nerves, passing adjacent to the lower cervical vertebrae. Ascending over the pulmonary apex, the neuron passes through the inferior and middle cervical ganglia and terminates in the superior cervical ganglion high in the neck. At this point, the sympathetic ramifications multiply. For every intermediate neuron that enters the superior cervical ganglion, approximately 15 third neurons (peripheral) exit, distributing the sympathetic nerve supply to the head and neck. The neural fibers responsible for facial sweating travel with the external branches of the carotid artery to the face. Sympathetic innervation reaches the pupillary dilator muscle via neurons that pass through the internal carotid artery plexus, through the gasserian ganglion, through the first branch of the trigeminal nerve, and ultimately through the long ciliary nerves to the iris. The internal carotid plexus also carries sympathetic neurons from the superior cervical ganglion to be distributed with the terminal branches of the third cranial nerve to Müller's muscle of each eyelid.[1]

SUBJECTS AND METHODS

In a retrospective study covering the period from 1964 to 1977, we found 450 cases of Horner's syndrome in which testing was done by electronic infrared pupillography. Although Horner's original description of this syndrome included blepharoptosis and miosis with anhidrosis, in this series we have included oculosympathetic paralysis without anhidrosis. Horner's syndrome was confirmed when its three pupillographic characteristics of anisocoria, delay of redilatation, and absence of dilatation to a psychosensory stimulus were demonstrable (Fig. 2).

After Horner's syndrome was confirmed, each patient was tested pharmacologically in an attempt to localize the

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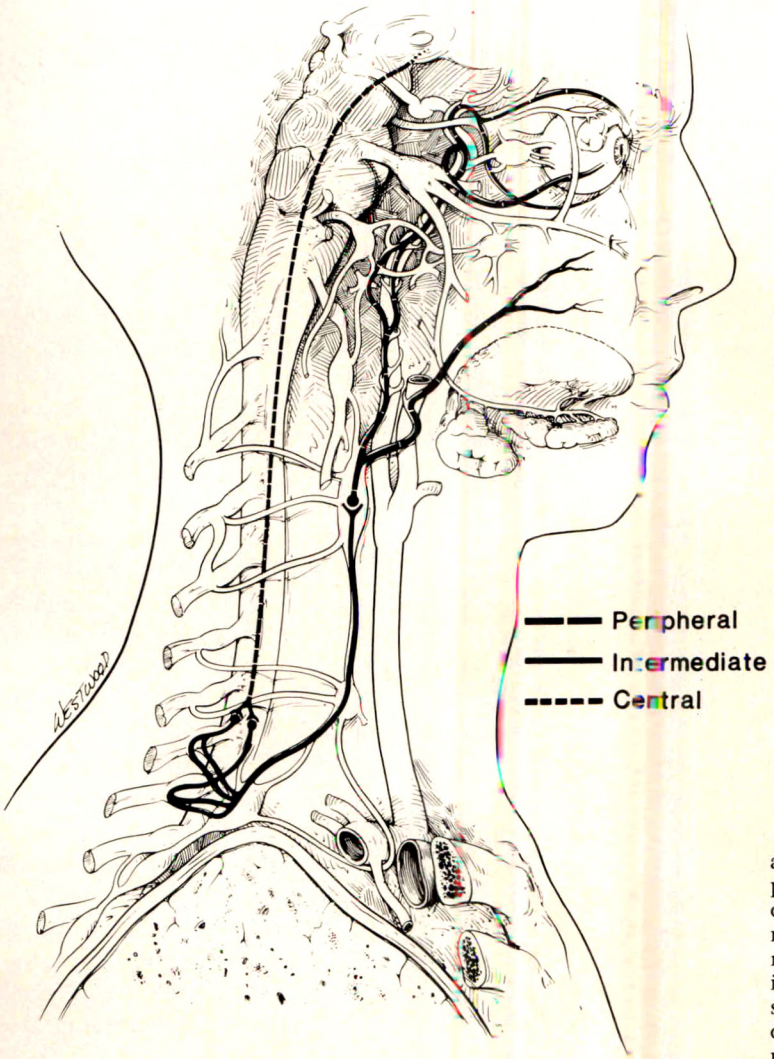


Fig. 1 (Maloney, Younge, and Moyer). Anatomy of sympathetic pathway showing central neuron, intermediate neuron, and peripheral neuron pathways. Note the proximity of pulmonary apex to sympathetic chain and lower cervical and upper thoracic nerves.

interruption to one of the three neurons of the sympathetic pathway. Initially, cocaine and epinephrine were used for this purpose in the following manner. After the baseline pupillogram, a drop of 4% cocaine was instilled into each eye, and this was repeated five minutes later. After 30 minutes, pupillography was repeated and the degree of pupillary dilatation was measured to the nearest 0.10 mm. No dilatation was interpreted as either preganglionic (intermediate) or postganglionic. Minimal dilatation (less than the normal pupil) was considered indicative of a central lesion. Then, one drop of 1% epinephrine was instilled into each eye, and this was repeated in five minutes. After 20 minutes, pupillography was repeated.

No further dilatation indicated a central lesion, poor or moderate dilatation indicated a preganglionic intermediate lesion, and marked dilatation indicated a peripheral lesion. In more recent years, since this drug has been available, hydroxyamphetamine has been used. After

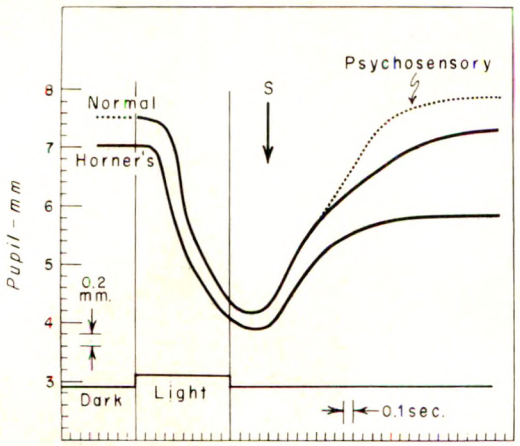


Fig. 2 (Maloney, Younge, and Moyer). Pupillographic characteristics in Horner's syndrome: anisocoria, the Horner's pupil being smaller and the degree of anisocoria being greater in darkness than in light; pupillary redilatation lag after transition from light to darkness; and absence of psychosensory dilatation in response to a sudden startling noise.

baseline pupillography, one drop of 1.0% hydroxyamphetamine was instilled into each eye, and repeated in five minutes. Pupillography was repeated 30 minutes later, and dilatation was measured to the nearest 0.10 mm. No response to hydroxyamphetamine was indicative of a complete postganglionic lesion, although a very minimal response was considered to be caused by an incomplete postganglionic lesion. Marked dilatation, sometimes with crossover beyond that of the normal pupil, indicated a preganglionic or intermediate lesion, and moderate dilatation was possibly indicative of a central lesion, although we did not try to distinguish these last two from each other because of the difficulty in defining "moderate." Thus, we combined these, and we tried to distinguish only the postganglionic from the other two lesions (Tables 1 and 2).

Results

The patients' ages ranged from 3 to 82 years, with a median of 53 years. Of the 450 patients, 270 (60%) were males. There was a predominance of males in four conditions: cluster headache (45 of 54; 83%), trauma (13 of 18; 72%), vascular occlusion (12 of 13; 92%), and Raeder's syndrome (14 of 18; 78%). Of the 450 patients, 441 (98%) had clinically detectable miosis, 396 (88%) had measurable blepharoptosis, 18 (4%) had anhidrosis, and 13 (3%) had heterochromia of the irises. Anhidrosis is undoubtedly common but was not searched for diligently in most of our cases. This retrospective study underemphasizes this important finding in localization; however, we are seeking this information in a current study. Of the 13 patients with heterochromia, ten (77%) had Horner's syndrome of congenital origin, and none of the ten had recent-onset disease. Het-

TABLE 1

Comparison of known location of lesion causing Horner's syndrome with location predicted by testing with cocaine and epinephrine

Known Location	No. of Patients	Pharmacologic Location			
		Central	Intermediate	Peripheral	Equivocal
Central	15	12	2	0	1
Intermediate	54	16	19	17	2
Peripheral	36	7	14	14	1
	105	35	35	31	4

*Includes 105 of 450 patients.

TABLE 2

COMPARISON OF KNOWN LOCATION OF LESION CAUSING HORNER'S SYNDROME WITH LOCATION PREDICTED BY HYDROXYAMPHETAMINE TEST*

Known Location	No. of Patients	Pharmacologic Location		
		Central or Intermediate	Peripheral	Equivocal
Central or intermediate	84	70	14†	0
Peripheral	78	2†	75	1†
	162	72	89	1

*Includes 162 of 450 patients.
†See Table 6.

erochromia of the iris associated with Horner's syndrome indicates a long duration of sympathetic denervation,[2] and this eliminates the need for further evaluation in search of other causes, such as undetected malignancy.

The cause was found in 270 (60%) of the cases. Of the 180 cases with no cause determined, 63 (35%) were re-examined from six months to 28 years after the initial finding of Horner's syndrome. The cause was not discovered subsequently in any case, indicating that, whatever the cause, the lesion probably was a benign and stable process. Of the 58 patients in the total series with systemic arterial hypertension, 48 (83%) had Horner's syndrome of undetermined cause. Similarly, the cause could not be determined in nine (69%) of the 13 patients with diabetes, in 11 (91%) of the 12 patients with vertebrobasilar occlusive disease, and in five (55%) of the nine patients with carotid arterial occlusion. We suspect that a local vascular event is a frequent but undetected cause of Horner's syndrome. Experimental evidence supports this postulate. In 1974, Sears, Kier, and Chavis[3] produced Horner's syndrome in eight rabbits by ligating the vascular supply to the superior cervical ganglion. These authors suggested that compromise of the vascular supply of the sympathetic pathway could account for Horner's syndrome.

The most frequent causes for the syndrome were tumor, cluster headache, and surgery (or radiation in addition to surgery) (Table 3). Because of the pattern of referral, the incidence of cluster headache may be more prevalent in our series than in a random sample of patients with Horner's syndrome. If many children had not been excluded from this series for lack of pupillography, the incidence of tumor might have been higher, because

TABLE 3

CAUSES OF HORNER'S SYNDROME*

Cause	Case	
	No.	%
Tumor	60	13
Cluster headache	54	12
Iatrogenic†	45	10
Raeder's syndrome	18	4
Trauma	18	4
Cervical disk protrusion	13	3
Congenital	13	3
Vascular occlusion	13	3
Vascular anomaly	9	2
Miscellaneous‡	27	6
Total	270	60

*Cause known in 270 (60%) of 450 cases.
†Primarily neck surgery and carotid angiography.
‡Includes pneumothorax, herpes zoster, cervical rib, and mediastinal lymphadenopathy.

Horner's syndrome in children, if not congenital, usually arises from malignant tumor.[4] The various tumors encountered in this series are listed in Table 4.

Of the 270 patients with a known cause, 34 (13%) had involvement of the central neuron, 120 (44%) the intermediate, and 116 (43%) the peripheral neuron of the sympathetic pathway. Some causes were associated with a particular neuron in the pathway; ten (91%) of the 11 patients with vascular occlusions had involvement of the central neuron, and 46 (77%) of the 60 with tumors had involvement of the intermediate neuron, as did 36 (84%) of the 43 with iatrogenic lesions. The 57 patients with cluster headache and the 20 with Raeder's syndrome all had involvement of the peripheral neuron.[5,6]

In 267 patients in whom the lesion was precisely located and identified, the known location was compared with the site predicted by pharmacologic testing (Table 1). One of us (W.F.M.) reviewed each test, interpreting the results uniformly, according to the modified schema from Foerster and Gagel[7] and enlarged upon by Thompson.[8] We did not continue the cocaine and epinephrine testing after hydroxyamphetamine became available, because the former drugs did not distinguish as clearly the preganglionic lesions from the postganglionic lesions as did hydroxyamphetamine. In those eyes tested with both sets of drugs, we waited 48 hours after cocaine testing to use the hydroxyamphetamine. In our institution, this delay was not generally possible because many patients were from distant places. Thus, the hydroxyamphetamine test became the usual test, and it continues to be the best drug test available.

TABLE 4

DISTRIBUTION OF TUMORS CAUSING HORNER'S SYNDROME

Tumor	No.
Benign	23 (38%)
Neuroma-neurofibroma	9
Thyroid adenoma	6
Meningioma	2
Dermal cyst	3
Osteoma	2
Pituitary adenoma	1
Malignant	37* (62%)
Metastasis	16
Primary lung carcinoma	6
Pharyngeal carcinoma	4
Hodgkin's lymphoma	3
Sarcoma	4
Thyroid carcinoma	2
Neuroblastoma	1
Lacrimal gland carcinoma	1
Total	60

*Thirteen of these had Horner's syndrome as their first sign of the malignancy.

DISCUSSION

In the analysis of the localizing value of pharmacologic tests, the term accuracy must be carefully defined. Clinically, we are interested in the diagnostic accuracy of the test or in the percentage of correct predictions, that is, in predicting the site of the interrupted neuron when the site is unknown.

In the hydroxyamphetamine test, the peripheral neuron was implicated pharmacologically in 89 of the patients (Table 2). Only 75 (84%) of these were proved later to have true peripheral lesions, whereas 14 (16%) had intermediate or central lesions that had been incorrectly localized by this test. Hydroxyamphetamine, then, has a diagnostic accuracy for peripheral neuron lesions of 84% (Table 5). However, hydroxyamphetamine localized lesions to more proximal levels in 97%, the higher diagnostic accuracy being the result of only two incorrect pharmacologic localizations to peripheral lesions (Table 5).

Another important term in the evaluation is sensitivity, that is, the ability of the test to confirm a site that is known. Sensitivity is the percentage of the total

TABLE 5

COMPARISON OF SENSITIVITY AND DIAGNOSTIC ACCURACY OF PHARMACOLOGIC TESTING*

Test	Pharmacologic Location	Sensitivity		Diagnostic Accuracy	
		No.	%	No.	%
Cocaine and epinephrine	Central	12/15	80	12/35	34
	Intermediate	19/54	35	19/35	54
	Peripheral	14/36	39	14/31	45
Hydroxyamphetamine	Central or intermediate	70/84	83	70/72	97
	Peripheral	75/78	96	75/89	84

*Data from Table 3. Sensitivity indicates % of all patients with a given known lesion location who also had a test result confirming that location; diagnostic accuracy, % of all patients with a test result which indicated a particular neuron who actually had the lesion located in that neuron.

cases with a known neuron site in which the test result indicated the same site of the neuron. Seventy-eight patients had known peripheral neuron lesions tested with hydroxyamphetamine (Table 2). The test result indicated that 75 patients had a peripheral lesion. Two had lesions incorrectly localized to the intermediate neuron, and one had an equivocal result. Therefore, the sensitivity of hydroxyamphetamine for peripheral neuron lesions is 96% (Table 5). The important difference is the accuracy to predict (diagnostic accuracy) compared with the accuracy to confirm (sensitivity). The diagnostic accuracy may differ considerably from the sensitivity (Table 5), a difference reflecting the number of cases in which localization by the test was incorrect. The cocaine and epinephrine test is not sufficiently accurate to reliably predict a lesion in any of the three neurons. Although hydroxyamphetamine demonstrates a high sensitivity for confirming peripheral neuron lesions, its diagnostic accuracy is somewhat less than optimal.

The rationale for using hydroxyamphetamine to localize the cause of Horner's syndrome is based primarily on three reported conditions: (1) that malignant neoplasm is a frequent cause of Horner's syndrome[9] and, therefore, that a patient with Horner's syndrome of undetermined cause is at significant risk of harboring an underlying malignant tumor; (2) that these tumors are almost exclusively located in the preganglionic (central or intermediate) neurons[10,11]; and (3) that the hydroxyamphetamine test can localize the lesion accurately.[11,12] Accordingly, in the patient with Horner's syndrome of unknown cause, if the hydroxyamphetamine test indicated a peripheral location, the condition probably is benign, whereas localization to one of the preganglionic neurons would indicate a high risk of harboring a malignant neoplasm. The latter patient would be referred for further diagnostic evaluation, whereas the former need not be. For use in this manner, hydroxyamphetamine has been recommended, and its clinical value extolled.[10-15]

Our findings are to some extent at variance with these three premises. In the first instance, our figures would indicate that with reference to Condition 1, 13 of 450 patients initially had Horner's syndrome, which was later determined to be caused by malignancy; thus, the risk of a patient harboring an undetected malignancy is less than 3%. Horner's syndrome

apparently is rarely the presenting sign of an undetected malignant tumor. Giles and Henderson[9] reported a 35.8% incidence of Horner's syndrome caused by malignant neoplasms, but they did not report the number of patients whose condition was diagnosed before the onset of Horner's syndrome. Further, with reference to Condition 2, six (16.2%) of the 37 malignant tumors in this series involved the peripheral neuron, and one was located in the central neuron; the remainder affected the intermediate neuron, hardly an exclusive confinement of tumor to the intermediate neuron, although when the lesion does not involve this intermediate neuron, these patients may have other localizing clinical signs. Raeder's syndrome is not an entirely benign lesion.[6] In this series, there were no patients with parasellar tumors responsible for Raeder's syndrome, because referral for pupillography was unnecessary in the diagnosis. Last, we have emphasized the important clinical difference between the diagnostic accuracy and the sensitivity of this test. Grimson and Thompson[11] reported that, in a series of 36 patients, the test was highly accurate in confirming the presence of a postganglionic lesion. Our results indicating sensitivity of 96% agree with this finding for postganglionic involvement, but in predicting a site among unknowns, the diagnostic accuracy was only 84% in localizing to peripheral neurons, and a comfortable 97% to more proximal neurons.

We attempted to find an explanation for the incorrect pharmacologic localization in the 11 and three patients whose lesions were in the intermediate and central neurons, respectively (Table 6). More than one neuron may have been involved, and such influences as diffuse tumor along the sympathetic pathway might easily involve the intermediate neuron or its termination at the ganglion, as well as the ganglion itself. Similarly, radiation treatment produces considerable compromise of viability, not only of tissue but also of vascular supply. Nonetheless, there were patients with incontrovertible second neuron involvement, in whom hydroxyamphetamine simply localized incorrectly. To us, an incomplete peripheral neuron lesion indicated a Horner pupil that responded minimally to hydroxyamphetamine (Paredrine), as compared with the normal pupil. Many of the cases whose data are reported in Table 6 have such interpretations, and perhaps one should be cautious in being adamant about such localizing value in this circumstance. The 97% figure in Table 5 is acceptable in localizing a lesion pharmacologically to the proximal neurons; not so reassuring, however, is the diagnostic ability of hydroxyamphetamine to accurately predict that a lesion is peripheral, when this is only 84% certain. A significant proportion of these were not benign lesions. Long-standing Horner's lesions, as in the congenital type, usually localize pharmacologically to the third neuron, though no one has yet documented congenital cases of known intermediate lesions or central lesions with subsequent drug testing. Possibly long-standing third neurons, initially intact, act like incomplete postganglionic lesions when tested with hydroxyamphetamine (H. S. Thompson, personal communication, March 1979).

Although Horner's syndrome was rarely (less than 3%) the initial sign of undetected malignancy in our series, patients with an undetected tumor are most in need of identification. Although the values of the hydroxyamphetamine test may be overemphasized in previous reports, one characteristic symptom that helps to identify these patients has not received enough emphasis. Of the 13

TABLE 6

DATA ON PATIENTS WHOSE HYDROXYAMPHETAMINE TESTS WERE INCORRECT IN LOCALIZATION

Case No.	Presumed Cause and Site	Neuron Involved	Hydroxyamphetamine Localization	Comment
1	Neuroblastoma in neck; Horner's syndrome present from age 6 weeks; patient age 8 years	2nd	3rd*	Possible involvement of superior cervical ganglion, or possibly unrelated congenital Horner's syndrome
2	Apical lung tumor	2nd	3rd*	No explanation obtained from chart; Horner's syndrome present before any treatment
3	Apical lung tumor	2nd	3rd*	No explanation obtained from chart
4	Metastatic breast tumor to supraclavicular nodes; vocal cord paralysis; radiation treatment	2nd	3rd*	Possible involvement of superior cervical ganglion; possible radiation necrosis or secondary vascular compromise
5	Metastatic brachial plexus tumor from breast	2nd	3rd	No explanation obtained from chart
6	Exploration for recurrent brachial plexus tumor; no tumor found, only radiation necrosis	2nd	3rd	Probable radiation effect, either direct or on vascular structures of 3rd neuron
7	Cervical laminectomy	2nd	3rd	No evidence from the operative notes of damage to sympathetic structures
8	Cervical fusion	2nd	3rd	Level of surgery below superior cervical ganglion
9	Sympathectomy for thoracic outlet syndrome	2nd	3rd*	Lesion definitely confined to 2nd neuron only
10	Motor vehicle accident, trauma to neck; arm paresthesias	2nd	3rd	No evidence of damage to structures higher in the neck
11	Cervical disk, L-3	2nd	3rd*	No reason found for 3rd neuron involvement
12	Wallenberg syndrome	1st	3rd*	No reason for false localization found on chart review
13	Multiple sclerosis, with high cord lesion	1st	3rd*	Possibly the central cord lesions involved the 2nd neuron at its source
14	Arnold-Chiari malformation; syringomyelia	1st	3rd*	No reason for false localization found on chart review

*Incomplete postganglionic.

patients with undetected malignancy in our series, ten (77%) had tumors (primary or metastatic) involving the pulmonary apex, resulting in Pancoast's syndrome. Because of the location of these tumors, these patients are more likely to have Horner's syndrome as an initial sign than any other type. In his classic description in 1932, Pancoast[16] characterized the syndrome as caused by a tumor of the superior pulmonary sulcus, producing Horner's syndrome and a characteristic arm pain caused by involvement of the adjacent sympathetic chain as well as the eighth cervical and first thoracic nerves. Nine of the ten patients with Pancoast's syndrome in our series had arm pain either as their sole symptom or as one of their primary initial complaints. Thus, nine of the 13 patients with undetected malignancy (69%) could have been identified initially by history alone. We believe that the symptom of arm pain needs to be re-emphasized as an integral part of Pancoast's syndrome because it provides a reliable clue to the identification of most of that small group of patients in whom Horner's syndrome is caused by an undetected malignancy.

Summary

Of 450 cases of Horner's syndrome examined by pupillography, 13% were caused by tumor but less than 3% were undetected malignant tumors. The largest group of lesions (40%) had an undetermined cause, presumably related to vascular disease. Although the hydroxyamphetamine test usually can differentiate postganglionic from preganglionic lesions, its clinical value and accuracy for this purpose has been overemphasized in previous reports. Of the 13 undetected malignant neoplasms, nine (69%) manifested as Pancoast's syndrome with arm pain, a reliable sign to identify patients with Horner's syndrome caused by undetected malignancy.

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OCULAR QUININE TOXICITY

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The use of quinine for the treatment of malaria has decreased since World War II, but quinine is still frequently used for muscle cramps[1] and as a filler in illegal narcotics. Quinine overdoses occur because of abortion attempts, suicide attempts, or accidental ingestion. Many reports have been written about ocular quinine toxicity, but there is still controversy surrounding the most basic questions.[2-4]

Vascular narrowing and optic disk pallor are present months and years after quinine overdose. However, arteriolar constriction and optic disk pallor,[5-7] venous dilatation and retinal edema,[8,9] and a normal fundus[10-13] are among the ophthalmoscopic findings reported shortly after overdose.

The appearance of vasoconstriction suggests that quinine has a primary vascular effect, leading to retinal ischemia.[14-16] However, in the acute stages, a normal electroretinogram and ophthalmoscopic appearance have led others to postulate a direct toxic effect on the retinal ganglion cell layer.[6,11,13] Some investigators have shown evidence for a direct toxic effect on several layers of the retina.[17,18]

Successful treatment of ocular quinine toxicity has been reported with vasodilators,[6,15,19,20] stellate ganglion block,[16,21,22] and corticotropin (ACTH)[5] administered intravenously. However, the natural course of the disease is one of improvement, which makes treatment results difficult to evaluate.

We recently studied a typical case of quinine overdose in a 25-year-old woman.

CASE REPORT

On Sept. 17, 1977, at 7:30 PM, a 25-year-old woman swallowed 12 to 15 tablets (5 grains each) of quinine (total 3.7 to 4.7g) as a suicidal gesture. At 11 PM, her friends found her lying on the floor obtunded and vomiting. Through the night she gradually became more alert, but she continued vomiting and complained of buzzing in her ears. At about 3 AM, she noted that her vision was bad, and at dawn she said she was totally blind.

At 10 AM, 14.5 hours after the ingestion, she came here. She had no light perception in either eye. Her pupils were 8 mm in diameter and were nonreactive to light but had a marked hippus and constricted moderately to near. The extraocular motility and anterior segments were normal. The retinas had a slightly hazy appearance with mild venous distention, symmetrical in the two eyes (Fig. 1, left). A fluorescein angiogram confirmed normal arterial caliber and mild venous distention (Fig. 1, right). Background fluorescence was normal.

Except for mild asthma, her medical history was unremarkable. Vital signs and results of general physical examination were normal, except for an apparent decreased hearing and her ocular findings. Routine serum electrolytes, glucose, blood urea nitrogen, calcium, creatinine, uric acid, phosphorous, total protein, albumin, direct and total bilirubin, and serologic test for syphilis were all within normal limits. The hematocrit was 40.3% and the white blood cell count was 16,000/mm^3 with 86% segmented neutrophils, 5% lymphocytes, 3% monocytes, and 6% immature neutrophils. Chest X-ray was normal. The electrocardiogram showed mild Q-T interval prolongation (a change typical of quinine toxicity[1]) and mild ST and T-wave abnormalities. A repeat electrocardiogram nine days later was normal. The patient was given oxygen by face mask, but no other treatment.

The following morning, 35 hours after the ingestion, the patient's central vision had returned. Her nausea had cleared, and she had only mild buzzing in the ears.

Visual acuity was 6/6 (20/20) in each eye without

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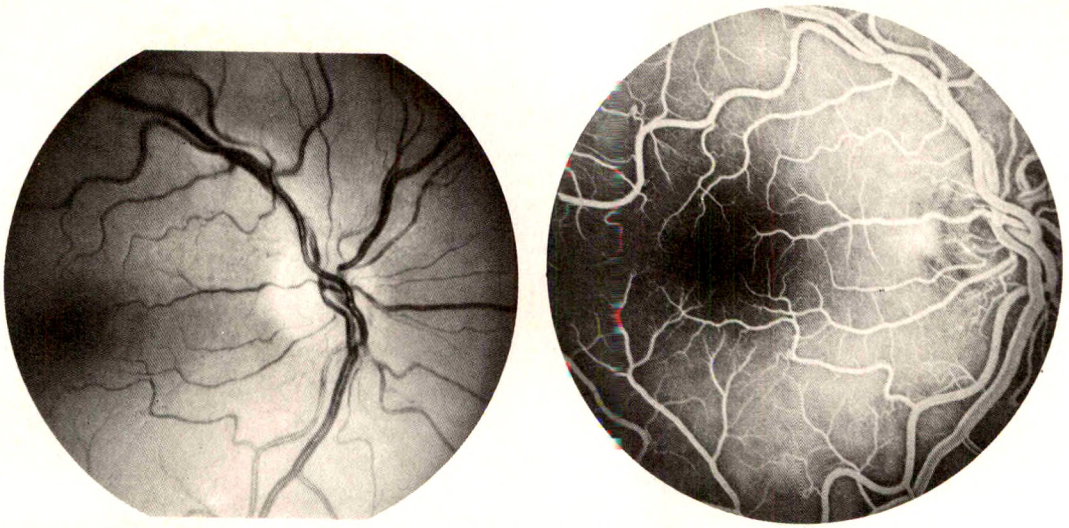


Fig. 1 (Brinton and associates). Left, Fundus photograph of the right eye on the first day (within 18 hours of the quinine ingestion). Right, Fluorescein angiogram of the right eye taken just after the fundus picture.

correction. The pupils reacted sluggishly to light but were still dilated and had marked hippus. The visual fields were markedly constricted (Fig. 2, top). The fundus appearance and fluorescein angiogram were essentially unchanged (Fig. 3, top left and right).

Four days after ingestion of quinine, the size of the central visual field had increased slightly, and bilateral temporal islands of visual field were first discovered (Fig. 2, bottom). Visual acuity was 6/4.5 (20/15).

Five days after ingestion of quinine, early vascular narrowing and optic disk pallor were first noted and the hazy retinal appearance and venous distention had resolved (Fig. 3, bottom left). During the next few days, the retinal vascular narrowing and optic disk pallor gradually became more prominent. The fluorescein angiogram nine days after ingestion showed a marked increase in the background fluorescence. (Fig. 4, top left and right). The visual fields remained unchanged (Fig. 5, top).

After six months, the visual acuity was 6/4.5 (20/15) in each eye and the anterior segment remained normal. Visual fields showed some loss in the size of the temporal islands but were otherwise unchanged (Fig. 5, bottom). The size of the visual field increased appropriately with increasing distance from the patient. The fundus showed a progression of the disk pallor and vascular narrowing. The choroidal pattern was also more readily visible. The fluorescein angiogram showed a marked increase in the visibility of the choroidal fluorescence, as well as the vascular narrowing (Fig. 4, bottom left and right).

The serum quinine level drawn one day after quinine ingestion was reported as 2.04 mg/100 ml by the Dade County, Florida, Medical Examiner's Office Toxicology Laboratory. Their experience is that serum quinine levels greater than 1.00 mg/100 ml are frequently fatal, though they find wide variability. Ten days after quinine ingestion the patient's serum quinine level was "nondetectable."

Results

The first electroretinogram was taken the first day, 18 hours after quinine ingestion, when the patient had no light perception. It had a normal appearance but there was marked slowing of the a-wave (Fig. 6). This returned to normal the second day after quinine ingestion when the patient's central vision returned. Other abnormalities included: (1) loss of oscillatory potentials with no tendency for recovery; (2) increased depth of the a-wave that returned toward normal after several days; (3) decreased size of the b-wave with a return toward normal in the first few days, but a late progressive decrease in size (Fig. 6).

Electroretinograms were obtained after 15 minutes of dark adaptation. Flashes of light from a photostimulator illuminated a hemispherical bowl 43 cm in diameter to produce a ganzfeld stimulus. Each flash had a light flux of 5 lux-sec at the patient's cornea. The pupil was dilated. In a normal eye this stimulus gen-

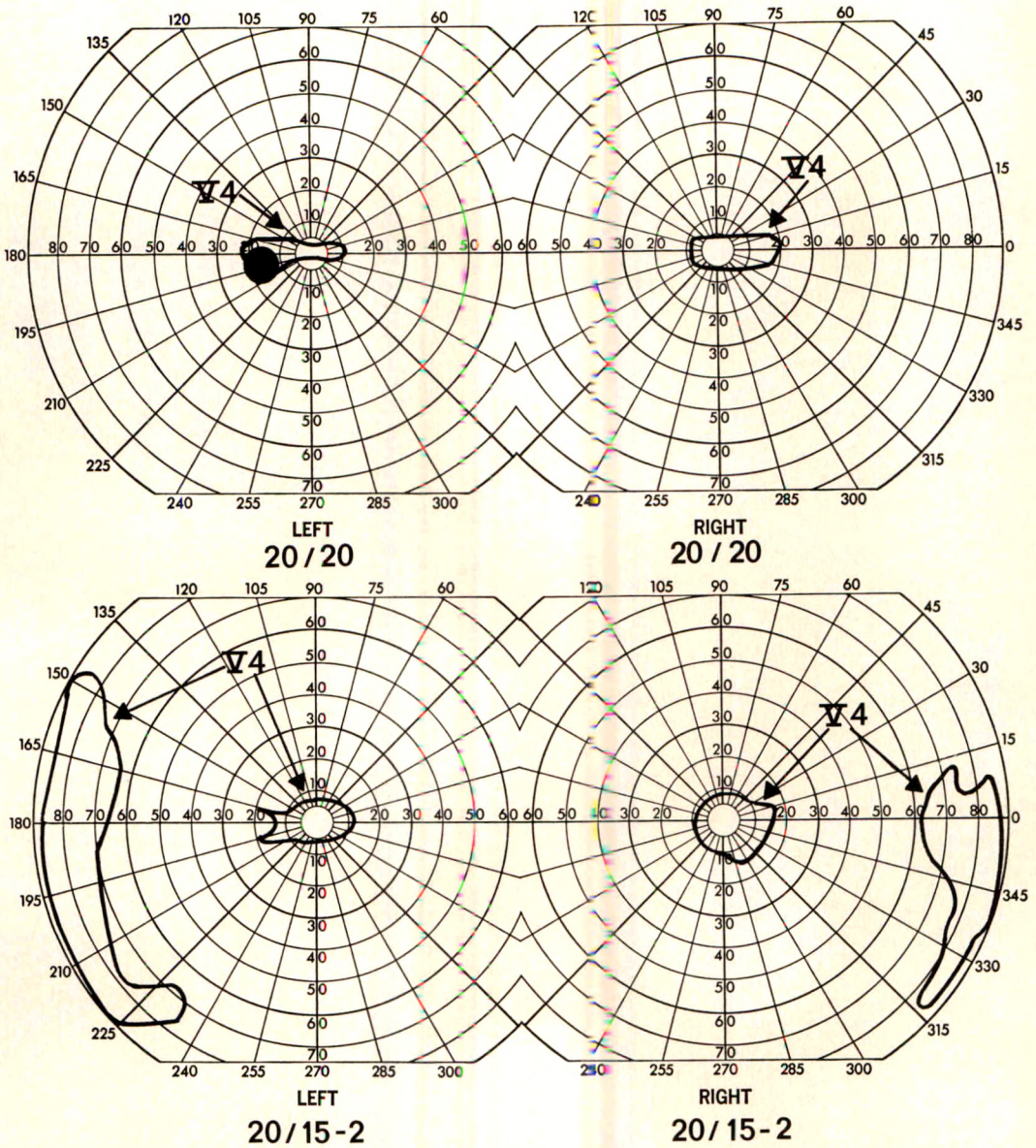


Fig. 2 (Brinton and associates). Goldman visual fields two and four days after ingestion of quinine tablets.

erates an electroretinogram composed of both rod and cone activity.

The electro-oculogram on the second and third days after quinine ingestion had a decreased light rise of 1.3 in each eye. On day 9 the light rise was slightly higher but still decreased at 1.6 in each eye. The normal in our laboratory is greater than 1.65. The light rise of the electro-oculogram was measured in the standard way.[2]

The visual-evoked potential showed increased latency in both eyes during all trials. The delayed visual-evoked potential was constant from three days through six months after quinine inges-

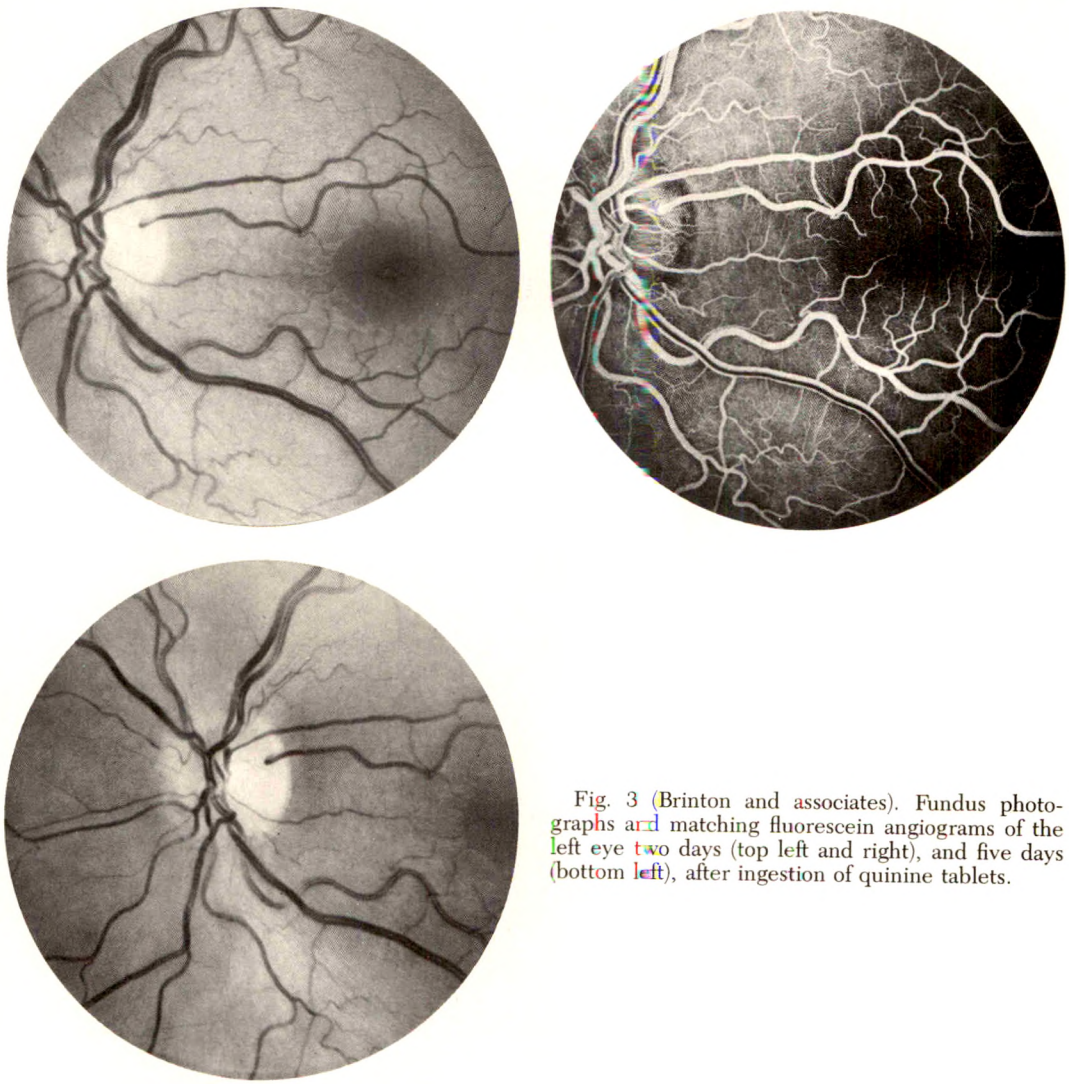


Fig. 3 (Brinton and associates). Fundus photographs and matching fluorescein angiograms of the left eye two days (top left and right), and five days (bottom left), after ingestion of quinine tablets.

tion. The stimulus for the visual-evoked potential consisted of a checkerboard pattern rear-projected onto a screen that subtended 25 degrees × 25 degrees in the patient's visual field. A moving mirror caused the checkerboard to reverse contrast once per second. Check sizes of 15 and 50 minutes of arc were used.

Dark adaptation performed six and nine days after quinine ingestion showed delayed cone adaptation and little or no rod function, in keeping with the patient's complaint of poor night vision.

Color testing with Farnsworth panel D-15 was normal in both eyes three and nine days and six months after quinine ingestion. With the American Optical Pseudo-isochromatic plates, she was correct on all except plate number 10 with each eye three days after quinine ingestion, and was correct on 15 out of 15 nine days and six months after quinine ingestion. Farnsworth-Munsell 100-Hue test was slightly abnormal after three days, with error scores of 76 in the right eye and 130 in the left eye.[24] After nine days,

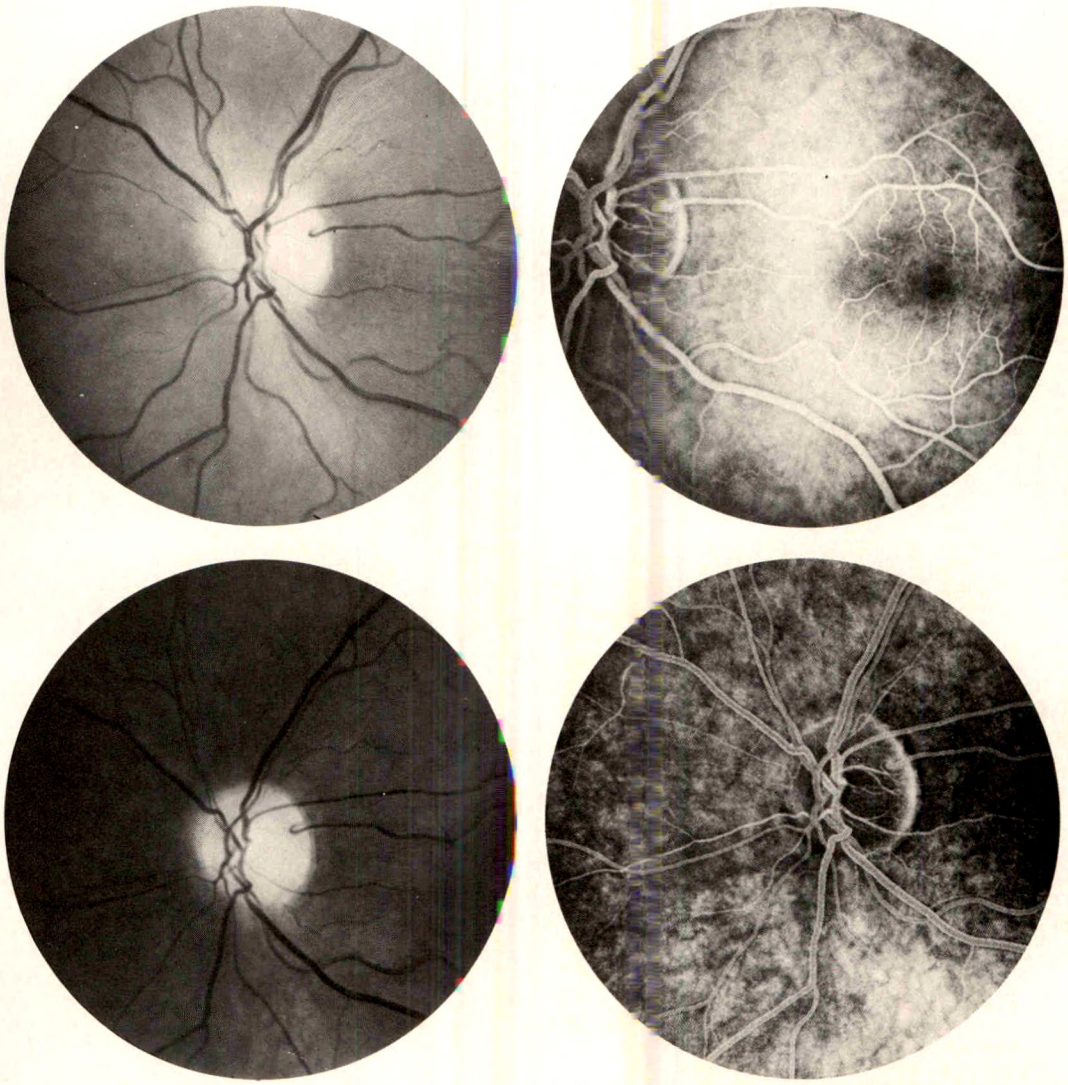


Fig. 4 (Brinton and associates). Fundus photographs and matching fluorescein angiograms of the left eye nine days (top left and right) and six months (bottom left and right) after ingestion of quinine tablets.

the results were clearly normal, with error scores of 45 in the right eye and 47 in the left eye.

Discussion

The findings support the opinion that during transient loss of vision from quinine overdose, the fundus appears relatively normal, with only mild venous distention and retinal haziness. The classic appearance of optic disk pallor and vascular narrowing begins to appear several days later, but takes months to develop fully.

Concerning pathogenesis, the idea that quinine acts directly on the retinal vasculature to cause retinal ischemia was based mainly on the late ophthalmoscopic appearance. However, on the first day the arterial caliber was normal (Fig. 1, left), with no light perception. The retinal ischemia theory is weakened further in

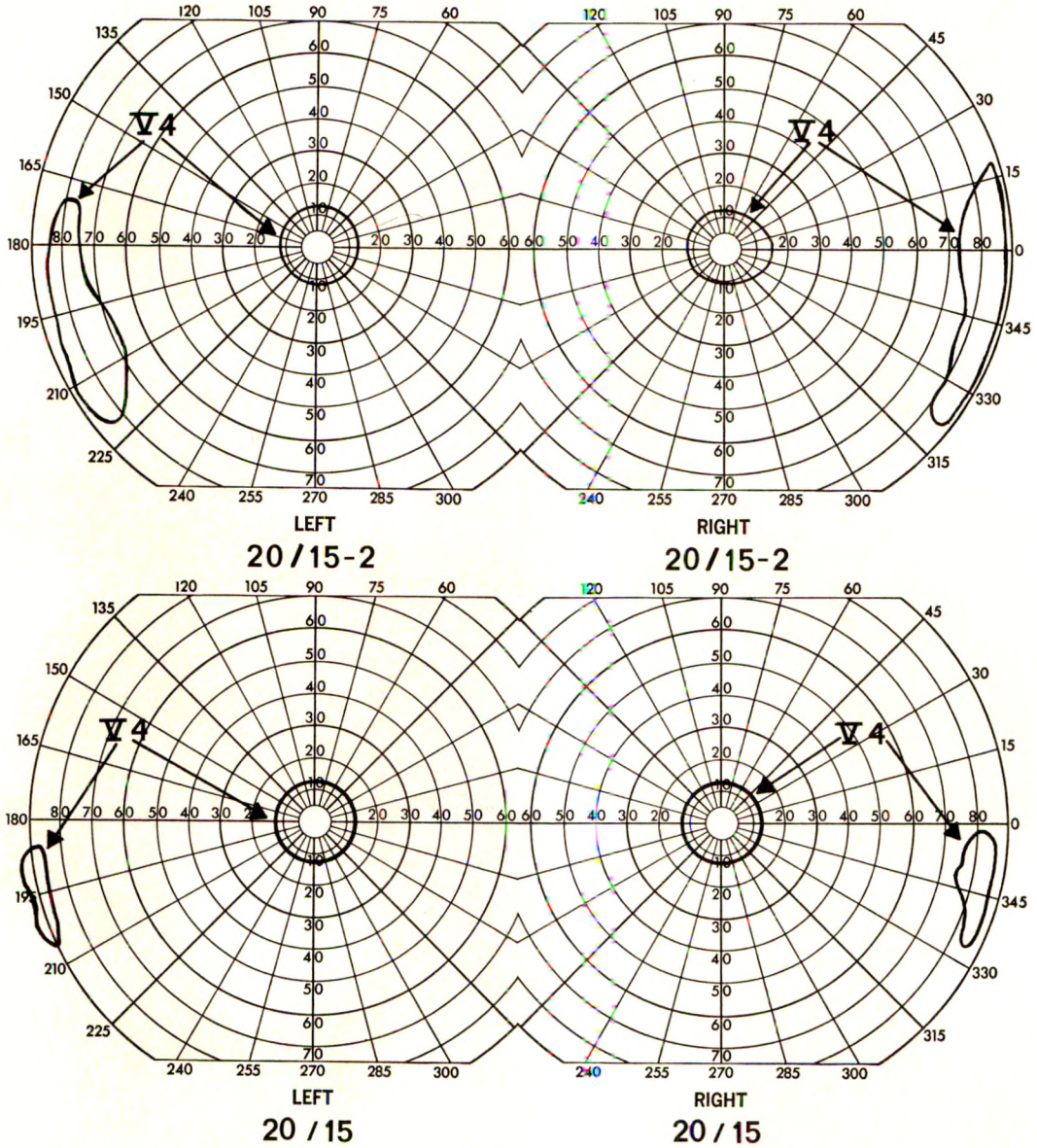


Fig. 5 (Brinton and associates). Goldmann visual fields nine days (top) and six months (bottom) after ingestion of quinine tablets.

that during the first few days visual function improved as vascular narrowing got worse.

The alternative hypothesis is that quinine is directly toxic to the retinal cells. Because earlier studies showed the electroretinogram to be normal,[11,12,21] quinine was thought to effect only the ganglion cells, an electroretinographically silent layer of the retina. However, the delayed a-wave (prolonged implicit time) seen in our patient when visual acuity was no light perception is good evidence for an acute direct effect on the photoreceptor

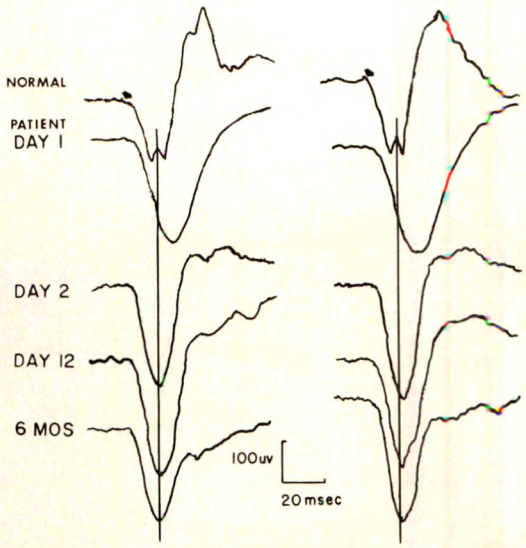


Fig. 6 (Brinton and associates). Electroretinograms comparing those of an age-and sex-matched normal subject with those of the patient on one, two, and 12 days, and six months after ingestion of quinine tablets for right and left eyes, respectively. The small black arrows indicate the time of the light stimulus. The black vertical lines indicate the implicit time of the a-wave in each eye of the normal subject.

cell layer, as well.[25] The late decline in b-wave amplitude suggests inner nuclear layer damage,[26] either by acting directly on the cells of the inner nuclear layer or by causing retinal vascular narrowing and ischemia. Previous reports describe subtle electrotretinographic abnormalities seen shortly after quinine exposure in humans,[17,27] as well as in animals.[19,28,29] Histopathologic changes in animals were found in the photoreceptor cell as well as the ganglion cell layers of the retina.[30,31] There has been no human histopathologic study to confirm this, however.

The decreased electro-oculogram light rise seen in this and in previous cases[11,32] suggests retinal pigment epithelial involvement.[23] Damage to the pigment epithelium may also be indicated by the increased visibility of the choroidal pattern in late fundus photographs and increased background fluorescence in late angiograms. Diffuse bone-spicule retinal pigment epithelial abnormality has been reported in one case of quinine toxicity.[33] Diffuse iris pigment epithelial damage has also been seen,[34] but in our case transillumination of the iris showed no defects.

There is, therefore, good evidence that quinine has a direct toxic effect on the photoreceptor cell layer, as well as the ganglion cell layer. There is evidence to indicate that quinine also affects the pigment epithelium and other layers of the retina.

The term "idiosyncratic" has been applied to quinine toxicity because of the variability in the toxic dose from patient to patient.[3,4] The recommended daily therapeutic dose is no more than 2.0 g per day and the fatal oral dose for adults is approximately 8 g.[1] Quinine toxicity is usual in doses greater than 4 g,[22] but there have been many case reports with much smaller doses. The ocular effects of quinine seem to be a dose-related toxic reaction that sometimes appears idiosyncratic because of variable individual susceptibility and a narrow therapeutic index.

The question, "Is there any effective treatment?" has never had a good answer. Recommendations for treatment are supported by little except case reports. Our case shows, as has been believed in the past, that central vision tends to recover without treatment.

Summary

A 25-year-old woman had no light perception in either eye 14.5 hours after ingesting 3.7 to 4.7 g of quinine. Fundus photographs showed mild venous distention and retinal haziness, but normal arteries. The electroretinogram, electro-oculogram, and visual-evoked potential were all mildly abnormal.

Two days after the patient ingested quinine, her visual acuity returned to 6/6

(20/20) with a markedly constricted visual field. The classic late fundus appearance of quinine toxicity, marked vascular narrowing, and disk pallor developed within six months. Visual fields remained constricted to 12 degrees with small temporal islands.

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HALLERMANN-STREIFF SYNDROME ASSOCIATED WITH SCLEROCORNEA, ANIRIDIA, AND A CHROMOSOMAL ABNORMALITY

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Congenital anomalies of the face and skull are frequently associated with ocular abnormalities. Numerous reports have described a syndrome consisting of dyscephaly with a bird-like face, dental anomalies, proportionate dwarfism, hypotrichosis and atrophy of the skin associated with microphthalmia, microcornea, and congenital cataracts.[1-12] This mandibulo-oculofacial dyscephaly, or Hallermann-Streiff syndrome, is thought to result from a sporadic mutation.[6] The only previous chromosomal abnormality demonstrated in Hallermann-Streiff syndrome was in a patient who had a discrepancy in the size of two members of the group D chromosome.[10]

We studied two distant cousins with Hallermann-Streiff syndrome, one of whom had previously unreported ocular signs of sclerocornea and aniridia and a chromosomal abnormality.

CASE REPORT

A boy was born in April 1978 after a full-term pregnancy and normal delivery to parents who are distant cousins (Fig. 1).

There was no history of maternal infection during gestation and the mother was 23 years old at the time of delivery. Shortly after birth the child was noted to have multiple congenital anomalies, including bilateral buphthalmos with congenital glaucoma, sclerocornea, bifid great toes, abnormal ulnar skin creases and a peculiar facial appearance characterized by micrognathia and a parrot-beaked nose (Fig. 2).

During the first week of life the patient underwent bilateral trabeculectomies. Postoperatively the intraocular pressure remained high, and we performed a bilateral thermosclerostomy when the patient was 3 months of age. This procedure initially controlled the intraocular pressure, but over the subsequent months the pressure increased to 28 mm Hg and we again performed a thermosclerostomy in each eye. The intraocular pressure increased again and both eyes received cyclocryotherapy.

In September 1978 we performed a 6.5-mm penetrating keratoplasty on the right eye. At surgery, the patient was found to have total aniridia. Because of the absence of the iris, the unrestricted lens had entered the wound, necessitating extracapsular cataract extraction. Postoperatively the corneal graft has remained clear and the intraocular pressure has been controlled with topically applied timolol (0.5%, twice each day) and acetazolamide (75 mg every six hours).

We performed a discission of the posterior capsule in January 1979, but the patient responded only to light and bright colors when fitted with a +12.0 diopter soft contact lens. Ophthalmoscopic examination disclosed advanced glaucomatous optic atrophy, but the posterior pole appeared normal. Recent radiologic examination of the patient's skull demonstrated hypoplasia of the mandible. As the patient has grown, he has developed hypotrichosis, frontal alopecia, and neural dysacousis.

When the patient was 1 year old, a chromosome analysis of peripheral blood lymphocytes (using the trypsin-Giemsa banding technique of Klinger[13]) revealed a 46,XY,10q+ karyotype, indicating extra chromosomal material in the long arm of the tenth chromosome (Fig. 3). There did not appear to be a translocation from another chromosome and the finding was interpreted as a spontaneous mutation resulting in partial duplication 10q+ syndrome. Similar chromosome analyses performed on the mother and father revealed normal karyotypes.

The patient has a 30-year-old second cousin with documented Hallermann-Streiff syndrome (Fig. 4), including similar facial characteristics, microcornea, congenital cataracts that were surgically removed when he was 4 and 5 years of age, and aphakic glaucoma. This patient also has dental anomalies, hypotrichosis, atrophy of the facial skin, and mental retardation. Chromosome analysis of this patient showed a normal karyotype.

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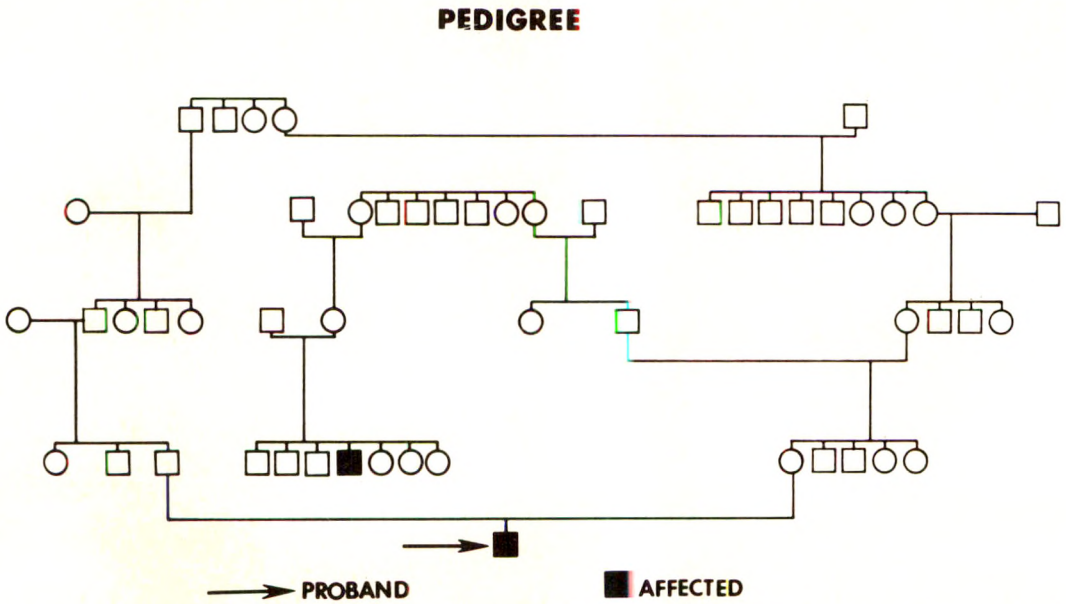


Fig. 1 (Schanzlin, Goldberg, and Brown). Family pedigree of patient with Hallermann-Streiff syndrome.



Fig. 2 (Schanzlin, Goldberg, and Brown). Infant with parrot-beaked nose, micrognathia, buphthalmos, and sclerocornea.

Discussion

The association of dyscephaly with ocular abnormalities has been the subject of many reports since Aubry's discussion of "sutural congenital alopecia" in 1893.[1] In 1948, Hallermann[2] reported the association of bird-like facies with congenital cataracts, and Streiff,[3] in 1950, reported mandibulofacial malformation with ocular changes. Ullrich and Fremerey-Dohna[4] described the association of congenital cataracts with hypotrichosis in 1953. In 1958 François[5] reviewed the previously reported cases and added one additional case; he concluded that they represented a new syndrome. The seven essential signs of the mandibulo-oculo facial dyscephaly, as reported by François, were dyscephaly with a bird-like face, dental anomalies, proportionate nanism, hypotrichosis, atrophy of the skin, bilateral microphthalmos, and congenital cataract. In 1960, Falls and Schull[6] reported six additional cases under the title of Hallermann-Streiff syndrome. In two of these there was a spontaneous rupture

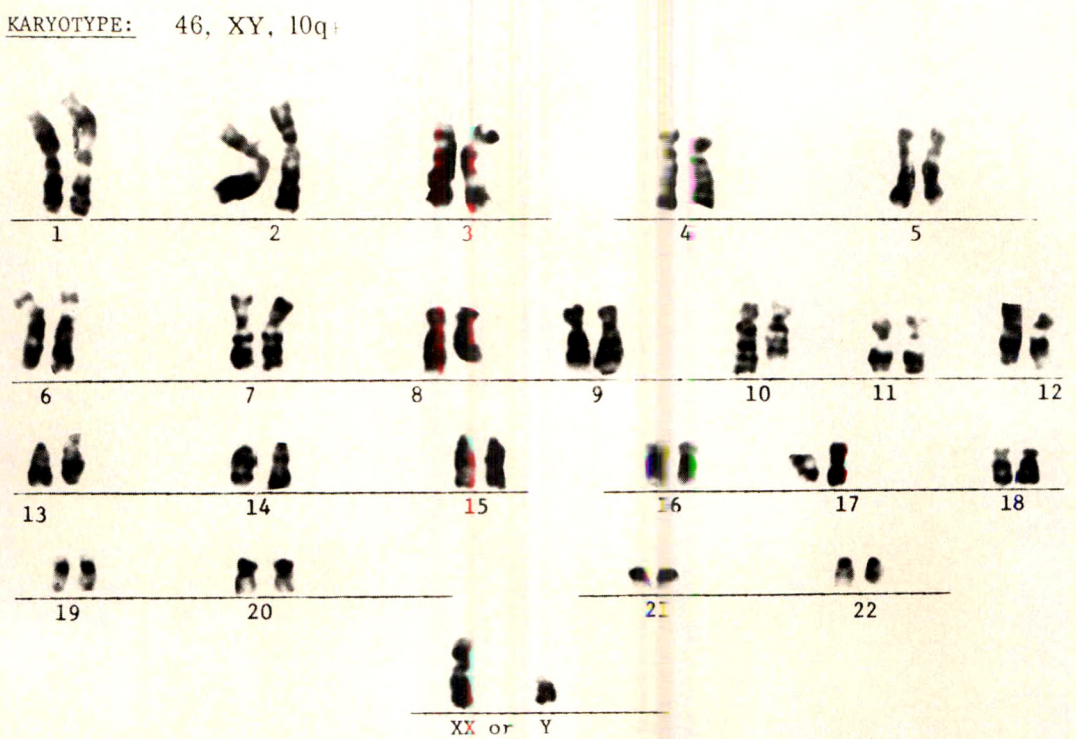


Fig. 3 (Schanzlin, Goldberg, and Brown). Karyotype of infant showing extra chromosomal material in the long arm of the tenth chromosome.

and reabsorption of the congenital cataract. Srivastava, Jain, and Nema[7] in 1966 reviewed the 28 previously reported patients with Hallermann-Streiff syndrome and added one of their own. These authors found dyscephaly with a bird-like face to be uniformly present; however, each of the other abnormalities, though found in the majority of patients, was not necessarily present in all.

The infant described here has many of the features of Hallermann-Streiff syndrome, including bird-like face, hypotrichosis, frontal alopecia, and dental anomalies. Additionally, he has bilateral buphthalmos with congenital glaucoma, complete scleralization of both corneas, and total aniridia.

Although the family histories in most of the cases of Hallermann-Streiff syndrome are incomplete, the syndrome is thought to be the result of a sporadic mutation. Consanguinity has been reported in two cases,[8,12] but the precise blood relationship in these cases was not documented. The infant described here is the product of a consanguinous union between distant cousins and is himself a distant cousin of a 30-year-old man with the classic findings of Hallermann-Streiff syndrome. This suggests a recessive mode of inheritance of the syndrome in this family.

Few patients with Hallermann-Streiff syndrome have had chromosome karyotyping. Normal sex chromatin was found by Falls and Schull,[6] as well as by Bonamour and Leopold.[9] Normal karyotypes were found in the one case of three tested by Hoefnagel and Benirschke[10] and in the case reported by Spoerl and Romano.[12] Carones[1] reported cytogenic studies of facia lata cultures and found a possible

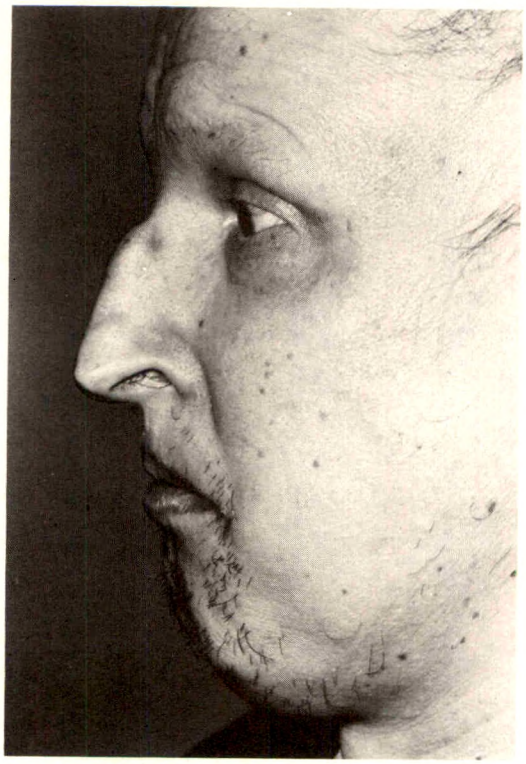


Fig. 4 (Schanzlin, Goldberg, and Brown). Cousin of infant in Figure 2, with parrot-beaked nose, micrognathia, microcornea, and a history of aspirated bilateral cataracts.

discrepancy in the size of two members of a pair of D chromosomes. Chromosome studies of our infant revealed a normal number of autosomes and a normal XY grouping, but demonstrated an elongation of chromosome 10. Chromosome studies of both parents and the affected cousin with Hallermann-Streiff syndrome showed normal karyotypes.

The chromosomal abnormality in this infant occurred as a result of a spontaneous mutation of the tenth chromosome, resulting in a partial duplication 10q+ syndrome. Other patients with partial duplication 10q+ syndrome have had microcephaly, small palpebral fissures, a small nose with antiverted nostrils, micrognathia, and malformed, low-set ears.[13] These patients often have anomalies of the digits and abnormal skin creases.

The bifid great toes and ulnar skin creases observed with this infant may be a forme fruste of the 10q+ chromosomal abnormality. The relationship of this chromosome abnormality to the typical physical findings of Hallermann-Streiff syndrome in this patient is unclear. It may be that the genetic abnormality responsible for the features of Hallermann-Streiff syndrome coupled with partial duplication 10q+ syndrome resulted in the unusual findings of buphthalmos, sclerocornea, and aniridia seen in this infant.

Summary

An infant with the features of Hallermann-Streiff syndrome also had the previously unreported ocular signs of sclerocornea, buphthalmos, congenital glaucoma, and aniridia. Chromosome analysis revealed an elongation of one of the arms of the tenth chromosome. The infant was the product of a consanguinous marriage between distant cousins and a distant cousin of the patient also had the typical features of the Hallermann-Streiff syndrome.

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Fifty years ago this month in THE JOURNAL:

The American Board for Ophthalmic Examinations has been in operation for fourteen years, and during that time has issued certificates to nearly nine hundred ophthalmologists. Such certification, together with the incidental examinations, is playing an important part in the development of ophthalmology in this country, but it is only one part of the work originally contemplated for the Board. A more far-reaching function will be the definite standardization of ophthalmic teaching. Much preliminary work is necessary before effective steps can be taken in this direction, and the corrective influences of time and even of what may be termed experimentation will be called into play before adequate processes are developed.

E. C. Ellett, *The American Board for Ophthalmic Examinations* *Am. J. Ophthalmol.* 13:824, 1930

VARIATIONS IN EXTRAOCULAR MUSCLE NUMBER AND STRUCTURE IN CRANIOFACIAL DYSOSTOSIS

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Major ophthalmologic findings in patients with craniofacial dysostosis (Crouzon's disease) consist of shallowing of the orbits with exorbitism, orbital divergence, exotropia, disk edema, optic atrophy, iris and choroidal colobomas, nystagmus, congenital cataract, and retinal detachment.[1-6] Many of these problems occur in relation to stenosis of the cranial sutures and cranial base deformities, but the cause of the intraocular lesions remains unknown.

Suggested explanations for abnormal ocular alignment in the syndromes of premature cranial bone stenosis (craniofacial dysostosis, acrocephalosyndactyly, oxycephaly, and others) include abnormal extraocular muscle vectors of action, altered orbital distance and angulation, and involvement of cranial nerves.

Yet another possible explanation for ocular misalignment is structurally abnormal or absent specific extraocular muscles. Absence of the superior rectus muscles has been reported in two patients with acrocephalosyndactyly (Apert's syndrome) by Weinstock and Hardesty[7] and in one patient by Cuttone and associates,[8] but the frequency of this anomaly in a large series of patients has not yet been determined.

A multispecialty team at the Children's Hospital of Philadelphia examined 154 patients with major craniofacial anomalies and observed the varying frequencies of strabismus (Table). Of 36 patients with craniofacial dysostosis, 12 required extraocular muscle surgery and five of 12 (42%) had extraocular muscle anomalies. No other patients were noted to have such anomalies at the time of surgery.

We studied the five patients with craniofacial dysostosis and strabismus who had anomalies in the number and structure of the extraocular muscles.

CASE REPORTS

Case 1—The patient, first examined in October 1975 at the age of 16 years, had craniofacial dysostosis and residual exotropia. He had had exotropia surgery elsewhere at the age of 13 years. Visual acuity was 6/18 (20/60) in the right eye and 6/12 (20/40) in the left eye with a severe myopic and astigmatic correction in both eyes. Evaluation of motility disclosed alternating exotropia of 20.0 prism diopters in all horizontal fields of gaze with absent elevation in abduction in both eyes.

We performed a bilateral medial rectus muscle resection. Careful exploration of the scleral surfaces disclosed the complete absence of superior rectus muscles bilaterally. Postoperative alignment was excellent, with persistent bilateral absence of elevation in abduction.

Case 2—The patient, first examined in December 1979 at the age of 1 year, had craniofacial dysostosis and a history of anterior craniectomy at the age of 2 weeks and ventriculoperitoneal shunt at the age of 2 months. Visual acuity was central, steady, and maintained in both eyes. Motility examination disclosed intermittent exotropia of 45.0 prism diopters at fixation distances of 6 m and 33 cm. We also noted a "V" pattern.

When he was 13 months old, we performed a bilateral lateral rectus muscle recession with superior movement of the tendons one-half tendon width. Careful examination of the scleral surfaces disclosed the complete absence of the superior rectus muscles in both eyes. Postoperative motility evaluation disclosed residual exotropia and persistent absence of elevation in abduction.

Case 3—The patient, first examined in August 1978 at the age of 10 months, had craniofacial

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TABLE

PATIENTS WITH CRANIOFACIAL ANOMALIES

Anomaly	No.	Strabismus	Strabismus Surgery
Craniofacial dysostosis (Crouzon's disease)	36	18	12
Isolated craniofacial dysostosis	33	2	0
Hypertelorism, pure	21	8	3
Hemifacial microsomia	15	4	1
Acrocephalosyndactyly (Apert's syndrome)	13	7	4
Mandibulofacial dysostosis (Treacher Collins)	7	2	0
Assorted (oxycephaly, kleeblatschädel syndrome, etc.)	29	12	8
Total	154	53	28

dysostosis. Visual acuity corrected for hyperopic astigmatism was central, steady, and maintained in both eyes. A motility examination disclosed 50.0 prism diopters of esotropia in the left eye with inability to abduct the eye past the midline and a marked overelevation in adduction of the right eye. The diagnosis of left sixth nerve palsy was considered.

When he was 12 years old, we performed a large left medial rectus muscle recession and a large right inferior oblique muscle recession. At surgery, exploration of scleral surfaces disclosed the absence of the right inferior rectus muscle. The left medial rectus muscle was tight, and forced abduction of the left eye was limited to the midline; structurally, the left lateral rectus muscle appeared normal. Postoperative cosmetic results were excellent.

Case 4—The patient, first examined at the age of 14 years in March 1975, had craniofacial dysostosis, hypoplastic optic nerves, posterior keratoconus, and severe myopia. Visual acuity with myopic correction was 6/60 (20/200) in both eyes at 6 m and 33 cm. A motility examination disclosed esotropia of 45.0 prism diopters and hypotropia of 30.0 prism diopters in the left eye and overelevation in adduction in both eyes.

In January 1976, we performed a large bilateral medial muscle recession, a moderate bilateral inferior oblique muscle recession, and a resection of the right inferior rectus muscle. At surgery, careful exploration disclosed the absence of the superior oblique tendon and superior rectus muscle of the left eye. We also noted a peculiar continuous band of tissue, extending clockwise from the superior border of the lateral rectus muscle to the superior border of the medial rectus muscle of the left eye, incorporating scattered skeletal muscle fibers and probably representing "muscularized" intermuscular septum. In the right eye, the sole anomaly was the absence of the superior oblique tendon. After two further procedures, the eyes remained in a straight position, but limitation of elevation in abduction in both eyes persisted.

Case 5—The patient, examined at the age of 1 year in July 1977, had craniofacial dysostosis and blepharoptosis in both eyes. Visual acuity was central, steady, and maintained in both eyes. A motility examination disclosed alternating esotropia of 30.0 prism diopters with overelevation in adduction in both eyes.

In August 1979, we performed a bilateral medial rectus muscle recession. Careful examination disclosed the absence of recognizable inferior oblique muscles bilaterally. Scattered skeletal muscle fibers running along the normal course of the inferior oblique muscle were interspersed in intermuscular septum in the inferotemporal quadrant. We did not explore for innervation to these fibers. These tissues were transected. Postoperatively, primary position alignment was excellent but overelevation in adduction persisted in the right eye alone.

DISCUSSION

The frequency of anomalous number and structure of the extraocular muscles in the syndromes of premature stenosis of cranial bones is unknown. Our five patients represented almost one half of those with craniofacial dysostosis who had ocular muscle surgery during the seven years' existence of the craniofacial team at the Children's Hospital of Philadelphia. Since patients with mild cases of this disorder may not visit an ophthalmologist and those with less noticeable dysfunctions (such as limitation of upgaze) may not wish to undergo surgery, it is difficult to relate our figures to the actual incidence of this anomaly in the total population of those with craniofacial dysostosis.

We used diagonally placed cul-de-sac incisions through the conjunctiva and the Tenon capsule in each eye, either in-

feronasal and superotemporal, or superonasal and inferotemporal, depending on the planned procedure. This permits access to all six muscles, add just a few minutes to surgical time, and adds little risk to the procedure. We isolate each muscle in turn, and examine its insertion; we do not cut the Tenon capsule or intermuscular septum surrounding the muscles. One suture, if necessary, may be used to close the conjunctival incision.

Before the advent of the fornix approach to the extraocular muscles, investigating the muscles was a major undertaking, requiring 360-degree peritomy or separate incisions through the conjunctiva and the Tenon capsule near each muscle. Vertical and oblique muscle surgery was less common in the past (and the majority of anomalies in this report concern these muscles). Therefore, reports of extraocular muscle anomalies in patients with craniofacial abnormalities have been infrequent. Modern cranioplastic and neurosurgical techniques, however, enable us to perform elective strabismus surgery and investigate muscle anomalies.

The presence of anomalies of extraocular muscle number and structure only in patients with craniofacial dysostosis in this series of 154 patients may be explained by the small number of patients with other craniofacial anomalies undergoing strabismus surgery, or it may be an intrinsic feature of this syndrome. The absence of the superior rectus muscles in both eyes of three patients with acrocephalosyndactyly[7,8] supports the former explanation, but the origin of these muscle anomalies remains poorly understood. Perhaps gross abnormalities of the size and position of the orbits in these patients result in anomalous muscle development.

The levator palpebrae superioris muscle differentiates from the medial aspect of the superior rectus muscle during the 22- to 30-mm stage of embryogenesis.[8] It is intriguing that we noted no blepharoptosis in any of the patients lacking the superior rectus muscle, nor was levator function decreased. This implies secondary atrophy or abiotrophy of the superior rectus muscles after levator muscle differentiation has occurred.

As functional potential for patients with syndromes of premature orbital bone stenosis improves, more of them will undergo strabismus surgery. Investigation of extraocular muscle number and structure anomalies will improve both our understanding of the frequency and variety of these disorders and our ability to compensate for them surgically.

Summary

Five of 12 patients with craniofacial dysostosis who needed strabismus surgery had anomalies of extraocular muscle structure and number. Two cul-de-sac incisions per eye are sufficient to investigate all muscles for anomalies and add little time or risk to the planned strabismus procedure. The origin of these anomalies and their frequency in craniofacial stenosis are unknown.

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NOTES, CASES AND INSTRUMENTS

PREPAPILLARY HEMORRHAGE AND PERSISTENT HYALOID ARTERY

WILLIAM V. DELANEY, JR., M.D.
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The embryonic hyaloid artery appears at the fifth week of gestation and reaches the lens to complete the tunica vasculosa lentis by the 11th week. From that time on, its functions are gradually taken over by other vessels and its atrophy is usually complete before parturition. It is not uncommon to find remnants of the hyaloid system[1,2] in the form of Bergmeister's papilla, a Mittendorf dot, or a hyaloid vessel. The presence of active blood flow in the postpartum hyaloid system is rare. Hemorrhage from a hyaloid artery has been said to occur.[1] I could not discover a direct reference or illustration, however. This study deals with spontaneous prepapillary hemorrhage in a young man with bilateral hyaloid artery remnants.

CASE REPORT

A 20-year-old man awoke the day before examination with a "feather-like" opacity obscuring vision in his right eye. He had had congenital esotropia and rotary nystagmus, and corrective surgery was done on the left eye in the first year of life. His first reliable visual acuity was 6/7.5 (20/25) in the right eye and 6/24 (20/80) in the left eye. His general health had been good and there was no family history of ocular disease other than senile cataracts.

Visual acuity on admission was 6/7.5 (20/25) in the right eye and 6/21 (20/70) in the left. Superior visual field in the right eye was limited. The intraocular pressure was 15 mm Hg bilaterally (applanation). Corneal diameter was 12 mm bilaterally. A remnant of the hyaloid artery system could be followed from the disk to lens in both eyes (Fig. 1). A blood clot obscured the disk and extended into the vitreous inferiorly in the right eye (Fig. 2). I found no peripheral retinal abnormalities, retinal folds, or points of traction. Aside from the hyaloid remnants, there were no abnormalities in the left eye.

During the next ten days the blood clot spread out slightly (Fig. 3) and the intravitreal blood partially cleared. The center of the clot thinned out, exposing a circular yellow defect flecked with pigment. Visual acuity was maintained at 6/6-2 (20/20-2) (Fig. 4). During the next five weeks the clot hemolyzed. Lines in the retina extended in all directions from the disk and involved the fovea although the visual acuity was 6/6-3 (20/20-3). No vitreous separation occurred throughout the course. The patient was lost to follow-up.

DISCUSSION

If the causes of intravitreal bleeding were ranked, persistent hyaloid vessels would be near the bottom of the list, judging from the scarcity of reports. I could not determine whether the hemorrhage described in my patient resulted from traction on disk vessels or rupture of a still patent hyaloid vessel. The hyaloid vessel appeared to be intact. The mild nature of the hemorrhage suggests it was from the venous rather than arterial circulation. The hyaloid vessel in the other eye was attached to the central retinal vein. Unfortunately the patient refused fluorescein angiography.

Surgeons are now treating anterior and posterior persistent hyperplastic primary vitreous with or without falciform folds with some success.[3,4] Persistent hyaloid vessels are a frequent finding in these developmental diseases[5-7] and must be considered possible complicating factors at surgery. They need to be kept in mind as a rare cause of intravitreal hemorrhage in the young patient.

Late manifestations should be recorded if the patient again seeks ophthalmologic attention.

From the Eye Research Institute of Central New York, Syracuse, New York. Supported in part by the Eye Defect and Vision Research Foundation.

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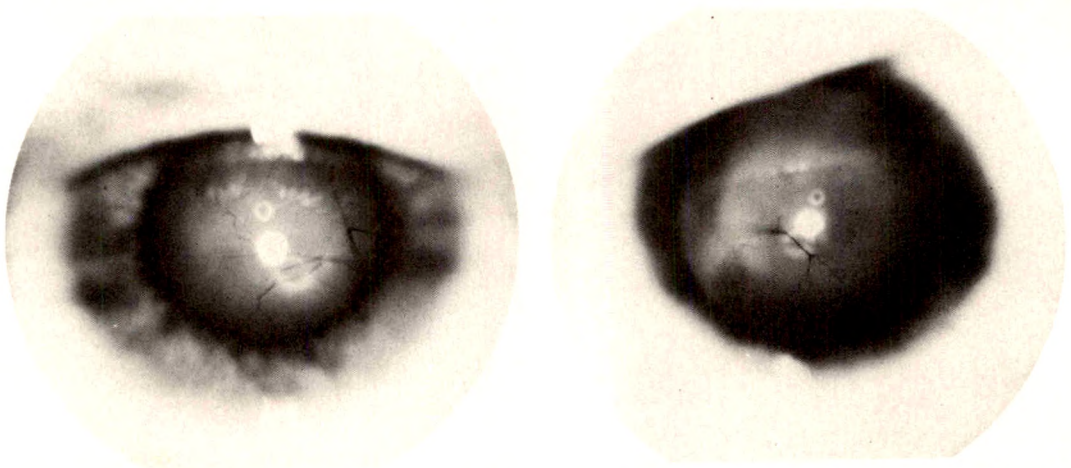


Fig. 1 (Delaney). Persistent hyaloid artery and partial tunica vasculosa lentis in the right (left) and left (right) eyes.

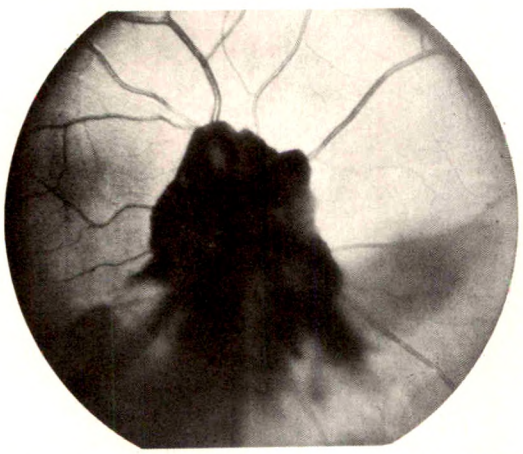


Fig. 2 (Delaney). Prepapillary hemorrhage and intravitreal blood in the right eye at first examination.

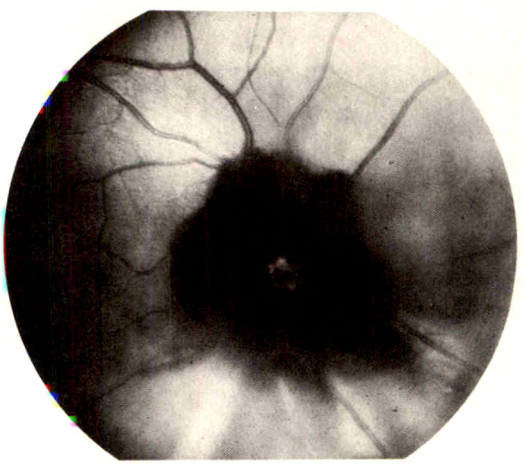


Fig. 3 (Delaney). Right eye ten days later.

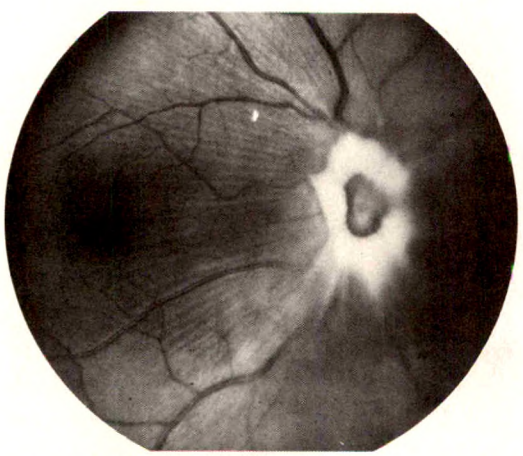


Fig. 4 (Delaney). Right eye 38 days later.

Summary

A patient with bilateral persistent hyaloid vessels suffered a prepapillary and intravitreal hemorrhage.

The clot hemolyzed and lines in the retina extended in all directions, involving the fovea. There was no vitreous separation and visual acuity was 6/6-3 (20/20-3). The patient was lost to follow-up shortly thereafter. Although persistent hyaloid vessels rarely cause intravitreal hemorrhage, they are often found in cases of persistent hyperplastic primary

vitreous and must be considered possible complicating factors at surgery.

ACKNOWLEDGMENTS

John Cetner, M.D., provided historical data and William Foglan, M.D., referred the patient.

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A FIXATION DEVICE FOR ENUCLEATED EYES

GLENN L. WING, M.D., AND JOHN J. WEITER, M.D.
Boston, Massachusetts

A common problem encountered during the pathologic examination of an enucleated eye and during practice surgery is obtaining adequate stabilization of the globe.

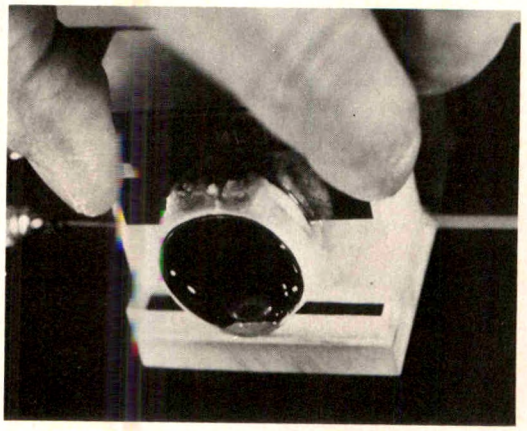


Fig. 1 (Wing and Weiter). Ophthalmic fixation device.

Pathologic preparation of a fixed eye involves the sectioning of the specimen into calottes. The usual pupil-optic nerve section requires two cuts through the globe. Often, the globe distorts and collapses during this procedure, resulting in a poorly cut section.

To help alleviate these problems, we have devised an ophthalmic fixation device. It is made from a methylmethacrylate block, into which a cup is moulded and lined with a silicone gasket. Two 14-gauge needles are inserted through the plastic block into the cup (Fig. 1).

One third of the globe sits in the cup and is held firmly when suction is applied through the two needles. Added to the cup is a Plexiglass cutting block on which the eye is sectioned (Fig. 2).

A Plexiglass strip attached to the un-

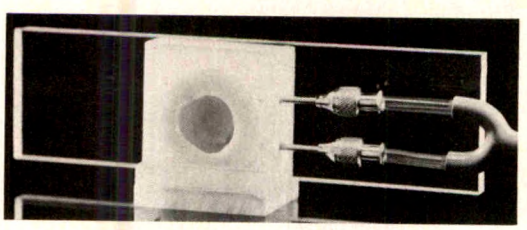


Fig. 2 (Wing and Weiter). Sectioning of the eye while it is held firmly in the cup with suction.

From the Department of Ophthalmology, Tufts University School of Medicine, New England Medical Center Hospital and the Boston Veterans Administration Medical Center, Boston, Massachusetts. This work was supported in part by National Eye Institute Grants 5 TO1 EY00054-08 (Dr. Wing), RO1 EY02246, and RO1 EY02572 (Dr. Weiter).

Reprint requests to Glenn L. Wing, M.D., Department of Ophthalmology, Tufts-New England Medical Center Hospital, 171 Harrison Ave., Boston, MA 02111.

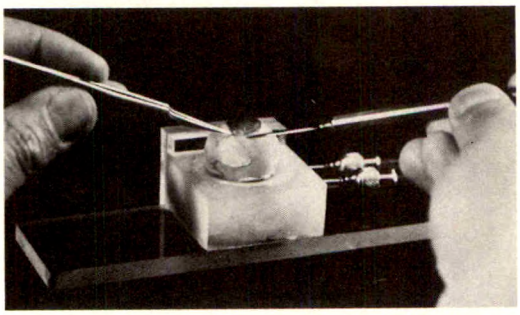


Fig. 3 (Wing and Weiter). Stabilized eye for practice surgery.

dersurface of the cup acts as a base when the cup and eye are turned upwards and used for practice surgery (Fig. 3).

By releasing the suction, one can easily remove the remaining ocular specimen from the cup.

Summary

We have constructed an ophthalmic fixation device that stabilizes an enucleated eye, and helps both in the pathologic examination of the eye and in ophthalmic practice surgery.

Acknowledgment

Otie Thomas helped construct the device.

MEDIAL RECTUS MUSCLE PALSY AFTER DENTAL ANESTHESIA

E. Anthony Petrelli, M.D., and Rodney E. Steller, D.M.D.

New Haven, Connecticut

Because of the complicated vascular, neurological, and lymphatic connections between the maxilla and the orbit, it is possible that conditions in one area affect the other. We report herein an ocular complication of local dental anesthesia.

From the Department of Ophthalmology and Visual Science, Yale University School of Medicine, and the Department of Ophthalmology, Hospital of St. Raphael, New Haven, Connecticut (Dr. Petrelli).

Reprint requests to E. Anthony Petrelli, M.D., 455 Orange St., New Haven, CT 06511.

Case report

A 42-year-old woman with a history of cardiac arrhythmia and controlled hypertension and a dental history of caries, periodontitis, and posterior bite collapse, underwent a supraperiosteal injection to anesthetize the middle superior alveolar nerve (Fig. 1). The dentist injected 1.8 ml of a 3% solution of mepivacaine HCl without vasoconstriction. The dentist used a 27-gauge needle and carefully aspirated the area to prevent intravascular injection. The patient was to have a crown preparation on the maxillary right first premolar tooth. Within five minutes of the injection, the patient experienced the onset of diplopia. Vital signs remained stable, and diplopia subsided within 90 minutes. On her next visit, she experienced the same amount and duration of diplopia after the same anesthetic injection. After her third dental treatment, the diplopia recurred and was photographed before it subsided (Fig. 2). A transient right medial rectus muscle palsy was evident. There were no sequellae.

Discussion

Brain[1] reported a permanent partial third nerve palsy occurring after removal of a right upper lateral incisor. The relationship between the tooth extraction and the palsy was not definitely established. Cooper[2] described a 46-year-old woman who had a right lower mandibular block followed in three minutes by loss of abduction of the right eye, which lasted for five hours. He postulated that the anesthetic solution entered the inferior alveolar artery and by back pressure entered the internal maxillary artery, and then the middle meningeal artery which supplies a part of the sixth cranial nerve.



Fig. 1 (Petrelli and Stellar). Position of needle in a supraperiosteal injection.

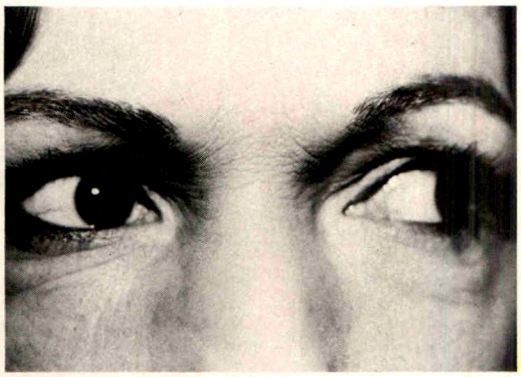


Fig. 2 (Petrelli and Stellar). Patient five minutes after maxillary block. Note partial right medial rectus muscle palsy.

He doubted that a direct injection occurred into the cranial cavity.

Blaxter and Britton[3] described three patients with ocular complications of dental anesthesia, all of whom received mandibular blocks. The first patient had a complete amaurosis along with loss of adduction for 20 minutes. The second patient also had a transient amaurosis. The third patient, a 30-year-old ophthalmologist, experienced a transient lateral rectus palsy muscle that almost caused an automobile accident when he left the dentist's office. Blaxter and Britton believed that embolism was unlikely because an oily solution was not used. They also postulated that the cause of the complication was an intra-arterial injection via the inferior alveolar artery to the maxillary artery and perhaps an anomalous supply to the orbit through the superior orbital fissure.

Rood[4] reported a case of inferior alveolar block followed by immediate blepharoptosis and ipsilateral "medial strabismus" lasting up to 45 minutes. Goldberg[5] described a 36-year-old woman who underwent an upper left molar extraction under local mepivacaine anesthesia. Within a few days of this treatment she experienced an abduction defect of the left eye associated with a myositis of the left medial rectus muscle, possibly caused by an immune response to mepivacaine anesthesia. In attempting to abduct the left eye, she developed a vertical pendular nystagmus.

In our patient, the partial third nerve palsy occurred immediately after an ipsilateral maxillary block with 3% mepivacaine without vasoconstriction on three separate occasions. In contrast, most reported cases have been associated with a mandibular block; presumably, an inadvertent intravascular injection into the inferior alveolar artery occurred. Careful aspiration before instillation ruled out intravascular injection in our case. Possibly the anesthetic entered the orbit directly through a defect in the bony wall in the maxillary sinus. Doggarty's[6] opinion was that communication between the maxilla and the orbit could take place by direct extension through bone and other connective tissue, along ramifications of nerves, or by veins and lymphatic vessels.

Although rare, ocular complications of local dental anesthesia do occur and in some cases can lead to permanent extraocular palsies[1] and even amaurosis.[7] Both the dentist and the ophthalmologist should be aware of this phenomenon.

Summary

A 42-year-old woman developed a transient partial third cranial nerve palsy after a supraperiosteal injection in the area of the middle superior alveolar nerve with mepivacaine HCl for dental anesthesia. This effect may have been caused by direct extension through a bony defect rather than by inadvertent intravascular injection of the anesthetic. Dentists and ophthalmologists should be aware of this phenomenon, which can result in permanent ocular palsy and even amaurosis.

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A NEW 20-GAUGE INTRAVITREAL CRYOPROBE

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Boston, Massachusetts
AND
WAYNE E. FUNG, M.D.
San Francisco, California

Cryotherapy during pars plana vitrectomy is a useful technique for treating posterior retinal tears and for the extraction of dislocated lenses or intraocular foreign bodies. It is also useful for softening a hard nucleus before phacofragmentation.[1] There are several intraocular cyroprobes available today. These have certain disadvantages when used with the Ocutome-Fragmatome system, since they are larger in diameter than the 20-gauge instruments, requiring a larger incision which subsequently leaks. The speed of the freeze-and-defrost cycle on these instruments is also imperfect.

In conjunction with the Frigitronics Company, we designed and tested a new intravitreal 20-gauge (0.89 mm) cryoprobe which has a rapid freeze-and-defrost capability (Figure). The probe is compatible with the standard Frigitronics cryotherapy console, operating with nitrous oxide gas. The length of the intraocular shaft is 35 mm, allowing it to be used in any part of the eye. Only the tip of the probe freezes. The iceball can be regulated in size by varying the pressure of the nitrous oxide gas at the console. There is an extremely rapid freeze-and-defrost cycle, so that the actual application of the iceball can be done rapidly, without prolonged contact with the retina.

We have used this intravitreal cryoprobe on several patients and found it to be excellent in regard to ease of insertion and removal from the pars plana sclerotomy sites. The freeze-and-defrost cycle is extremely rapid and reliable and the iceball has been of appropriate size for the treatment of posterior retinal breaks. It is also useful to insert the probe into the center of a hard, sclerotic nucleus before fragmentation. The iceball produces a softening of the hard lens nucleus, which makes subsequent phacofragmentation and removal much easier.

The size of the iceball can be varied with the console pressure. At a pressure of 500 psi, a 2.5 × 4-mm iceball forms with a 30-second freeze. At a pressure of 600 psi, a 4 × 5-mm iceball forms with a 30-second freeze. In actual practice during vitreous surgery, we have found that a pressure of 475 psi produces an iceball approximately 2 mm in diameter only at



Figure (Bradbury and Fung). The 35-mm, 20-gauge intravitreal probe. Only the tip of the probe freezes.

From the William P. Beetham Eye Unit, Joslin Clinic, Joslin Diabetic Foundation, Boston, Massachusetts (Dr. Bradbury); and the Department of Ophthalmology, Pacific Medical Center, San Francisco, California (Dr. Fung).
Reprint requests to Michael J. Bradbury, M.D., 1 Joslin Place, Boston, MA 02215.

the tip of the probe, and that there is a rapid freeze and defrost. When the lens nucleus is frozen before phacofragmentation, the probe is inserted into the center of the nucleus and a large iceball is allowed to remain for ten or more seconds. This maneuver is repeated several times and some time then allowed for the nuclear lens fibers to hydrate and soften. Experimental evidence for this has been published.[2]

Summary

A new intraocular 20-gauge (0.89-mm) cryoprobe is compatible with the Ocutome-Fragmatome system. The iceball forms only at the tip and there is a rapid freeze and defrost cycle. Actual clinical testing during vitreous surgery showed the probe to be reliable and easy to use. It is useful for the treatment of posterior tears, the removal of dislocated lenses, and softening the lens nucleus before phacofragmentation.

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CARDIOPULMONARY ARREST AFTER RETROBULAR BLOCK

Richard M. Rosenblatt, M.D., Donald R. May, M.D., and Krikor Barsoumian, M.D.

Davis, California

Ophthalmic surgeons often choose local anesthesia rather than general anesthesia to minimize anesthesia-induced complications and mortality. Local anesthesia, however, also has inherent risks and complications. We studied a case of local anesthetic toxicity which occurred immediately after a retrobulbar injection and which resulted in a cardiopulmonary arrest with a benign outcome.

Case Report

A 61-year-old woman was scheduled for the removal of a cataract. We selected local anesthesia for the surgery because the patient had many medical problems adult-onset diabetes mellitus with diabetic retinopathy and chronic renal failure, arteriosclerotic heart disease with hypertension and chronic congestive heart failure, and sideroblastic anemia. She took digoxin on a regular basis; 0.25 mg every other day; methyldopa, 250 mg twice each day; furosemide, 120 mg three times each day; and isophane insulin suspension 7 units, each morning. Pertinent preoperative laboratory values included the following: hematocrit, 25%; blood urea nitrogen, 101 mg/100 ml; serum creatinine, 5.3 mg/100 ml; albumin, 2.9 g/100 ml; total protein, 5.1 g/100 ml; calcium, 8.5 mEq/liter; phosphorous, 5.8 mEq/liter; and glucose, 163 mg/100 ml; and electrolyte values were normal.

The patient was brought to the operating room before she received any medication. There we noted that her blood pressure was 190/100 mm Hg compared to her previous blood pressure of 160/90 mm Hg. We attributed this to her apprehension and she was sedated slowly with 5 mg of intravenously administered diazepam. Her blood pressure, however, remained high.

We performed a retrobulbar block using a 23-gauge, 1½-inch retrobulbar needle. After the needle had been inserted into the orbit the piston of the syringe was withdrawn to ensure that a vessel had not been entered and no blood return into the syringe was noted. We then injected 2 ml of an anesthetic solution by mixing 10 ml of 0.5% bupivacaine HCl, 10 ml of 2% mepivacaine HCl, and a 1-ml ampule of hyaluronidase (150 units). The patient abruptly lose consciousness and became apneic. We noted asystole on the electrocardiogram, which persisted for about 15 seconds, followed by a sinus tachycardia of 140 beats per minute. Her blood pressure, initially unobtainable, was 210/100 mm Hg. We immediately performed a tracheal intubation and began mechanical ventilation. We then gave her incremental doses of 50 μg of nitroglycerin intravenously to maintain the systolic blood pressure in the range of 150 to 200 mm Hg. We gave her a 25-g ampule of dextrose because we presumed that the syncope was caused by insulin-induced hypoglycemia. There was no response.

The patient remained unconscious for 20 minutes. During this time, she was unresponsive to pain, had no reflexes, and was apneic. Vital signs, aside from the hypertension, were stable. The presumptive

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diagnosis was that an inadvertant intravascular local anesthetic injection had occurred which spread directly to the brain. A spinal tap was performed 15 minutes into the episode to obtain cerebrospinal fluid for later quantitation of local anesthetic levels. Gas chromatographic assay revealed only the presence of mepivacaine HCl, 2.5 μg/ml. We performed an ophthalmoscopic examination before the spinal tap to exclude the possibility of occult intracranial hemorrhage with increased intracranial pressure. We saw no papilledema.

Twenty minutes after the retrobulbar block, the patient began to breathe spontaneously and slowly regained consciousness. Shortly thereafter the endotracheal tube was were removed. Neurologic examination at this time disclosed only weakness in all extremities and hyperreflexia. There were no sensory deficits except for ocular anesthesia consistant with the retrobulbar block. She had no recall of the incident nor of the retrobulbar block. These neurologic deficits resolved completely over the next two hours.

No adverse sequelae resulted from the cardiopulmonary arrest. Her postoperative electrocardiogram was unchanged and cardiac isoenzymes were normal. She was discharged one week later. The cataract extraction will be performed at a later date.

Discussion

The toxic effects of local anesthetics are well known.[1] Inadvertant vascular injection usually produces a characteristic response—a generalized convulsion. We noted no seizure but did note loss of consciousness, apnea, and asystole. The marked difference in the toxic response that occurred in our case can be attributed to the fact that the local anesthetic was injected into the cerebral circulation and not the systemic circulation. The arterial blood supply to the eye is from the opthalmic artery. An injection of local anesthetic into the opthalmic artery or one of its tributaries theoretically could produce retrograde flow of the local anesthetic up to the point where the ophthalmic artery meets the internal carotid artery. Antegrade flow of the local anesthetic in the internal carotid artery would then follow. We presumably witnessed such a phenomenon with local anesthetic being carried via the opthalmic and internal carotid arteries to the thalamus and other midbrain structures. Elliot, Quilici, and Elison[2] have shown experimentally that direct deposition of local anesthetic to the brain results in electrical silence. This would account for the apnea and obtundation. The transient asystole can be explained by the abrupt loss of autonomic tone with subsequent compensation, hence the post-asystole tachycardia and rebound hypertension.

Similar atypical distributions of anesthesia after an inadvertant arterial injection of local anesthetic have been reported. Schwartz, May, and Shore[3] examined a patient with diplopia after an injection of lidocaine to the region of a right upper molar. They postulated retrograde flow into the inferior alveolar artery and thence to the nucleus of the sixth cranial nerve by way of the middle meningeal artery. Other authors have also reported cases involving various cranial nerves and even transient blindness after intraoral injections of local anesthetic.[4,5]

Our case is unusual in that we were confronted with a life-threatening complication. It nevertheless illustrates the safety of local anesthetics. If appropriate life-support measures are taken, a favorable outcome from such an incident can be anticipated.

Summary

A 61-year-old woman experienced a cardiopulmonary arrest immediately after the retrobulbar injection of a mixture of 2 ml of 0.5% bupivacaine HCl, 2% mepivacaine HCl, and hyaluronidase. The patient was immediately resuscitated but remained unconscious for a total of 20 minutes. All neurologic deficits resolved completely over the two hours following the cardiopulmonary arrest and no adverse sequelae were noted.

We assumed that this cardiopulmonary arrest was precipitated by the local anesthetic being transported via retrograde flow through the ophthalmic artery and

then by antegrade flow through the internal carotid artery to the thalamus and other midbrain structures.

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Ophthalmic Miniature

Add to all these improvements backwards, another modern fancy, that grey printing is more beautiful than black. Hence the English new books are printed in so dim a character as to be read with difficulty by old eyes, unless in a very strong light, and with good glasses. Whoever compares a volume of the Gentleman's Magazine, printed between the years 1731 and 1740, with one of those printed in the last ten years, will be convinced of the much greater degree of perspicuity given by black than by the grey. Lord Chesterfield pleasantly remarked this difference to Faulkener, the Printer of the Dublin Journal, who was vainly making encomiums on his own paper, as the most complete of any in the world. 'But, Mr. Faulkener,' says my lord, 'don't you think it might be still farther improved, by using paper and ink not quite so near of a colour?'—For all these reasons I cannot but wish that our American printers would, in their editions, avoid these fancied improvements, and thereby render their works more agreeable to foreigners in Europe, to the great advantage of our bookselling commerce.

Benjamin Franklin In *The English Language*,
Edited by W. F. Bolton
Cambridge, Cambridge University Press, 1966

MEETINGS, CONFERENCES, SYMPOSIA

EDITED BY THOMAS CHALKLEY, M.D.

OXFORD OPHTHALMOLOGICAL CONGRESS 1980 MEETING

The 65th annual meeting of the Oxford Ophthalmological Congress was held in the friendly confines of Balliol College and Somerville College July 6 to July 9, 1980, under the aegis of the Master Barrie R. Jones of London.

Most members of the Congress live in Balliol College and for a few brief days lead the life of college students, sleeping in the college dormitories and dining in the Hall. Members note carefully that the gates close each night at 12 midnight and those resident members who wish to stay out later must notify the night porter of their intentions. Rumor has it that in years past some members preferred rooms with windows opening on to Broad Street so that it was unnecessary to disturb the porter.

The meeting opened at Sunday evening dinner. This was followed by a reception in the old Common room of Balliol College at which time new members and members attending the Congress for the first time were introduced to the Master and the Council.

The scientific program, held in the large auditorium of the of the University laboratory of zoology, comprised three symposia. The first on pain in ophthalmology was chaired by Professor Allan Bird of London; vascular pain, neurogenic pain, ocular pain and inflammatory pain were discussed. The afternoon was devoted to a variety of papers, "Adenovirus keratoconjunctivitis in Bristol," "Evaluation of carotid stenosis," "Remyelination during optic nerve compression," and "Traumatic chorioretinopathy."

Tuesday morning a modified intraocular irrigating fluid was described and then the difficulty many patients have in complying with recommendations to instill drops in their eyes at regular intervals. Vitrectomy for severe diabetic disease was discussed. The Doyne lecture by Patrick Trevor-Roper told of the noysome cataract (noysome, "troublesome or annoying"). He related a history of ophthalmology that is destined to be a classic, bringing together old and new from literary, historical, and analytic sources.

That afternoon there was a symposium on the teaching of ophthalmology that considered all levels, undergraduate and graduate and the training of the mature ophthalmologist and the commitment of Britain to Third World ophthalmology.

Wednesday morning was devoted to a discussion of current problems with a long discussion on the management of problems of entropion, blepharoptosis, and postenucleation socket.

The annual banquet of the Congress is always crowded. Members and guests who may have missed the scientific side come from London and the surrounding countryside for the dinner.

The Council elected the following honorary members for their many contributions to ophthalmology: Dorothy Campbell of Coventry, Dame Ida Mann of Perth, Harold Ridley of London, and Maurice L. Whiting of London. They join the list of previously named honorary members: G. T. Willoughitby Cashell, Ian Frasier, Frank W. Law, and Frank W. Newell.

The 1981 Congress is scheduled for July 6-8, 1981. Information concerning the program may be obtained from N. R. Galloway, Honorary Secretary, 14 Park Terrace, The Park, Nottingham NG1 5DN England.

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ARE METASTATIC EVALUATIONS INDICATED BEFORE ENUCLEATION OF OCULAR MELANOMA?

The incidence of matastases at the time of diagnosis of uveal melanoma has been the subject of recent controversy. Zimmerman and associates[1-3] stated that no more than 0.5% of uveal melanomas metastasize by the time of first examination, and that two thirds of the deaths from metastatic disease after enucleation can be attributed to surgical dissemination of tumor cells. Manschot and von Peperzoel,[4] on the other hand, suggested that tumor seeding is an early, inevitable event in the progression of these tumors and is responsible for postoperative mortality from metastatic disease. We believe that the incidence of metastasized tumors at the time of diagnosis must be determined if we are to resolve the controversy surrounding the progression and treatment of uveal melanoma. A thorough evaluation to determine the presence or absence of metastases in patients with ocular melanoma in whom enucleation is contemplated is necessary to accomplish this objective.

Previous conclusions stressing the infrequency with which metastases occur in these patients have been based on retrospective analyses of previous pathology and autopsy series. It is our impression that preoperative evaluations for the presence of metastatic disease in these series were limited or not performed at all. We suggest that previous estimates of the incidence of metastatic disease at the time of diagnosis are only minimum estimates. Recent clinical experience suggests that the true incidence may be considerably higher. In two series by Robertson and Young, cited by Zimmerman and McLean,[3] metastases were observed at the time of diagnosis in 6% (four of 67) and 6.5% (eight of 122) of their cases.

Our clinical impression has been that an increasing number of patients with

TABLE

MEDICAL AND LABORATORY DATA ON SEVEN PATIENTS WITH METASTATIC UVEAL MELANOMA

Case No., Age (yrs), Sex	Ocular Symptoms	Systemic Symptoms	Cell Type	Liver-Spleen Scan	Liver Function Tests	Site of Metastases	Comments
1, 83, F	Yes	Yes	Mixed	+	Increased	Liver	Died 9 wks after diagnosis
2, 64, M	No	Yes	Epithelioid	+	Increased	Liver	Died 1 mo after diagnosis; metastases diagnosed before choroidal melanoma
3, 37, M	Yes	No	Mixed	+	Normal	Liver	Died 6 mos after diagnosis
4, 61, F	Yes	No	Mixed	Not done	Increased	Liver, lungs, subcutaneous	Died 2 days after admission; both primary choroidal melanoma and metastases diagnosed post mortem
5, 57, M	Yes	No	Epithelioid	Not done	Not done	Liver, kidney, bone marrow	Died 1 wk after enucleation; metastases diagnosed post mortem
6, 39, M	Yes	No	Spindle B	+	Normal	Liver	Alive 6 mos after diagnosis
7, 72, M	Yes	Yes	Mixed cell	Not done	Not done	Liver	Patient had globe-filling melanoma and massive hepatomegaly; died 1 yr later

metastatic uveal melanoma are being identified before enucleation, because patients are being examined for the presence of metastases preoperatively. Since 1976, we have had the opportunity to review the cases of seven such patients (Table 1). In four of these patients, there was no clinical suggestion of metastases at the time of diagnosis, although one developed acute abdominal symptoms three weeks later (Case 1). The other three patients had hepatomegaly. One of these had no ocular symptoms (Case 2) and was only later found to have primary choroidal melanoma. The majority of patients from whom this series was drawn were not systematically examined for the presence or absence of hidden metastases. Although we are reluctant to attempt statistical analysis of such a small group of patients these observations have prompted us to initiate an extensive cooperative review.

Several studies have indicated that the liver is usually the first, and often the only, site of metastases from uveal melanoma.[5,6] Extrahepatic metastases usually occur in conjunction with hepatic involvement. We suggest that every patient with ocular melanoma in whom enucleation is contemplated should undergo basic tests to determine the presence or absence of metastatic disease to the liver. A complete set of liver enzyme studies should be obtained on every patient. Patients with melanoma metastatic to the liver usually have increased serum alkaline phosphatase or lactate dehydrogenase levels.[7] Gamma-glutaryl transpeptidase may eventually prove to be more accurate in detecting liver involvement than either alkaline phosphatase or lactate dehyrogenase assay.[5] Although some authors believe that liver scanning is unwarranted in patients with normal liver-function enzymes,[7] the observation of abnormal liver scans in two of our patients with normal liver enzyme levels suggests that liver scanning and analysis of liver enzymes should be routinely performed on all patients. If these tests suggest the presence of metastases, a liver biopsy is indicated. Equivocal results may require the use of such invasive studies as hepatic arteriography or peritoneoscopy with direct visualization and biopsy of suspicious nodules.

The cost of obtaining routine liver enzyme studies and performing a liver scan is approximately $300. We believe these studies are in the patients' best interest as well as helpful in providing insight into the progression of this disease. For example, one of our patients was found to have two discrete metastatic nodules in association with a small choroidal melanoma. Despite these findings, the patient elected to undergo enucleation and local resection of the two nodules and intrahepatic artery chemotherapy. The prognosis is, of course, guarded, but the patient has shown no evidence of recurrence of metastases for eight months. Patients with painless eyes and extensive metastatic disease would be spared an unnecessary enucleation and possibly treated with palliative radiation as the situation requires. For those patients with blind, painful eyes or large tumors threatening to perforate the globe, enucleations could still be performed for palliation.

DANIEL M. ALBERT,
MICHAEL D. WAGONER,
AND MORTON E. SMITH

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CORRESPONDENCE

Letters to the Editor must be typed double-spaced on 8½ × 11-inch bond paper, with 1½-inch margins on all four sides, and limited in length to two manuscript pages.

An Easy Way To Convert Visual Acuity Measurements

Editor:

Visual acuities are increasingly being recorded metrically. Most practitioners, however, have been trained to think in terms of standard Snellen notation. Converting can be annoying and troublesome. I have discovered a "Rule of Three" to make conversions quickly and simply.

To convert Snellen to metric, simply multiply the denominator by 0.3. For example, to convert 20/70 to metric, multiply 70 by 0.3. The metric equivalent is 6/21.

To convert from metric to Snellen, divide the denominator by 3 and add a zero. For example, to convert 6/15 to Snellen, dividing 15 by 3 gives 5, and adding the 0 gives 20/50.

I hope "Peterson's Rule of Three" is as helpful to others as it has been to me.

W. SCOTT PETERSON, M.D.
Waterbury, Connecticut

A Slit Lamp Chest Shield

Editor:

A common problem encountered by ophthalmologists occurs during proper positioning of certain patients in front of the slit lamp. Even with the patient's chin and forehead correctly placed, the examiner may still encounter difficulties with slit-lamp focusing and applanation tonometry because of the forward protrusion of the patient's chest or abdomen onto the slit-lamp table. This often causes interference with and resistence to the lamp's moving stage.

To help overcome this problem, we devised a chest shield that attaches to our Zeiss slit lamps (Figure). The shield consists of ¼-inch Plexiglass, bent and shaped into a rectangular open box, and two large Plexiglass hooks that suspend it from the slit lamp's chin bar.

The chest shield permits the full and unrestricted forward excursion of the lamp's moving stage toward the patient, and acts as a barrier between the patient and the slit-lamp table.

The shield can be removed easily at the completion of the examination. It can be modified to fit any other type of slit lamp or fundus camera.

GLENN L. WING, M.D.
JOHN J. WEITER, M.D.
Boston, Massachusetts

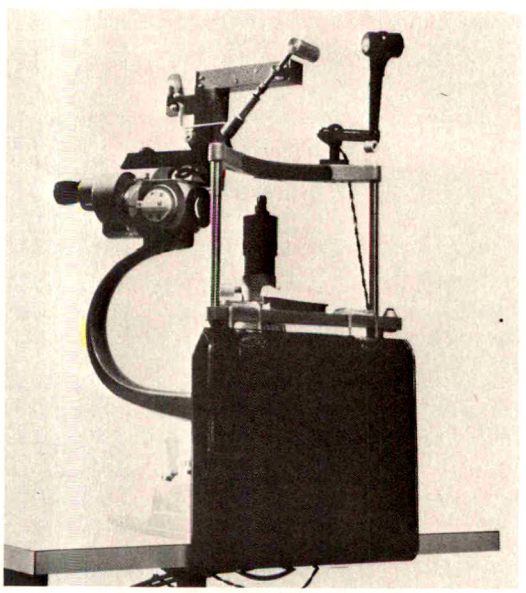


Figure (Wing and Weiter). Plexiglass chest shield (25 × 25 × 4 cm) hooked onto the slit lamp's chin bar.

Large-Print Reading for the Waiting Room

Editor:

Last week I was in my ophthalmologist's office. On his table full of magazines in the waiting room, there was only one magazine in large print. It was a publication consisting of about ten articles from the Reader's Digest.

Realizing that patients in a doctor's office are often fearful or apprehensive, I began thinking: Why didn't he have a bunch of different items of reading material in large print, for patients who cannot read a regular magazine?

The following suggestions should be of interest to ophthalmologists who read your publication, since they have crowded waiting rooms, and their patients include many people who cannot read the fine print in regular magazines and books: (1) It is possible to purchase from a Christian book store a paperback copy of the Bible in large print, for about $16. Reading the Bible, while in a doctor's waiting room, could help many apprehensive patients. (2) One of Norman Vincent Peale's publications, "Creative Help for Daily Living," is now available in large type. A subscription to this magazine does not cost any definite amount, but a contribution of $5.00 to the Foundation is considered adequate. This magazine, like the large-print Bible, could be interesting and reassuring to readers in an ophthalmologist's waiting room. (3) When business cards are printed, the name of the company or the name of the person is often in large print, whereas the office address and the telephone number are in fine print. Out of courtesy to their patients, I believe that ophthalmologists, optometrists, and opticians should order business cards that can be read easily by a person with a visual defect.

UEL P. MCCULLOUGH
Goodyear, Arizona

Determining Mean Intraocular Pressure

Editor:

In the article, "Prognostic significance of optic disk cupping in ocular hypertensive patients," by M. E. Yablonski, T. J. Zimmerman, M. A. Kass, and B. Becker (Am. J. Ophthalmol. 89:585, 1980), the republication of data for a group of individuals with suspected glaucoma "using mainly the same subjects" as the study by Hart and associates,[1] may be misinterpreted. Both studies conclude that the larger the cup/disk ratio and the higher the mean intraocular pressure (IOP) over a five-year follow-up period, the more likely the eye is to develop visual field loss. Hart and associates found that the initial IOP at the first visit was not a good predictor of future progressive damage. The difference between the predictive value of the initial IOP compared to the mean IOP over time should be self-evident. The glaucomatous eye has an IOP not only higher but also more unstable than normal. One measurement (that is, the initial IOP) is not likely to show the increase in IOP to which the optic nerve head is exposed over time. Yablonski and associates found that 100% of eyes with a cup/disk ratio greater than 0.6 and a mean IOP (over five years) of 28 mm Hg or more lost visual field without therapy. This reemphasizes that the higher the prevailing IOP, the more likely that progressive damage will occur. However, the reader should not take the data to mean that a patient with a cup/disk ratio greater than 0.6 and an IOP of more than 28 mm Hg on the initial visit has a 100% risk of visual field loss in five years. The data of Yablonski and associates would be useful in those patients for whom IOP measured at several visits is already available, or for whom, after a suitable period of observation without treatment, the mean IOP level can be estimated. In this regard, it would be clinically useful

to know after how many visits the mean IOP becomes an apparently strong predictor. Could a good estimate of mean IOP be made after the first year, or after the second year? Would several measurements closely spaced near the initial visit provide as good an estimate of prevailing IOP? It would be helpful to know how many IOP measurements per patient went into the mean IOP figure in either paper. With this information, the clinician could use more accurately the disk and IOP status in deciding which patients to treat and when to treat them.

HARRY A. QUIGLEY, M.D.
Baltimore, Maryland

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Reply

Editor:

Dr. Quigley's letter raises important issues. We agree entirely that a mean intraocular pressure is more representative of a patient's ocular condition than an initial intraocular pressure. This conclusion has been reached by other investigators as well,[1,2] and we used mean intraocular pressures in our study for this reason. The number of intraocular pressure readings per patient recorded during the five-year follow-up period of the study ranged from 15 to 82.

We further agree with Dr. Quigley that an intraocular pressure of more than 28 mm Hg and a cup/disk ratio greater than 0.6 do not imply impending glaucomatous visual field loss. Neither an intraocular pressure of 28 mm Hg nor a cup/disk ratio of 0.6 is a "magic number" separating subjects considered at high risk of developing glaucoma. These numbers are arbitrary separations based upon a retrospective review of a small number of patients and are not presented as guidelines for therapy. Many other factors (for example, age and family history of glaucoma) influence the prognosis for subjects with ocular hypertension.[3,4]

Dr. Quigley asks how many intraocular pressure readings were required until a reasonable approximation of the mean five-year intraocular pressure was obtained. Because this was a retrospective study, the data were not collected in a systematic fashion to allow us to answer this question. The problem is complicated further by known diurnal, seasonal, and hormonal influences on intraocular pressure. Furthermore, intraocular pressure may follow upward or downward trends or cyclical patterns over time,[5] so that it may be impossible to determine a representative intraocular pressure level in some patients. Prospective studies are required to answer these questions.

MICHAEL E. YABLONSKI, M.D.
THOM J. ZIMMERMAN, M.D.
MICHAEL A. KASS, M.D.
BERNARD BECKER, M.D.
St. Louis, Missouri

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Corneal Indentation To Relieve Glaucoma

Editor:

In his article "Corneal indentation to relieve acute angle-closure glaucoma" (Am. J. Ophthalmol. 88:1091, 1979), D. R. Anderson recommends the method

he describes as being safer than hyperosmotic agents.

With respect to systemic side effects I agree, but ocular side effects could be greater. There may be damage to the endothelium by stretching the inner corneal layer when the usual anterior-convex shape of the cornea is changed to a plane shape or even to a posterior-convex shape.

Since several cycles of indentation seem to be necessary to achieve the desired effect and since the endothelium is already compromised by high intraocular pressure levels, I suggest that endothelial cell counts should be done before this method is generally recommended.

A soft contact lens might also be placed over the cornea before the indentation in order to minimize or prevent epithelial abrasions.

KLAUS D. TEICHMANN, F.R.C.S.(C.)
Paddington, Australia

Reply

Editor:

The surface area of a flattened cornea is less than that of the naturally curved cornea (so that the endothelium is compressed a bit rather than stretched), and even the indented cornea does not necessarily have a stretched internal surface area. Nonetheless, I suppose it is theoretically possible that distortion of the cornea can injure the endothelium. My judgment is that such injury is unlikely, so I do not share Dr. Teichmann's concern. In any event, we have not recognized any instances of persistent corneal edema after corneal indentation. In order to study subclinical injury by endothelial cell counts or measurements of corneal thickness, one would need control subjects who had their angle-closure glaucoma controlled by other means. Otherwise, one could not distinguish between injury from the corneal indentation and injury from having undergone an acute attack. If such injury to the endothelium were discovered, one would have to ask whether similar injury occurs during Schiøtz tonometry, indentation gonioscopy, or massage of the globe before cataract extraction.

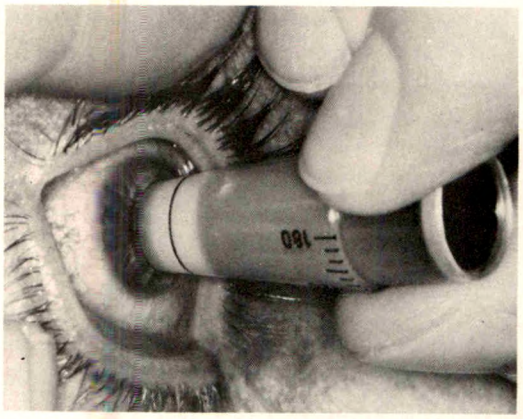


Figure (Anderson). Applanation prism held to the cornea.

Injury to the epithelium is a more serious problem with corneal indentation, especially when a cotton-tipped applicator is used. For this reason I usually use the Goldmann applanation prism to achieve the corneal indentation. To do this, the applanation prism is removed from the slit-lamp and held with the fingers to the cornea of the patient who is seated or in a reclining position (Figure). Of course, any other smooth instrument can be used. Dr. Teichmann's suggestion of using a soft contact lens may also cause epithelial abrasions, even if a cotton-tipped applicator were used (although I have not tried his method).

DOUGLAS R. ANDERSON, M.D.
Miami, Florida

Retrolental Fibroplasia and the Retinal Vasculature

Editor:

I would like to comment on the article, "Angioma-like mass in a patient with retrolental fibroplasia" (Am. J. Ophthalmol. 89:647, 1980) by C. C. Barr, T. A. Rice, and R. G. Michels. Flynn and asso-

ciates[1] found that focal wrinkling and whitening of the inner limiting membrane follow regression of the mesenchymal shunt in retrolental fibroplasia. In this case the angle made by the inferotemporal preretinal membrane and the horizontal raphe is consistent with this epiretinal membrane being a remnant of the inferotemporal advancing retinal vascular (mesenchymal) line, as reported by Foos and Kopelow.[2] Epipapillary membranes have been described as common in retrolental fibroplasia.[3]

Although it is accepted that the retinal vasculature is mature at birth, this process may be incomplete in the postoral retina[2]; this is one explanation of retrolental fibroplasia reported in full-term infants.

The retina becomes vascularized in the second trimester,[4] not in the third as stated by the authors. An angioma-like mass in retrolental fibroplasia has been reported previously.[5]

GLEN COLE, M.B.
Bedford Park, Australia

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Implant Follow-Up

Editor:

In recognition of the mobility of our society, I would like to add to the recent letter by Kenneth Hoffer (Am. J. Ophthalmol. 89:877, 1980) concerning implant follow-up. Follow-up is important not only to maintain the necessary records, but to patient care.

As ophthalmologists, we have an obligation to provide excellent medical and surgical care for the immediate condition and to make it available in the future.

Surgical care and fitting of contact lenses have always included adequate follow-up. Before performing surgery or fitting contact lenses, the ophthalmologist should discuss the required follow-up procedures with the patient and family.

The out-of-town patient should be followed up long enough to ensure the absence of immediate postoperative or fitting problems.

When discharged from care, the patient should be given a letter or form with details of the date of surgery, type of surgery, type of intraocular implant or contact lenses specifications, and current medications. The form should be given to the patient in case the patient has problems elsewhere or does not go to the referred ophthalmologist. If an individual ophthalmologist is not specified (the best procedure), the patient should be given the names of several ophthalmologists, preferably ones familiar with the procedure that has been carried out. I have seen patients told to see their local "eye doctor" for follow-up surgical care who have gone to optometrists for this medical care.

It is unfair to the patient and to the ophthalmologist when the patient, with or without a problem, comes to an ophthalmologist's office without information that might be essential to that patient's care. Simple communication can improve rapport and care and minimize problems if complications should occur.

FRANK J. WEINSTOCK, M.D.
Canton, Ohio

V-Z vs Z-Plasty

Editor:

In the article, "Combined Z-plasty and horizontal shortening procedure for

ectropion" (Am. J. Ophthalmol. 89:525, 1980), by A. M. Putterman, the author stated that, to the best of his knowledge, his was the first report of this technique. I regret to inform him that I described the operation in question in 1959.[1] A comparison of Figures 2 to 4 from my article with Figure 1 from that of Putterman will bear out this assertion.

I specifically noted that the same technique could be used with good results in other palpebral disorders, such as recent lacerations involving the margin, traumatic and congenital colobomas, small tumors needing removal, and senile-spastic entropion whenever the eyelid is distended. I also pointed out that the contour of the excised part of the eyelid may vary from case to case and is only of secondary importance. I had even thought of christening the procedure a U-Z, but this would have smacked too much of a monogram.

ALBERTO URRETS-ZAVALIA, M.D.
Cordoba, Argentina

REFERENCE

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Reply

Editor:

It has been my experience that if one searches far back enough in published reports, one almost invariably finds an operative technique that has some similarity to a new procedure. However, the new procedure usually has distinct differences from the older one. I believe this is true in Dr. Urrets-Zavalia's comparison of his 1959 V-Z procedure with my combined Z-plasty and horizontal shortening operation.

The V-Z operation removes a full-thickness triangle of eyelid; the combined Z-plasty and horizontal shortening procedure differs by removing a full-thickness pentagon of eyelid. The segment of eyelid removed in the V-Z procedure is a standard 7 to 12 mm, whereas the amount of eyelid resected in the combined procedure is more variable, depending on the amount needed to produce a tight eyelid. The V-Z technique uses a "tongue and groove marginal union"; the combined procedure connects a straight marginal incision. The V-Z plasty resects normal eyelid; the combined Z-plasty and horizontal shortening technique removes a scarred segment of eyelid. The V-Z operation is performed without a suture tarsorrhaphy; the combined procedure employs one. The V-Z procedure is performed on the lower eyelid; the combined procedure is done on lower or upper eyelid. Most importantly, the V-Z procedure is an operation advocated for acquired senile ectropion; the combined Z-plasty and horizontal shortening is an operation specifically designed for cicatricial ectropion.

I offer my apologies to Dr. Urrets-Zavalia for not including his article among the references in my article.

ALLEN M. PUTTERMAN, M.D.
Chicago, Illinois

BOOK REVIEWS

World Blindness and its Prevention. By Sir John Wilson. New York, University Press, 1980. Hardcover and paperbound, 93 pages, preface, index. $17.95.

"Prevention of blindness" has been an abstract goal, one with which everyone could agree but which few could define specifically. The situation is changing, however. Directions are being formally espoused, priorities debated, and programs formulated, with the International Agency for the Prevention of Blindness serving a central role as stimulus and

coordinator of and forum for these activities. Originally conceived as a formal report of the Agency's first General Assembly, held in Oxford in the summer of 1978, this slim volume is considerably more than that: it is a historical record of the foundation of both the Agency and the World Health Organization's program for the prevention of blindness; a concise and graphic description of the problem; and a statement of the principles that will guide future developments.

In an eloquent preface, Sir John Wilson says that blindness can be dramatically reduced with existing technology. All that is needed is political will, presumably to be supplied by the scores of national committees associated with the Agency, and professional leadership capable of adapting and applying existing technology to the problems of disadvantaged communities in the developing world, where much of the blindness is concentrated.

By way of orientation, the major causes of "avoidable" blindness are then described. Of the four given highest priority, three (trachoma, onchocerciasis, and xerophthalmia) are preventable, and one (cataract) curable. More lengthy discussions are rightfully left to relevant WHO publications.

In the section that follows, Professors A. E. Maumenee and Barry Jones discuss the "new breed" of ophthalmologists needed to provide leadership in this area. Trained in epidemiology, nutrition, and related public health fields, such individuals would be capable of designing and evaluating innovative, multidisciplinary programs addressing these problems. The suggestion that specialized "Collaborating Centers" be established to provide training and consultation and to carry on research in this area has already borne fruit. Dr. Carl Kupfer presents distressing demographic projections suggesting rapid growth in the number of blind individuals in the years to come, with age-related degenerative diseases accounting for an increasing share of the problem. Brief descriptions of ongoing blindness prevention activities serve to remind us of how much is yet to be done.

The supplement contains several useful documents: the first two reports of WHO's Program Advisory Committee and a summary of available blindness data from around the world.

Even if the reader disagrees with some, or all, of the suggestions and conclusions contained in this book, they provide a frame work for constructive discussion and perhaps the interest, involvement, and commitment so desperately needed.

ALFRED SOMMER

Queratomileusis y Queratofaquia. By Jose I. Barraquer. Bogata, Colombia, Barraquer America, 1980. Hardcover, 517 pages, 426 black and white figures, 133 color figures. $50

Dr. Barraquer's book comes at a time when the interest in refractive surgery is at its peak. Even though he has been working on this subject for over 30 years and his techniques have been known and practiced by some American eye surgeons for over 15 years, these have aroused widespread interest only in the past few years.

Dr. Barraquer noticed in 1948 that lamellar keratoplasty modified the shape of the cornea with keratoconus and improved the refraction of the eye. He considered this the beginning of refractive keratoplasty. In 15 chapters, he condenses many years of laboratory and clinical research in this special area of corneal surgery. The term keratomileusis means corneal sculpture or modification of its form or structure. Keratophakia means the introduction of a lens into the

cornea. This book, therefore, deals with these two techniques of refractive surgery, but it also provides interesting background on other types of refractive surgery.

The first chapter describes experiments to cryopreserve tissue, early instrumentation designs, and surgical techniques. Dr. Barraquer believed adequate cryopreservation of the corneal tissue used in keratophakia was important to preserve the transparency of this tissue. He devised a technique to protect the corneal disk from severe freezing damage, and he believes that with his technique there is an adequate survival of corneal cells.

Dr. Barraquer points out that this was the first time mathematics and electronic calculations were applied directly to a surgical technique to modify the visual function of the eye. Those who tried his techniques before and after the development of computers know that surgical techniques have been greatly improved by the use of electronic calculators.

In later chapters, Dr. Barraquer describes in detail the components of every instrument used for refractive surgery and for anterior segment surgery in general. The surgical techniques are described and the normal postoperative course are described step by step.

A most interesting chapter is that on preventing and treating operative complications. These range from complications caused by the surgeon's mistakes to complications caused by a malfunctioning cryolathe. The description of postoperative complications indicates that this is not a simple surgical technique and that the surgeon must be aware of a variety of complications that may require reoperation. This chapter is well illustrated and adds credibility to Dr. Barraquer's techniques, because there is no surgical procedure without potential complications.

The last chapter includes the necessary mathematical information for these surgical techniques including basic formulas for keratophakia and keratomileusis.

The question of survival of corneal stromal cells after freezing has not been answered in this book. Studies with isotopes (^{35}S) have shown that stromal cells can survive freezing and be metabolically active only if frozen and thawed at specific freezing rates after treatment with a cryophylactic agent.

The book is profusely illustrated with drawings and black and white and color photographs. It will be most useful to corneal surgeons interested in refractive keratoplasty. We hope it will be translated into English.

FRANK M. POLACK

Retinal Detachment and Diagnosis and Treatment. By William E. Benson. New York, Harper and Row, Inc., 1980. Hardcover, 208 pages, 191 black and white figures, 10 color figures. $27.50

This short primer on retinal detachment contains 12 chapters. The first describes symptoms and signs and defines the special terminology of retinal detachment. For example, a break is caused by vitreous traction and a hole by retinal atrophy. The second chapter is a well-illustrated presentation of the pathophysiology of detachment. The third chapter about predisposing conditions is particularly effective. It is replete with percentages from many sources. The paragraph on lattice degeneration is typical: "Lattice is the direct cause of 21 per cent of all retinal detachments[8] and is present in 41 per cent.[3] In 30 to 45 per cent of cases the detachment is caused by atrophic holes in lattice.[8,13]" Chapter 4 is a historical survey of the conceptions that led to an understanding of the disorder and the methods of repair that followed.

Its five pages of text are a model of concise writing. Chapters 5 and 6 cover differential diagnosis and methods of examination.

The remainder of the book is about surgical management. Here Benson's short form is inadequate. The beginner will be perplexed by statements such as "drainage should be avoided unless necessary for surgical success." Nevertheless, Dr. Benson covers the subject and there is an adequate bibliography.

The text reads easily. The story of retinal detachment unfolds in an interesting and logical way. Dr. Benson anticipates readers' questions and provides answers that are reasonable although occasionally open to doubt. The major fault with this book is its brevity. Subsequent editions, which are sure to follow, will no doubt remedy this.

HARVEY LINCOFF

SYMPOSIA

Intraocular Lens Implants in Children. Edited by David A. Hiles. New York, Grune & Stratton, 1980. Hardcover, 276 pages, bibliography, index, more than 100 black and white figures. $23.50

BYRON, H. M.: **Medico-legal considerations of implant surgery in minors**

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Proceedings of the Paul Cibus Club 1979. Edited by Edward Okun and Glen Paul Johnston. St. Louis, Paul Cibis Club, 1979. Paperbound, 216 pages, 164 black and white figures. $20

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ARRIBAS, N. P.: **Macular holes with retinal detachment**

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VAISER, A.: **Applications of a new 3 × 5 mm oval sponge exoplant**

Ageing of the Lens. Edited by François Regnault, Otto Hockwin, and Yves Courtois. Amsterdam, Elsevier, 1980. Hardcover, 316 pages, 49 tables, 15

color figures, 175 black and white figures. $56

PIATIGORSKY, J., BHAT, S. P., JONES, R. E., AND SHINOHARA, T.: **d-Crystallin synthesis in the embryonic chick lens. First look at d-crystallin gene sequences**

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REGNAULT, F.: **Report on the meeting of the Cataract Collaborative Research Group**

Current Topics in Eye Research. Volume 2. Edited by Jose A. Zadunaisky and Hugh Davson. New York, Academic Press, Inc., 1980. Hardcover, 308 pages, author index, subject index, 36 black and white figures. $35

WITKOVSKY, P.: **Excitation and adaptation in the vertebrate retina**

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FAURE, J. P.: **Autoimmunity and the retina**

Ophthalmology Update. Edited by K. Mizuno and Y. Mitsui. Amsterdam, Excerpta Medica, 1980. Hardcover,

205 pages, subject index, 152 black and white figures, 30 color figures. $41.50

HAMASAKI, D. I.: **Review of experiments dealing with the development of the visual system**

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LITTLE, H. L.: **Practice of laser photocoagulation**

Recent Advances in Ultrasound Diagnosis. Volume 2. Edited by Asim Kurjak. Amsterdam, Excerpta Medica, 1980. Hardcover, 560 pages, index, 510 black and white figures, 60 tables. $97.50

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Biological action of ultrasound in relation to the radiation safety question
The value of ultrasound in perinatal medicine from the clinician's point of view
The influence of diagnostic ultrasound in perinatal medicine
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Ultrasonic assessment of fetal dynamic behavior. Physiology and clinic
Recent advances in echocardiography
New clinical applications
Ultrasound monitoring of ovarian stimulation
Ultrasound and the radiologist
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Free communications

ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

Acta Ophthalmologica

THE VARIABLE ANGLE-MIRROR, A NEW TOOL FOR THE STUDY OF OCULAR DOMINANCE AND EYE FIXATION. Björk, A. (Dept. Ophthalmol., and Med. Exam. Ctr. of Swedish Air Force, Stockholm, Sweden). Acta Ophthalmol. 58:202, 1980.

A simple tool for studying optical dominance consisting of two plane mirrors moveably fixed to one another was developed. The patient looks straight ahead at the reflection of his own face and observes which eye is covered by the joint between the two mirror halves. By changing the angle a further check-up on eye dominance can be obtained. The method is simple and gives a quick idea of the eye dominance. (4 figures, 1 table, 7 references)—Author's abstract

ANGIOTENSIN-CONVERTING ENZYME IN UVEITIS AND SARCOIDOSIS. Rømer, F. K., Schmidt, P., and Geday, H. (Depts. Med., and Ophthalmol., Univ. Aarhus, Denmark). Acta Ophthalmol. 58:243, 1980.

Serum angiotensin-coverting enzyme was studied in a group of 29 sarcoidosis patients (among whom five had uveitis) and in 22 patients with non-sarcoid uveitis. Angiotensin-converting enzyme was normal in all non-sarcoid patients and elevated in 62% of the patients with sarcoidosis. Thus an elevated serum angiotensin-converting enzyme is closely linked to sarcoidosis and may be a valuable tool in evaluating patients with uveitis in search of a sarcoid origin. (3 tables, 11 references)—Authors' abstract

American Journal of Human Genetics

POPULATION GENETIC STUDIES OF RETINITIS PIGMENTOSA. Boughman, J. A., Conneally, P. M., and Nance, W. E. (Dept. Hum. Genet., Medical College of Virginia, Richmond, Va.). Am. J. Hum. Genet. 32:223, 1980.

A questionnaire survey characterized a sample of 670 probands with retinitis pigmentosa and allied disorders. Segregation analysis provided some evidence for a small proportion of sporadic cases and for decreased segregation ratios of the dominant and recessive genotypes, which could be attributed to delayed age of onset in some cases. The overall incidence of retinitis pigmentosa was indirectly calculated to be approximately 1 in 3,700 while the incidence of autosomal recessive retinitis pigmentosa, including at least two genocopies, was estimated to be about 1 in 4,450. Family data analysis included the calculation of the likelihood that each family represented autosomal recessive, autosomal dominant, and X-linked inheritance patterns. These likelihoods were then converted to relative probabilities and summed over the sample population to yield estimates of the proportions of the three Mendelian types. This large, heterogeneous sample indicated that approximately 84% of the cases in the United States may be autosomal recessive, while about 10% are dominant and 6% X-linked recessive. (9 tables, 27 references)—Authors' abstract

Anesthesia and Analgesia

MYOCARDIAL REINFARCTION FOLLOWING LOCAL ANESTHESIA FOR OPHTHALMIC SURGERY. Backer, C. L., Tinker, J. H., Robertson, D. M., and Vlietstra, R. E. (Dept. Anesth., Ophthalmol., and Int. Med., Mayo Clinic, Rochester, Minn.). Anesth. Analg. 59:257, 1980.

Ten thousand ophthalmic operations managed with local anesthesia and/or retrobulbar block were performed over a 15 year period. Of these 195 patients who underwent 288 separate operations had previously suffered a documented myocardial infarction. Not a single postoperative reinfarction or death occurred in this group. During the same period, only 26 such operations were per-

formed on 21 patients with prior myocardial infarction under general anesthesia. This zero percent reinfarction rate is significantly less than the 6.1% reinfarction rate previously reported from this institution for 587 operations on patients with documented preoperative myocardial infarction who had general or major regional anesthesia for nonophthalmic surgery. In the prior study, intrathoracic, upper abdominal, and great vessel operations, lasting three hours or more, were associated with significantly greater reinfarction rates than the total population. Eliminating these from the prior series yields a 3.6% reinfarction rate for all other operations, which is still significantly greater than the zero reinfarction in the present series of ophthalmic procedures. The group that had general anesthesia for ophthalmic surgery is too small for meaningful comparisons, and one cannot conclude that local is safer than general anesthesia. However, one can conclude that local anesthesia and/or retrobulbar block for ophthalmic surgery does not pose special risks for reinfarction in the patient with a preoperative myocardial infarction. (6 tables, 17 references)—Authors' abstract

Annals of Internal Medicine

MITRAL VALVE PROLAPSE AND OPHTHALMOPLEGIA. A PROGRESSIVE, CARDIONEUROLOGIC SYNDROME. Darsee, J. R., Miklozek, C. L., Heymsfield, S. B., Hopkins, L. C., and Wenger, N. K. (Dept. Med. and Neurol., Emory University, Atlanta, Ga.). Ann. Intern. Med. 92:735, 1980.

Disorders characterized by both neurologic (ataxia, ophthalmoplegia, ptosis, neuromyopathy) and cardiologic (heart block, cardiomyopathy) abnormalities have been previously called the "ophthalmoplegia plus" syndromes. Most are not due to a specific enzyme defect or metabolic abnormality and thus may be similar phenotypic expressions of diverse causes. The authors studied seven patients with progressive external ophthalmoplegia and variable ataxia, with mitral valve prolapse and mitral regurgitation that progressed in severity as did the neuromuscular manifestations. Abnormal skeletal muscle biopsies showed "ragged-red" fibers or congenital fiber type disproportion; serum alanine levels were elevated; in-vivo and in-vitro tests of pyruvate metabolism gave abnormal results; C4 complement was decreased; and the patients' fibroblasts bound immunoglobulin when incubated with autologous serum. These data suggest a distinct neuromuscular disorder with metabolic and immunologic features associated with mitral value prolapse and progressive mitral regurgitation. (5 figures, 2 tables, 28 references)—Authors' abstract

Annals of Neurology

OPTOKINETIC NYSTAGMUS AND PARIETAL LOBE LESIONS. Baloh, R. W., Yee, R. D., and Honrubia, V. (Dept. Neurol. Reed Neurol. Res. Ctr., and Dept. Ophthalmol., Jules Stein Eye Inst., and Dept. Surg., Univ. California, Los Angeles, Calif.). Ann. Neurol. 7:269, 1980.

In an attempt to define better the mechanism of impaired optokinetic nystagmus caused by parietal lobe lesions, the authors recorded the eye movements of two patients. One had a slowly enlarging parietal glioma, and the other, an infarction involving the parietal and occipital lobes. In both patients, ipsilateral foveal pursuit and full-field pursuit of a surrounding optokinetic drum were impaired while voluntary and involuntary saccades (fast components) were normal. Foveal pursuit was impaired more than full-field pursuit, suggesting the existence of two separate pathways by which visual signals for optokinetic nystagmus slow phases reach the oculomotor centers in the brainstem. The authors conclude that the ipsilateral slow phase optokinetic nystagmus deficit seen in their patients resulted from damage to the foveal pursuit pathway that runs from the occipitoparietal association area to the ipsilateral brainstem horizontal gaze center. (3 figures, 2 tables, 39 references)—Authors' abstract

Brain

VISION IN THE TEMPORAL CRESCENT IN OCCIPITAL INFARCTION. Benton, S., Levy, I., and Swash, M. (Sec. Neurol. Sci., London Hospital, London). Brain 103:83, 1980.

Four patients with hemianopias caused by

occipital infarction are described. In each of these patients there was useful residual vision in the unpaired temporal crescent. The temporal crescent is dependent on nasal retinal cells and is important for visual-orientating responses and aids in initiating rapid body or eye movements in response to sudden movements in the periphery. It can thus be regarded as having a particular role in ambient vision. The monocular peripheral field is used to orient the eyes and limbs so as to bring the object of regard within the view of the central and foveal fields for a closer visual examination and then for manipulation by the hands and mouth. (7 figures, 39 references)—David Shoch

WORD-FORM DYSLEXIA. Warrington, E. K., and Shallice, T. (National Hospital, Queen Square, London). Brain 103:99, 1980.

In this study the authors report their investigations of two patients with an acquired dyslexia characterized by letter-by-letter reading, whole word reading being apparently impossible. It has been shown that this phenomenon of letter-by-letter reading cannot be accounted for by visual or perceptual factors nor by impairment of visual span of apprehension. The exceptionally slow speed of reading was documented and a clear relationship between word length and reading speed established. Performance on tasks considered to maximize whole word reading which at the same time prevent the possibility of letter-by-letter reading, namely, reading script and reading with tachistoscopic presentation, was impaired. The satisfactory performance of these two patients on tasks of picture interpretation suggests that the two components of the syndrome simultanagnosia, letter-by-letter reading and piecemeal perception of complex scenes, are dissociable. The authors conclude that in this type of acquired dyslexia there is damage to the system through which a visual word-form is attained. (3 figures, 5 tables, 29 references)—Authors' abstact

Clinical Genetics

GENETIC HETEROGENEITY OF CONGENITAL GLAUCOMA. Genčík, A., Genčíková, A., and Gerinec, A., (Dept. Clin. Genet., and Ophthalmol. Clin., University Hospital, Bratislava, Czech.). Clin. Genet. 17:241, 1980.

Analysis of 126 families comprising 205 patients with congenital glaucoma demonstrates that in Gypsies this disease follows the pattern of autosomal recessive inheritance with complete penetrance, while in the non-Gypsy population, its mode of inheritance is most probably multifactorial. In Gypsy patients with congenital glaucoma, the eyes are always bilaterally affected, the onset of the disease is in the prenatal period, and its course is rather severe. The population frequency of the disease is extremely high (among Gypsies), and the consanguinity rate among parents is as high as 41%. In non-Gypsy patients, 26.6% of all cases are only unilaterally affected, and the course of the disease is generally milder with a later onset. The population frequency in a non-Gypsy population is much lower, the consanguinity rate is not increased, and an excess of males (1.55:1) is significant. (2 figures, 2 tables, 22 references)—Authors' abstract

European Neurology

OPTIC NEURITIS AND MULTIPLE SCLEROSIS. AN IMMUNOLOGICAL STUDY. Frick, E., and Stickl, H. (Neurol. Clin., Munich University Hosp., Grosshadern and Bavarian Vaccination Inst., Munich, Germany). Eur. Neurol. 19:185, 1980.

A large group of patients were examined for the presence of antibody-dependent lymphocyte cytotoxicity against myelin basic protein. Ninety-four percent of the patients with active multiple sclerosis had positive tests. On the other hand, only 5% of 200 patients with other neurological complaints had positive findings. The authors believe that a positive antibody-dependent lymphocyte cytotoxicity against myelin basic protein in patients with optic neuritis is in all probability the first sign of multiple sclerosis and almost pathognomonic for it. (1 figure, 33 references)—David Shoch

Journal of the American Medical Association

A NEW FORM OF EHLERS-DANLOS SYNDROME. FIBRONECTIN CORRECTS

DEFECTIVE PLATELET FUNCTION. Arneson, M. A., Hammerschmidt, D. E., Furcht, L. T., and King, R. A. (Sec. Occupational Med., Hematol., and Hum. Genet., Dept. Med., Univ. Minnesota, Minneapolis, Minn.). J.A.M.A. 244:144, 1980.

In a kindred with a mild, recessively inherited variant of the Ehlers-Danlos syndrome, a platelet aggregation defect segregated concordantly with skin and joint abnormalities. This defect was partially corrected in vitro by addition of normal plasma or cryoprecipitate. The plasma of the patients with Ehlers-Danlos syndrome failed to support the aggregation of normal gel-filtered platelets in response to collagen; this defect was completely corrected by the addition of normal human fibronectin. Since fibronectin is an important adhesive glycoprotein in connective tissue and is required for normal platelet interactions with collagen, the authors propose that both platelet malfunction and joint hypermobility in this kindred are likely explained by a defective fibronectin. (6 figures, 14 references)—Authors' abstract

Journal of Investigative Dermatology

POTENTIAL OCULAR COMPLICATIONS FROM PUVA THERAPY AND THEIR PREVENTION. Lerman, S., Megaw, J., and Willis, I. (Lab. for Ophthalmic Res., Emory Univ., Atlanta, Ga. and Veterans Admin. Hosp., Decatur, Ga.). J. Invest. Dermatol. 74:197, 1980.

During the last decade psoralens have become increasingly popular in treating psoriasis. The well-known photosensitizing action of these drugs has led to increasing concern regarding potential ocular complications, particularly in patients receiving prolonged psoralen therapy. The authors have demonstrated that this drug can be found in lenses of rats injected (intraperitoneally) with 4-8 mg/kg of 8-methoxypsoralen and that its presence can lead to a photosensitized enhancement of lenticular fluorescence. The authors have recently shown that free 8-methoxypsoralen can also be detected in human lenses for at least 12 hours following oral ingestion. Since the free drug tends to diffuse out of the lens during this period of time (as long as photic stimulation is prevented) it may be possible to prevent these photochemical reactions if the patient avoids exposure to ambient light for 12-24 hours immediately following ingestion of the drug. It may also be possible to protect these patients with special glasses which are capable of reflecting all ultraviolet radiation (up to 400 nm) while completely transmitting the visible radiation (400-750 nm). (4 figures, 40 references)—Authors' abstract

Journal of Medical Genetics

EXCESS OF CANCER DEATHS IN GRANDPARENTS OF PATIENTS WITH RETINOBLASTOMA. Bonaïti-Pellié, C., and Briard-Guillemot, M. L. (Groupe de Recherches de Genetique Epidemiologique, Chateau de Longchamp, and Unité de Recherches de Génétiqute Medicale, Hospital des Enfants-Malades, Paris, France). J. Med. Genet. 17:95, 1980.

An excess of cancer deaths was found in grandparents of 308 children with retinoblastoma. This excess was found in all types of retinoblastoma, unilateral and bilateral, sporadic and familial. The authors postulated that the excess could be the result of a factor of susceptibility to cancer, different from the retinoblastoma gene, which would increase the mutation rate in retinal and germ cells as well as in other tissues. (11 tables, 21 references)—Authors' abstract

Journal of Neurology, Neurosurgery and Psychiatry

VISUAL FIELD ABNORMALITIES IN MULTIPLE SCLEROSIS. Patterson, V. H., and Heron, J. R. (Dept. Neurol., North Staffordshire Hosp. Ctr., Stroke-on-Trent, Staffs). J. Neurol., Neurosurg., Psychiatry 43:205, 1980.

Multiple sclerosis generally has a predilection for the anterior visual pathways. Indeed, some studies have shown demyelination of the optic nerves in almost all cases of multiple

sclerosis, whether or not the eyes were involved. Because of this, rather sophisticated methods have been developed for detecting visual difficulties in patients with suspected multiple sclerosis. The authors believe that these sophisticated tests are unnecessary and, in order to prove this point, examined visual fields with a tangent screen in 54 patients with multiple sclerosis or optic neuritis. Visual fields were abnormal in all patients with definite multiple sclerosis, 94% with probable multiple sclerosis, and 81% with possible multiple sclerosis. Three quarters of the patients with multiple sclerosis and no history of visual symptoms had abnormal fields. The commonest defect found was an arcuate scotoma. As a diagnostic test of visual pathway involvement in multiple sclerosis, tangent screen examination compares favorable with more sophisticated methods. (2 figures, 4 tables, 20 references)—David Shoch

Journal of Neurosurgery

BILATERAL CORTICAL BLINDNESS ASSOCIATED WITH CAROTID STENOSIS IN A PATIENT WITH A PERSISTENT TRIGEMINAL ARTERY. Heeney, D. J., and Koo, A. H. (Dept. Radiol., Santa Clara Valley Med. Ctr., San Jose, Calif.). J. Neurosurg. 52:709, 1980.

Unilateral cortical blindness is usually the result of ischemia in the carotid circulation while bilateral involvement is typically attributed to the vertebrobasilar system. The patient described here is the exception to this rule, since he had bilateral cortical blindness as a result of an ulcerated plaque in the left internal carotid artery. This was possible only because there existed a caroticobasilar anastomosis in the form of a persistent trigeminal artery. The patient gave a history of three episodes of complete blindness, one episode lasting for an hour, one for five minutes, and one for ten minutes. A left carotid endarterectomy was performed and the patient has had no further episodes of blindness since that time. The trigeminal artery is usually seen only in the very small embryo and is not normally patent after the 14-mm stage. Occasionally it remains as an anomaly and, more rarely still, leads to pathologic findings as in the case reported here. (2 figures, 11 references)—David Shoch

Journal of the Royal Society of Medicine

INDICATIONS FOR FLUORESCEIN ANGIOGRAPHY IN DISEASE OF THE OCULAR FUNDUS. A REVIEW. ffytche, T. J., Shilling, J. S., Chisholm, I. H., and Federman, J. L. (St. Thomas' Hosp., London). J. Royal Soc. Med. 73:362, 1980.

The indications for fluorescein angiography are briefly reviewed but the most important part of the article is a paragraph on the dangers of angiography. There apparently have been 20 reports of fatalities directly caused by the injection of fluorescein. These deaths have been related to underlying cardiovascular or cerebrovascular disturbances. However, there is a 0.4% incidence of severe complications after injection, including vasovagal attacks, bronchospasm, respiratory arrest, circulatory collapse, cardiac infarction, stroke, and cardiac arrest. Minor complications and side effects are far more common. Nausea occurs in about 10% of patients and vomiting in 1% to 7%. Other minor disturbances include itching, rashes, sneezing, and phlebitis at the site of injection. (12 references)—David Shoch

Metabolic and Pediatric Ophthalmology

COMBINED DEFICIENCY OF XANTHINE OXIDASE AND SULFITE OXIDASE. OPHTHALMOLOGICAL FINDINGS IN A 3-WEEK-OLD GIRL. Beemer, F. A., and Deeleman, J. W. (Univ. Children's Hosp., Het Wilhelmina Kinderziekenhuis, Utrecht, The Netherlands). Metabol. Pediatr. Ophthalmol. 4:49, 1980.

Sulfite oxidase deficiency has been discussed frequently in published reports but the patient described here is only the fourth to be reported. In addition to sulfite oxidase deficiency, the child also had xanthine oxidase deficiency. The latter causes the excretion of excess quantities of xanthine in the urine.

There have been no ophthalmologic abnormalities associated with it. Sulfite oxidase is a rare defect and involves cysteine metabolism. This causes increased excretion of S-sulfocysteine, taurine, sulfite, and thiosulfate.

The importance for ophthalmology is that these children have dislocated lenses. They also have other neurologic abnormalities. Although this is obviously a rare metabolic defect, it deserves to be mentioned since it should be added to the differential diagnosis of dislocated lenses. (3 figures, 10 references) —David Shoch

Ophthalmology

NATURAL HISTORY OF VITREOUS HEMORRHAGE IN DIABETIC RETINOPATHY. Ziemianski, M. C., McMeel, J. W., and Franks, E. P. (Dept. Retina Res., Eye Research Inst. of Retina Foundation, Boston, Mass.). Ophthalmol. 87:306, 1980.

Eighty-five eyes with severe vitreous hemorrhage on initial visit were followed for three to ten years without treatment. A comparison of initial and final visual acuities showed vision to have worsened in 36 eyes (42%), improved in 25 eyes (30%) and remained unchanged in 24 eyes (28%). Vision in 60 eyes was 5/200 or worse when last examined. Of 42 eyes followed three to six years (group 1), the low vision in 24 eyes (57%) was due to vitreous hemorrhage, while of 18 eyes that were followed seven to ten years (group 2), six (33%) had low vision due to vitreous hemorrhage. Traction detachments were found in five eyes (28%) in group 2 as compared with three (7%) in group 1. The study showed that ultimately 68% of the eyes had a visual acuity of 5/200 or worse. (7 figures, 3 tables, 13 references)—Authors' abstract

MANAGEMENT OF INFECTIOUS ENDOPHTHALMITIS. Forster, R. K., Abbott, R. L., and Gelender, H. (Bascom Palmer Eye Inst., Anne Bates Leach Eye Hosp., Univ. Miami, Miami, Fla.). Ophthalmol. 87:313, 1980.

Diagnostic anterior chamber and vitreous aspiration confirmed an infectious etiology in 78 of 140 eyes (56%) with suspected endophthalmitis. In 27 eyes the vitreous aspirate was positive, while the anterior chamber aspirate was negative. Intraocular antibiotics were used in 88 eyes including 50 which underwent therapeutic vitrectomy. Vision of 6/6 (20/20) to 6/12- (20/400) was achieved in 57% of recently operated, culture-positive eyes treated with intraocular antibiotics, and in 59% of those treated with combined vitrectomy and intraocular antibiotics. (4 tables, 18 references)—Authors' abstract

CILIO-OPTIC VEIN ASSOCIATED WITH PHAKOMATOSIS. Zaret, C. R., Choromokos, E. A., and Meisler, D. M. (Depts. Ophthalmol., and Neurol., Northwestern Univ., Chicago, Il.). Ophthalmol. 87:330, 1980.

This presentation demonstrates the fluorescein angiographic characteristics of the cilio-optic vein. These congenitally enlarged vessels appear at the disk edge and dip into the optic nerve to anastomose with branches of the central retinal vein. Fluorescein angiography shows lamellar filling of the vessels in the early choroidal phase. In one patient, these findings are demonstrated bilaterally with the use of bilateral simultaneous angiography. A second type of communication is presented, a retinociliary vein which drains the retina into the choroidal circulation. In both types presented, a branching hypofluorescent pattern extends from the disk vessel into the choroid. The patients described here had either neurofibromatosis or the Sturge-Weber syndrome. This anomalous disk vessel should not be confused with optociliary shunt, disk neovascularization, cilioretinal artery, or arteriovenous shunt. (19 figures, 2 tables, 14 references)—Authors' abstract

NEWS ITEMS

EDITED BY THOMAS CHALKLEY, M.D.

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For adequate publicity, notices of postgraduate courses, meetings, and lectures must be received at least four months before the date of occurrence.

INTERNATIONAL SOCIETY ON METABOLIC EYE DISEASE: FIFTH SYMPOSIUM

The Fifth Symposium of the International Society on Metabolic Eye Disease will be held in New York City, Oct. 24-26, 1982. For further information, write Heskel M. Haddad, M.D., Program Chairman, 1200 Fifth Ave., New York, NY 10029.

TULANE MEDICAL CENTER: VITRECTOMY WORKSHOP

A vitrectomy workshop will be held at the Tulane Medical Center, Dec. 5 and 6, 1980. The registration fee is $450. For further information, write Office of Continuing Education, Tulane Medical Center, 1430 Tulane Ave., New Orleans, LA 70112.

WILLS EYE HOSPITAL: PEDIATRIC OPHTHALMOLOGY UPDATE 1980

Wills Eye Hospital will sponsor a seminar, "Pediatric Ophthalmology Update 1980," Sept. 26 and 27, 1980. For further information write Ms. Wilson, Pediatric Ophthalmology Seminar, P.O. Box 388, Philadelphia, PA 19105.

BAYLOR COLLEGE OF MEDICINE: CONTACT LENS TECHNOLOGY COURSE

The annual Baylor College Contact Lens Technology Course will be held at the Cullen Eye Institute, Dec. 4-6, 1980. For further information, write David Paton, M.D., Professor and Chairman, Cullen Eye Institute, Baylor College of Medicine, 6501 Fannin (NC 200), Houston, TX 77030.

NEW YORK MEDICAL COLLEGE: SEMI-ANNUAL OCULOPLASTIC SURGICAL DISSECTION COURSE

The New York Medical College will sponsor its semi-annual oculoplastic surgical dissection course Oct. 22-25, 1980. The fee is $65. For further information, write Ms. P. Tamkin, c/o Dr. P. Guibor, 630 Park Ave., New York, NY 10021.

SOCIETY OF GERIATRIC OPHTHALMOLOGY: SIXTH ANNUAL MEETING

The Society of Geriatric Ophthalmology has been accepted as part of the Satellite group by the American Academy of Ophthalmology. It will hold its sixth meeting in Chicago Nov. 4, 1980, at the Conrad Hilton Hotel. For further information, write A. L. Kornzweig, M.D., Secretary-Treasurer, Jewish Home and Hospital for Aged, 120 W. 106th St., New York, NY 10025.

LONG ISLAND JEWISH-HILLSIDE MEDICAL CENTER: CLINICAL DAY ON ORBITAL DISEASE

The Department of Ophthalmology of the Long Island Jewish-Hillside Medical Center and the Health Sciences Center of the State University of New York will sponsor a clinical day on orbital disease, March 4, 1981, in New Hyde Park, New York. For further information, write Continuing Education Coordinator, Long Island Jewish-Hillside Medical Center, New Hyde Park, NY 11042.

WENNER-GREN CENTER AND SMITH-KETTLEWELL EYE RESEARCH FOUNDATION INTERNATIONAL SYMPOSIUM

A Wenner-Gren Center and Smith-Kettlewell Eye Research Foundation International Symposium on Functional Basis of Ocular Motility Disorders will be held in Stockholm, Sweden, Aug. 31 to Sept. 3, 1981. For further information, write The Symposium Secretariat, Wenner-Gren Center, Sveavagen 166, S-113 46 Stockholm, Sweden.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM: ANNUAL CONGRESS

The annual congress of the Ophthalmological Society of the United Kingdom will be held at the University of Southampton, Medical School, April 8-10, 1981. For further information, write Miss M. M. Hallendorff, Ophthalmological Society of the United Kingdom at the Royal College of Surgeons, 35/43 Lincoln's Inn Fields, London WC2A 3PN England.

STATE UNIVERSITY OF NEW YORK: POSTGRADUATE COURSE IN OPHTHALMOLOGY

The Department of Ophthalmology of the State University of New York Upstate Medical Center in Syracuse will present its 31st annual postgraduate course in ophthalmology, Dec. 5 and 6, 1980. The tuition fee is $70 and the residents' fee is $35. For further information, write James L. McGraw, M.D.,

State University of New York, Upstate Medical Center, 750 E. Adams St., Syracuse, NY 13210.

Pan-American Ophthalmologic Microsurgery Society: Annual Meeting

The Pan-American Ophthalmologic Microsurgery Society will hold its annual meeting May 9, 1981, in Acapulco, Mexico, on the day preceding the XIII Pan American Ophthalmology Congress.

American Intra-Ocular Implant Society: Fourth Annual Intraocular Lens Symposium

The fourth annual United States Intraocular Lens Symposium will be held March 25-28, 1981. For further information, write Ms. Debbi Miller, Director of Meeting Services, American Intra-Ocular Implant Society, P.O. Box 3140, Santa Monica, CA 90403.

Washington State Academy of Ophthalmology: Annual President's Meeting

The Annual President's Meeting of the Washington State Academy of Ophthalmology will be held in Seattle, Jan. 30 and 31, 1981. For further information, write Washington State Academy of Ophthalmology, 2033 Sixth Ave., Suite 900, Seattle, WA 98121.

Long Island Jewish-Hillside Medical Center: Ophthalmology Seminars 1980-1981

Long Island Jewish Hillside Medical Center, New Hyde Park, New York, will hold the following ophthalmology seminars on Mondays from 8 P.M. to 9:30 P.M.:

Oct. 13, 1980, Ocular Motility: Management of Superior Oblique Paresis Syndromes; Nov. 10, 1980, Vitrectomy: Vitrectomy Techniques for the Anterior Segment Surgeon; Feb. 9, 1981, Topics in Glaucoma—Laser Iridectomy; and April 13, 1981, Clinical Ultrasonography—The Role of Ultrasonagraphy in Directing Patient Management.

For further information, write Continuing Education Coordinator, Long Island Jewish-Hillside Medical Center, New Hyde Park, NY 11042; telephone (212)-470-2114.

Wilmer Institute of Johns Hopkins Hospital: Third Annual Current Concepts in Ophthalmology Course

The Wilmer Institute of Johns Hopkins Hospital will present the Third Annual Current Concepts in Ophthalmology Course, Jan. 10-17, 1981, in Puerto Rico. For further information, write Program Coordinator, Johns Hopkins University School of Medicine, Office of Continuing Education, 720 Rutland Ave., Baltimore, MD 21205.

Miami Ophthalmological Society: 1980-1981 Board of Directors

The new Board of Directors for the Miami Ophthalmological Society are as follows: David J. Singer, president; Stephen M. Kulvin, vice-president; Ira G. Weiner, secretary; Dale G. Davis, recording secretary; Lee R. Duffner, treasurer; and Ricardo Martinez-Ferrer, immediate past-president.

Rabies Vaccines Protect 74 Exposed Through Eye Graft

The 73 men, women, and children who were immunized against rabies late in 1978 with the new human-fetal-lung-tissue vaccine "all developed excellent antibody titers," according to viral epidemiologist Larry J. Anderson of the Center for Disease Control in Atlanta. Most were medical and hospital personnel who had close contact with the donor or recipient of a corneal transplant which later proved to be infected with rabies virus. A diagnosis of rabies," urged Dr. Anderson, "should be considered for any patient with severe atypical Guillian-Barré symptoms or who has progressive encephalitic myelitis."

Correction

In the article, "Long-term follow-up of occlusion of the central retinal vein in young adults," by Ira A. Priluck, Dennis M. Robertson, and Robert W. Hollenhorst (Am. J. Ophthalmol. 90:190-202, 1980), the printer mistakenly substituted a color plate from another article. Please remove the incorrect color plate following page 196, and replace it with the correct color plate on the facing page. Reprints have been corrected.

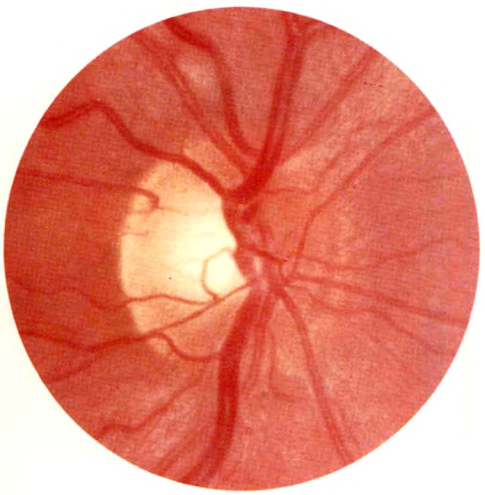


Fig. 6 (Priluck, Robertson, and Hollenhorst). Optic disk of a 31-year-old man who earlier had cilioretinal artery occlusion of unknown cause.

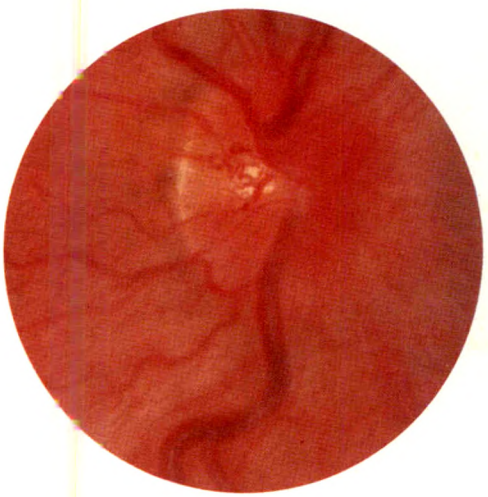


Fig. 7 (Priluck, Robertson, and Hollenhorst). Six months later there is evidence of incomplete central vein occlusion, with engorgement of veins, swelling of optic nerve, and many, small, peripapillary hemorrhages and exudates. Anomalous vasculature is visible on temporal optic nerve head.

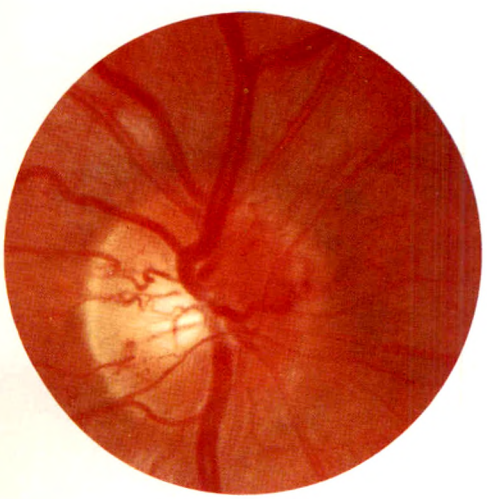


Fig. 8 (Priluck, Robertson, and Hollenhorst). One year later numerous collateral venous channels are visible. Disk swelling has subsided; hemorrhages and exudates have absorbed. A prominent opticociliary vein has developed along the 3 o'clock disk meridian.

New Research Grants Awarded by the National Eye Institute
April 1, 1980 to June 30, 1980

Research Project Grants

Institution	Principal Investigator	Title of Project	Amount Awarded
University City Science Center Philadelphia, Pennsylvania	Robert Alper	Corneal structural glycoproteins	$ 57,979
Harvard University Boston, Massachusetts	Robert W. Baughman	Neurotransmitters in the central visual pathway	$110,799
Adelphi University Garden City, New York	Frederick A. Bettelheim	The interaction of phosphopeptides with lens proteins	31,424
College of Medicine and Dentistry of New Jersey Newark, New Jersey	Annette Beyer-Mears	Aldose reductase inhibitors administered topically	54,692
Eye Research Institute of Retina Foundation Boston, Massachusetts	Sheldon M. Buzney	Properties of retinal vascular cells in tissue culture	51,844
Syracuse University Syracuse, New York	Steven C. Chamberlain	Adaptive mechanisms that maintain visual sensitivity	50,819
W. Alton Jones Cell Science Center Lake Placid, New York	Robert L. Church	Genetic dissection of ocular connective tissues	134,675
Estelle Doheny Eye Foundation Los Angeles, California	Mark C. Citron	Local circuitry for pattern recognition in the retina	57,250
Massachusetts Eye and Ear Infirmary Boston, Massachusetts	John V. Donlon, Jr.	Stress response to eye surgery. Plasma catecholamines	27,100
Wayne State University Detroit, Michigan	Edward S. Essner	Structure and permeability of the choriocapillaris	36,679
University of Texas Health Science Center Houston, Texas	Robert E. Ferrell	Genetic studies of retinoblastoma and the RB-1 locus	31,738
University of Iowa Iowa City, Iowa	Jonathan D. Fratkin	Proliferation of retinal vascular endothelium in vitro	10,000
Oakland University Rochester, Michigan	Frank J. Giblin	Role of sulfhydryl groups in X-ray and other cataracts	49,949
Eye Research Institute of Retina Foundation Boston, Massachusetts	Jeffrey P. Gilbard	Drying of the ocular surface	52,350
Johns Hopkins University Baltimore, Maryland	Bert M. Glaser	Ocular angiogenic factors. Bioassays and biochemistry	57,036
University of Mississippi Medical Center Jackson, Mississippi	James B. Grogan	Antigen exposure via the anterior chamber of the eye	75,035
New York Medical College Valhalla, New York	Anthony Herp	Protease inhibition in corneal ulcers	55,360
Santa Clara Valley Medical Center San Jose, California	John R. Hoston	Supranuclear control of fixation eye movements	36,138
University of California Davis, California	Cristina M. Keeney	Descemet's membrane in corneal disorders	34,175

Institution	Principal Investigator	Title of Project	Amount Awarded
Medical University of South Carolina Charleston, South Carolina	Peter W. Land	Ganglion cells in rat retina	$ 43,116
University of Pennsylvania Philadelphia, Pennsylvainia	Alan M. Laties	The eye in experimental diabetes. Retinal changes	94,499
Yeshiva University New York, New York	Charles R. Legendy	Extraretinal influences on striate cortex neurons	97,544
University of Michigan Ann Arbor, Michigan	Paul R. Lichter	Carbonic anhydrase inhibitors in treating glaucoma	107,639
Columbia University New York, New York	Jong J. Lim	Endothelial cell adhesion and transport mechanisms	48,589
Zweng Memorial Retinal Research Foundation Menlo Park, California	Hunter L. Little	Alteration of hemorheology and diabetic retinopathy	8,915
Louisiana State University Medical Center New Orleans, Louisiana	James G. May	Far-field recording of optic nerve responses	45,001
Medical University of South Carolina Charleston, South Carolina	Steven C. McLoon	Neuronal patterns in the developing visual system	12,250
Mount Sinai School of Medicine New York, New York	Joel S. Mindel	Depolarizing nicotinic antagonists and eye position	96,207
University of California Los Angeles, California	Dale L. Phelps	Oxygen therapy of oxygen induced retinopathy	64,446
University of Michigan Ann Arbor, Michigan	Anne C. Rusoff	Paths of individual axons in the visual system	53,736
Tulane University of Louisiana New Orleans, Louisiana	Guenther Schoellmann	Some molecular aspects of photoreceptor function	$ 40,255
Pennsylvania College of Optometry Philadelphia, Pennsylvania	John B. Siegfried	Oscillatory potentials in the visual system	34,931
Albany Medical College Albany, New York	Kurt Simons	Fusion in infants at risk for heterotropia	52,950
Beth Israel Medical Center New York, New York	Casimir A. Swinger	Refractive keratoplasty. Studies on an animal model	63,570
University of California San Francisco, California	Khalid F. Tabbara	Autoimmune disease and the eye. Sjorgren's syndrome	56,573
University of Iowa Iowa City, Iowa	H. Stanley Thompson	Pupillary and VER indicators of optic nerve disease	39,129
State University of New York Buffalo, New York	Susan B. Udin	Effect of visual experience on CNS connectivity	58,356

Institution	Principal Investigator	Title of Project	Amount Awarded
Boston University Boston, Massachusetts	Geraldine Waloga	Internal regulators of phototransduction	$ 28,600

Core Center Grants

Institution	Principal Investigator	Title of Project	Amount Awarded
Purdue University West Lafayette, Indiana	William L. Pak	Interdisciplinary studies of visual processes	$168,590

National Research Service Award Fellowships

Institution	Principal Investigator	Title of Project	Amount Awarded
Harvard University Cambridge, Massachusetts	Michael Ariel	Physiological study of photoreceptor transmitter	$ 18,380
Louisiana State University New Orleans, Louisiana	Penny A. Asbell	Immune complex model of herpetic disciform edema	22,040
University of Cambridge Cambridge, United Kingdom	Curtis L. Baker, Jr.	Visual mechanisms of relative motion perception	13,380
Eye Research Institute of Retina Foundation Boston, Massachusetts	Jettie M. Burnett	Corneal reparative response to epithelial injury	22,040
Yale University New Haven, Connecticut	Maria Czako-Graham	Messenger RNA translation in retina photoreceptor cells	19,040
Duke University Durham, North Carolina	Kathleen A. French	Processing of information from barnacles multiple eyes	18,380
Harvard University Boston, Massachusetts	Elizabeth D. Hay	Extracellular matrix in corneal morphogenesis	16,098
Darthmouth College Hanover, New Hampshire	John H. Hayden	Cytoskeletal proteins in the lens and cornea	15,817
Harvard University Cambridge, Massachusetts	Eric M. Lasater	Interactions at the inner plexiform layer of the vertebrate	18,380
University of Texas Health Science Center Houston, Texas	Paul C. Madtes, Jr.	The retinal gaba-ergic system in developing retina	18,380
Massachusetts Institute of Technology Cambridge, Massachusetts	Arthur M. Mercurio	Glycosylation of photoreceptor membrane proteins	18,380
Harvard University Boston, Massachusetts	Thomas M. Miller	Cell-free synthesis of a lens membrane protein	18,380

Institution	Principal Investigator	Title of Project	Amount Awarded
U.S. National Institutes of Health Bethesda, Maryland	Bruce A. Pfeffer	Proteoglycans from the inter-photoreceptor matrix	$ 13,380
University of California Berkeley, California	Arthur S. Polans	Synaptic chemistry in the retina of tiger salamander	18,380
Baylor College of Medicine Houston, Texas	Laurence M. Rapp	Regional differences in retinal morphology and physiology	15,817
Eye Research Institute of Retina Foundation Boston, Massachusetts	Deborah D. Sendele	Experimental xerophthalmia and keratomalacia in vitamin A	21,236
Medical College of Wisconsin Milwaukee, Wisconsin	John L. Ubels	Penetration and metabolism of retinoids in the cornea	18,380

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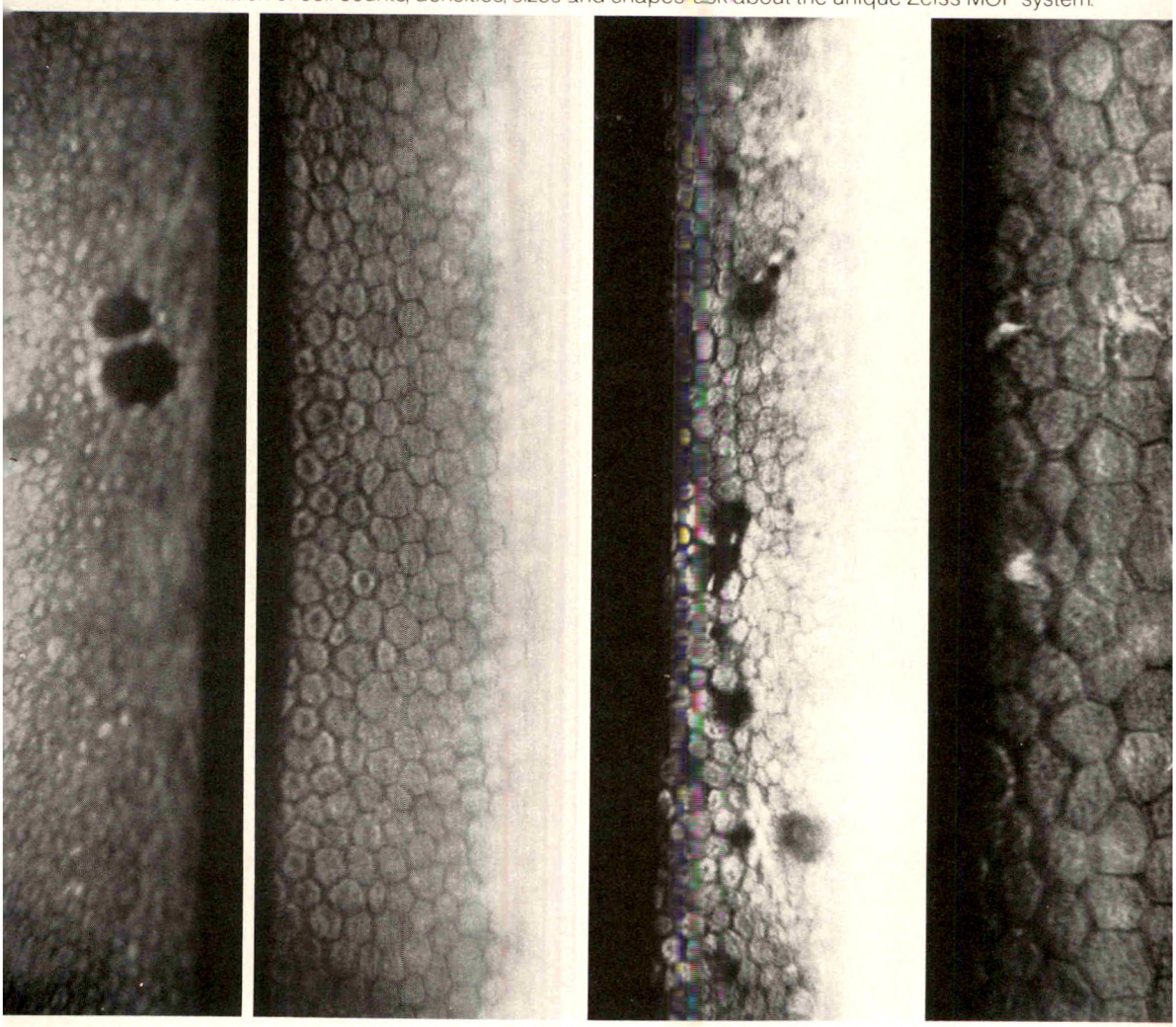
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H. Dwight Cavanagh, M.D., Atlanta, Georgia
Howard Eggers, M.D., New York, New York
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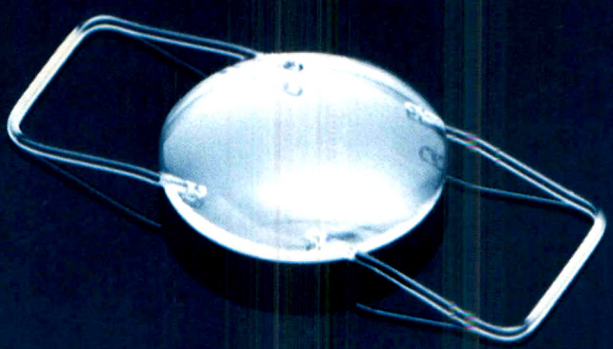
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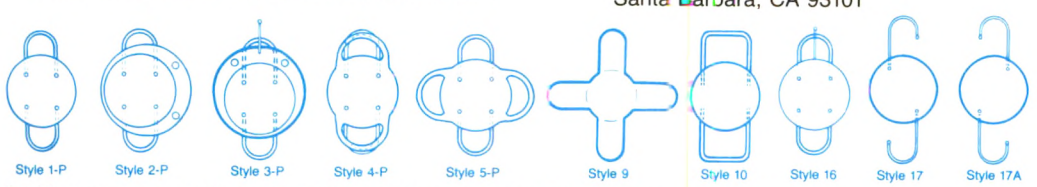
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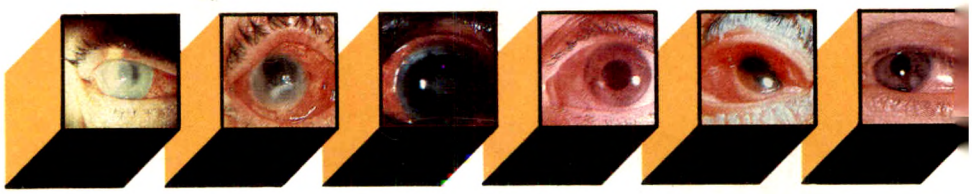
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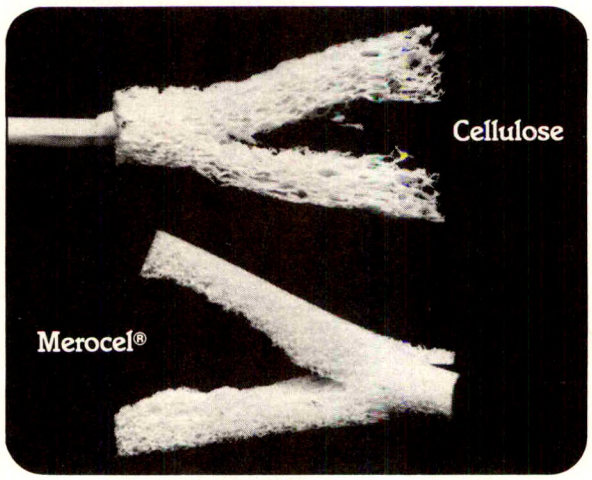
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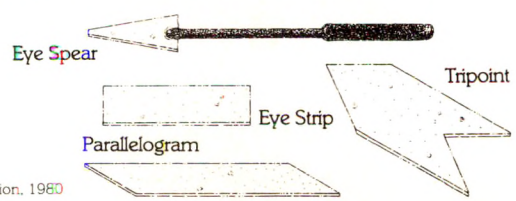
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1. Vitrectomy from the Anterior Approach, H.E. Kaufman M.D.-Ophthalmic Surgery Vol. 6 (2) 58-65



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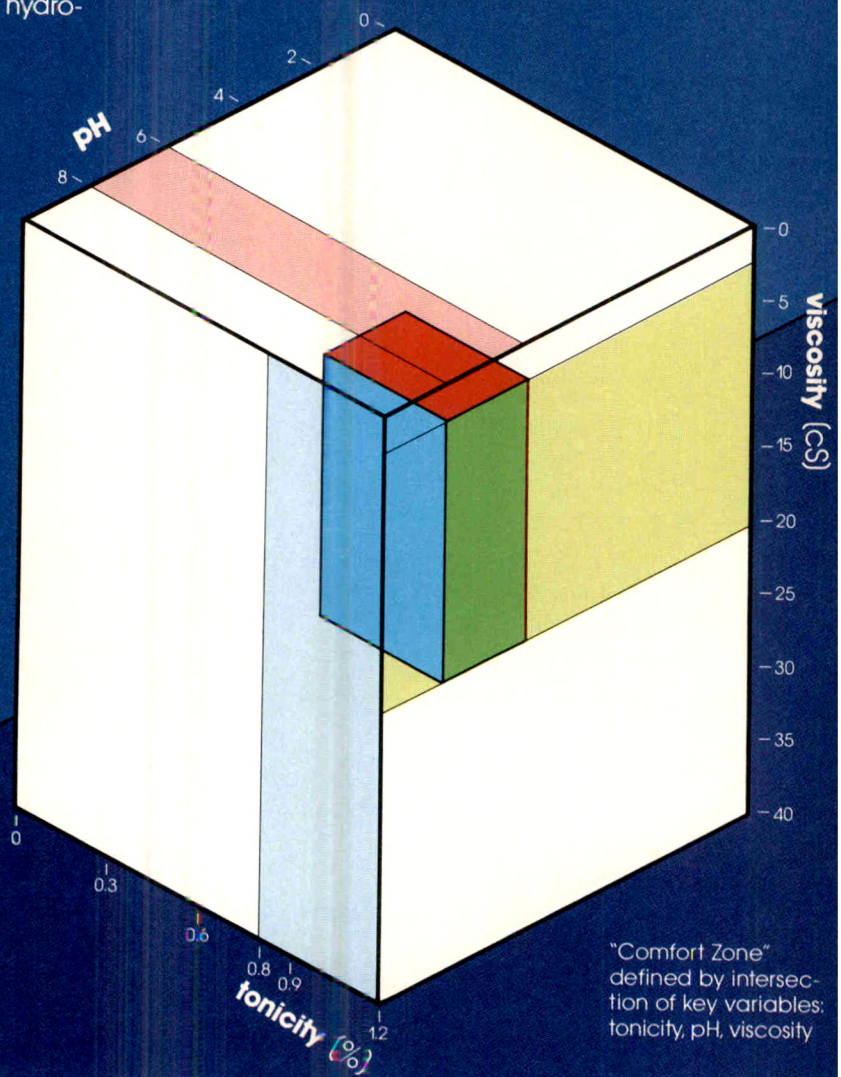
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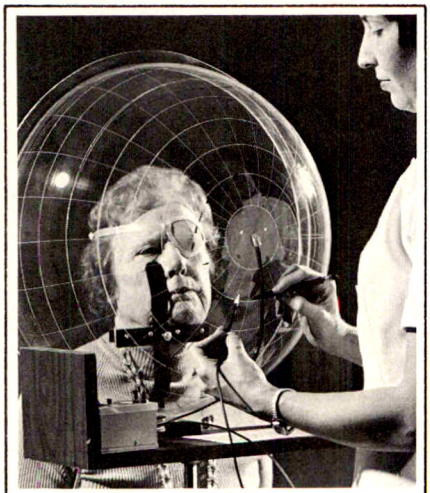
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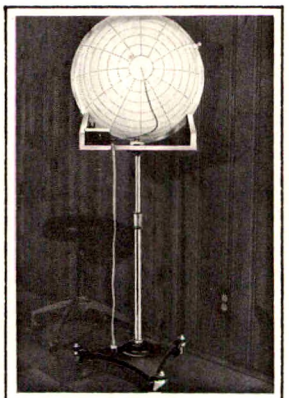
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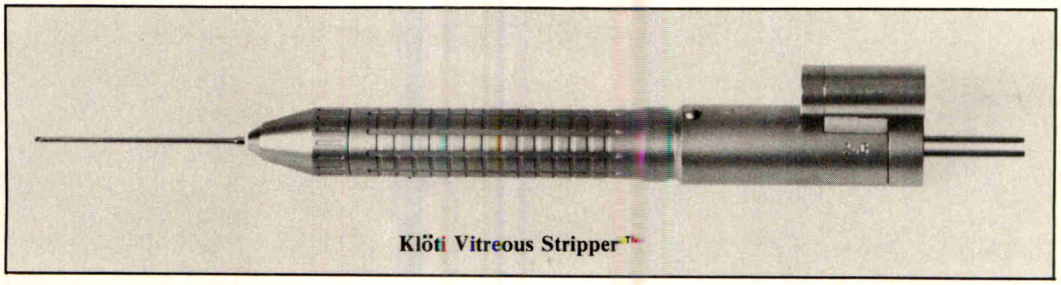
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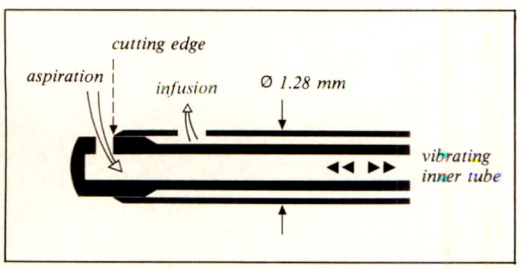
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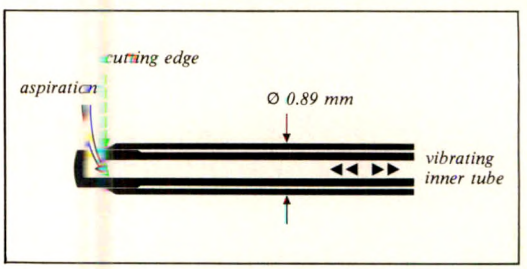
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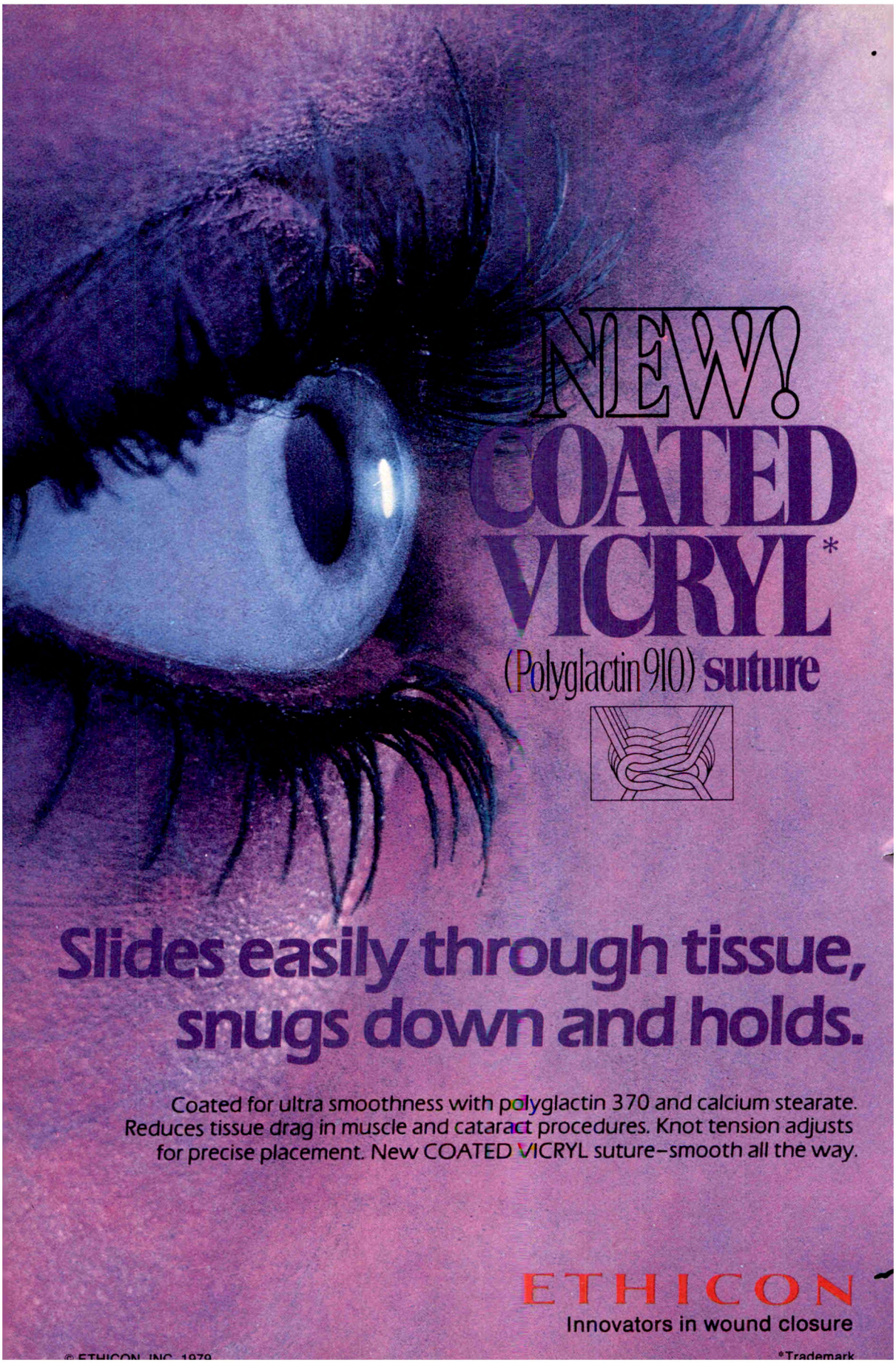
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The safety and effectiveness of VICRYL (polyglactin 910) and Coated VICRYL sutures in neural tissue and in cardiovascular tissue have not been established.

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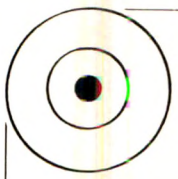
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PROGRAM

Richard H. Keates, M.D., Chairman

7:00 pm—SYMPOSIUM: Controversial Questions in Contact Lenses.
Panel: H. Dwight Cavanagh, M.D., Paul R. Honan, Jr., M.D., Perry S. Binder, M.D., R. Linsy Farris, M.D., Oliver H. Dabezies, Jr., M.D.

7:30 pm-8:15 pm Contact and Intraocular Lens Presentations

7:30 pm—Richard A. Saunders, M.D.: Empirical Fitting of Hard Contact Lenses in Infants and Young Children. *Discussant:* Perry Rosenthal, M.D.

7:41 pm—Louis A. Wilson, M.D.: Thimerosal Hypersensitivity: A Cause of Ocular Redness and Irritation amongst Patients Wearing Soft Contact Lenses. *Discussant:* Rudolf M. Franklin, M.D.

7:52 pm—A. Robert Bellows, M.D.: Endophthalmitis in Aphakic Patients with Inadvertent Filtering Blebs Wearing Contact Lenses. *Discussant:* Frederick H. Theodore, M.D.

8:04 pm—J. V. Greiner, M.D.: Effect of Contact Lens Wear on the Conjunctival Mucus Systems. *Discussant:* Roswell Pfister, M.D.

8:15 pm—SYMPOSIUM: Controversial Questions in Implants and Refractive Surgery.
Panel: Richard P. Kratz, M.D., Henry M. Clayman, M.D., Jerald L. Tennant, M.D., Kenneth J. Hoffer, M.D., Stephen A. Obstbaum, M.D.

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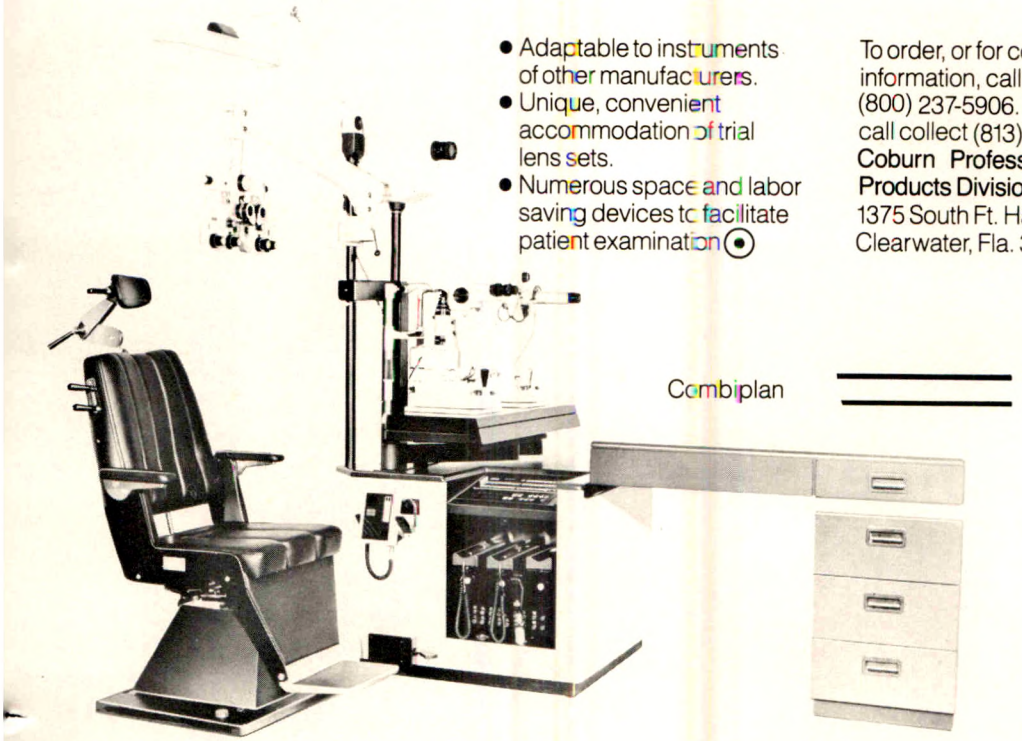


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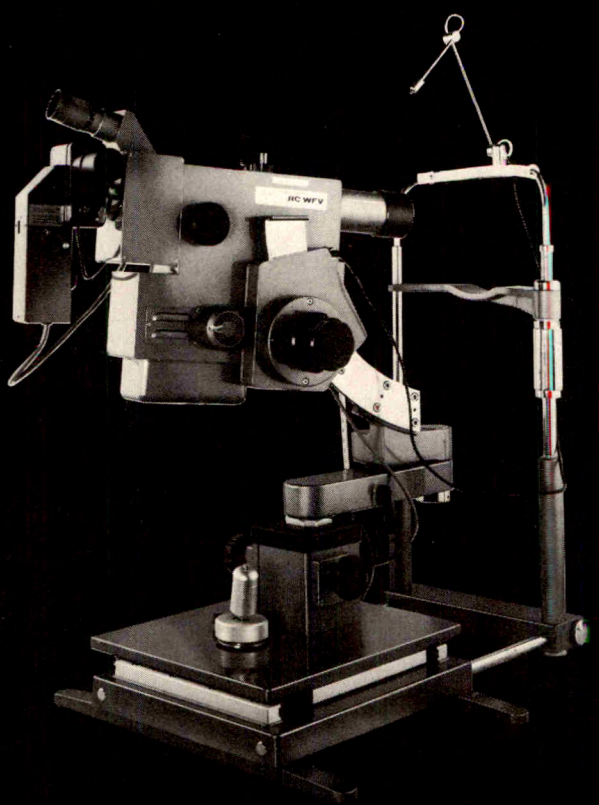
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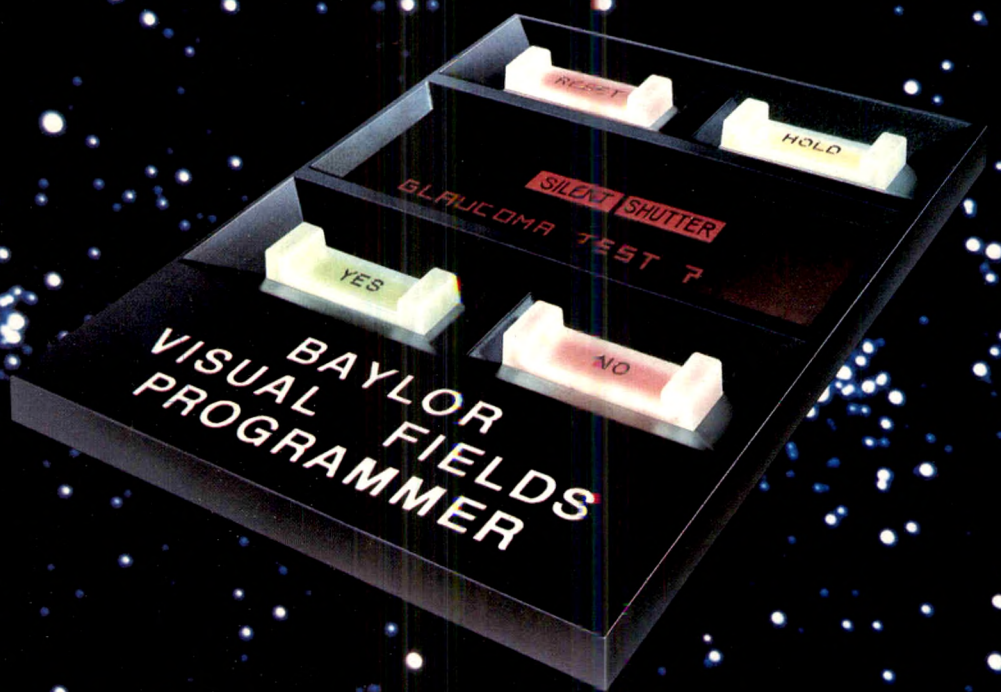


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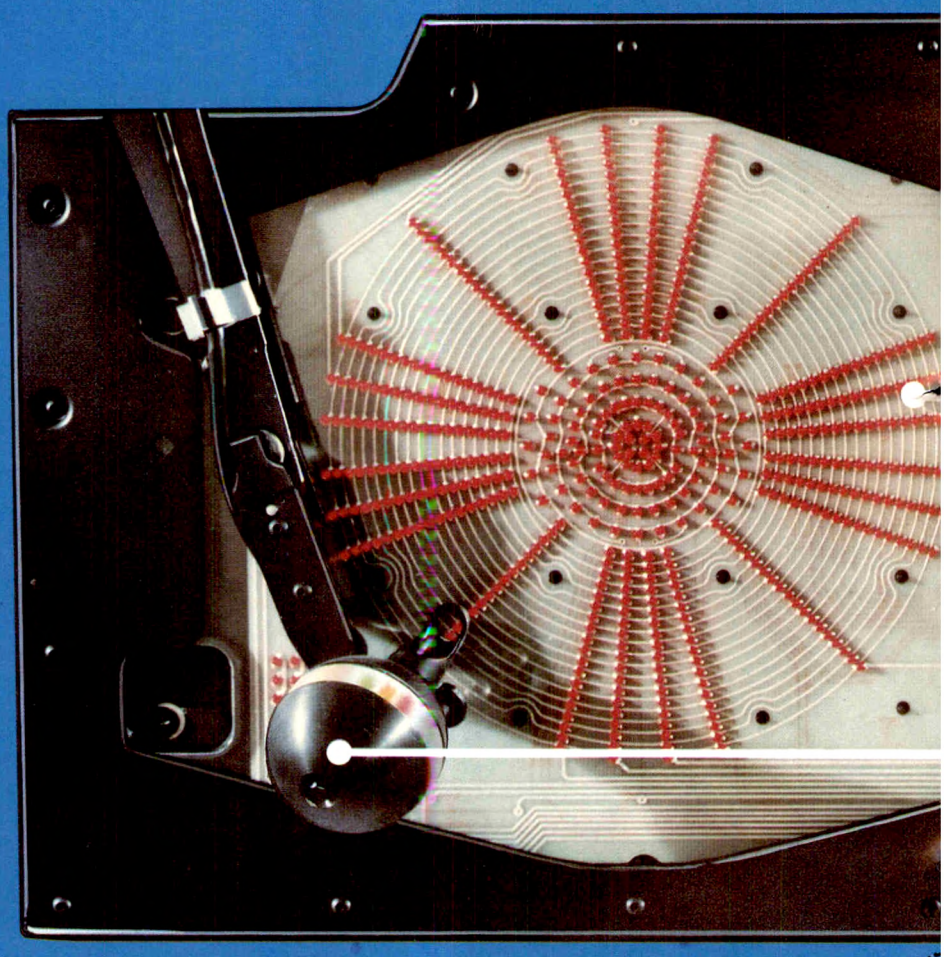
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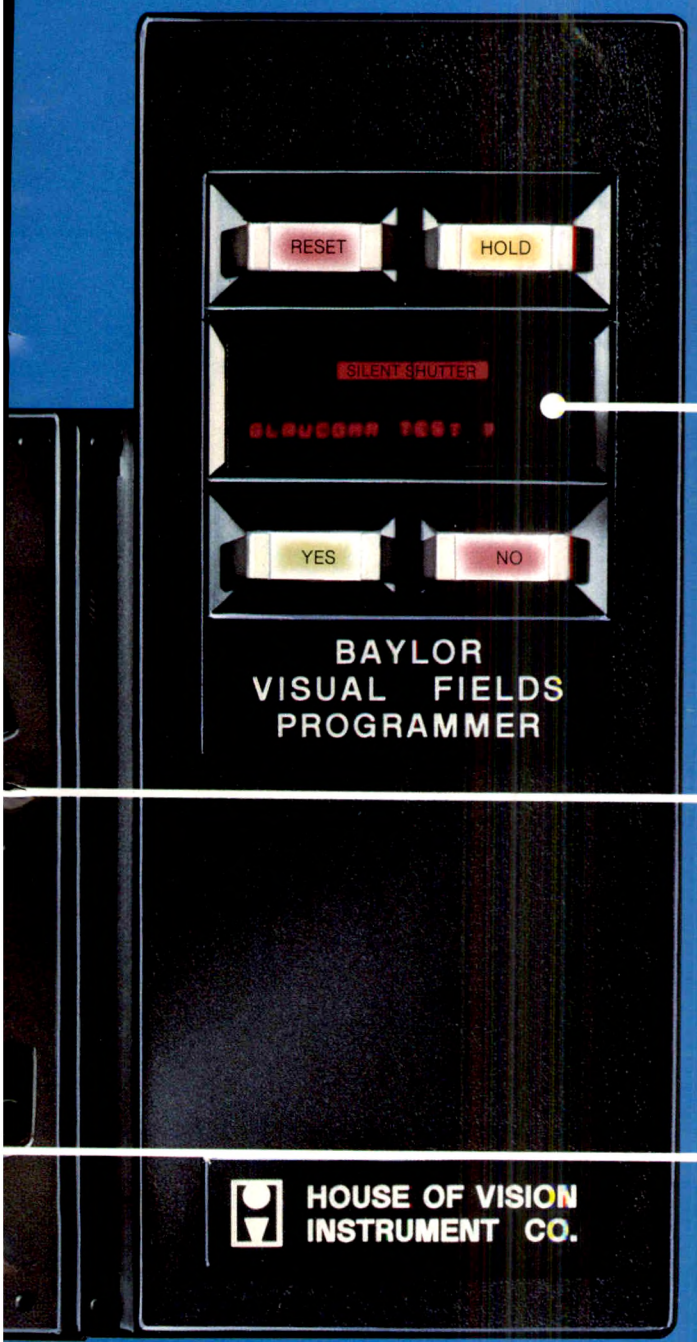
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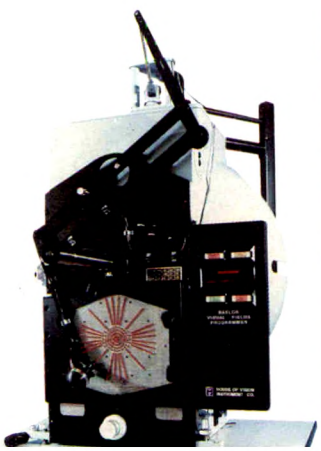
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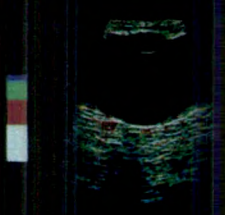


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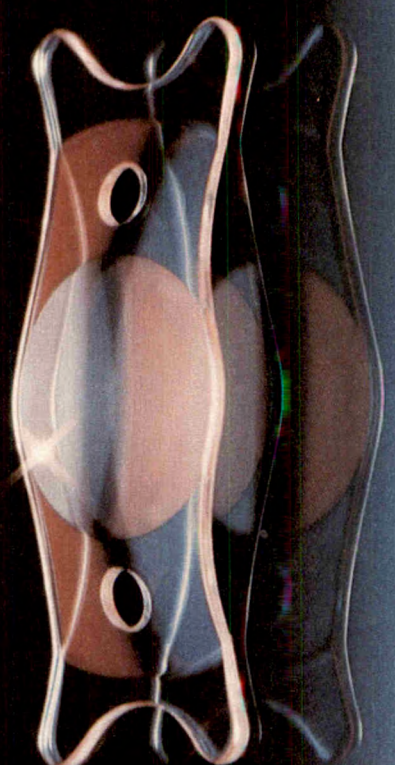
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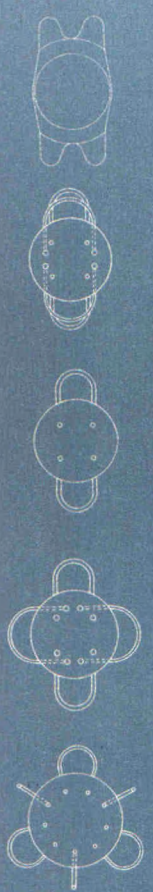
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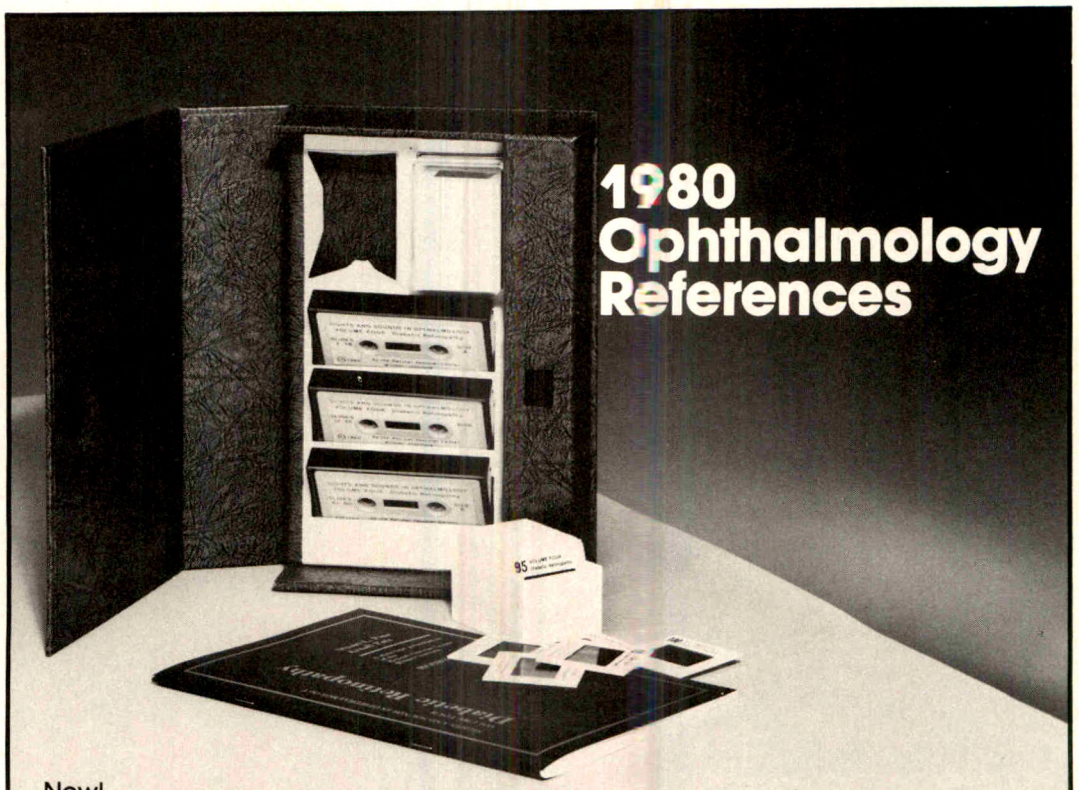


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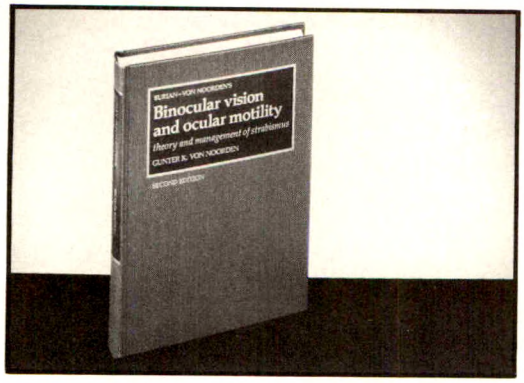
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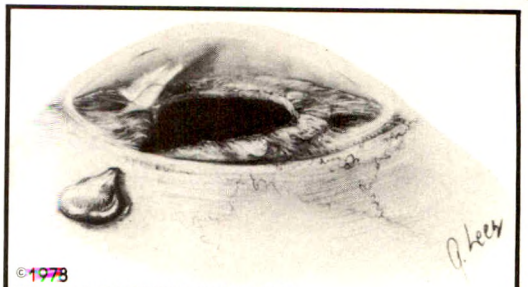
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Fig. 86-11.

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Fig. 86-11. A, Vitreous wick syndrome characterized by prolapse of vitreous gel through limbal wound onto external surface of globe.

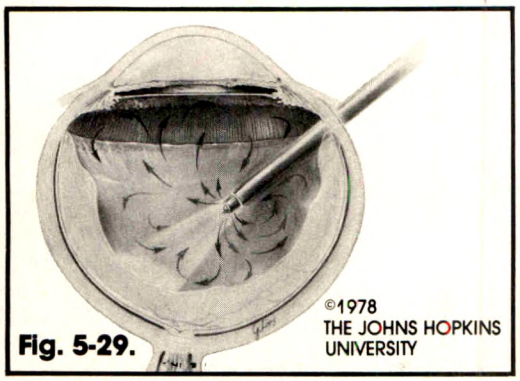


Fig. 5-29. Intact anterior lens-capsule protects anterior-chamber tissues from bulk flow of irrigating solution and particulate matter during vitrectomy. Also, air bubbles inadvertently introduced through infusion system remain posterior to lens capsule and are easily removed.

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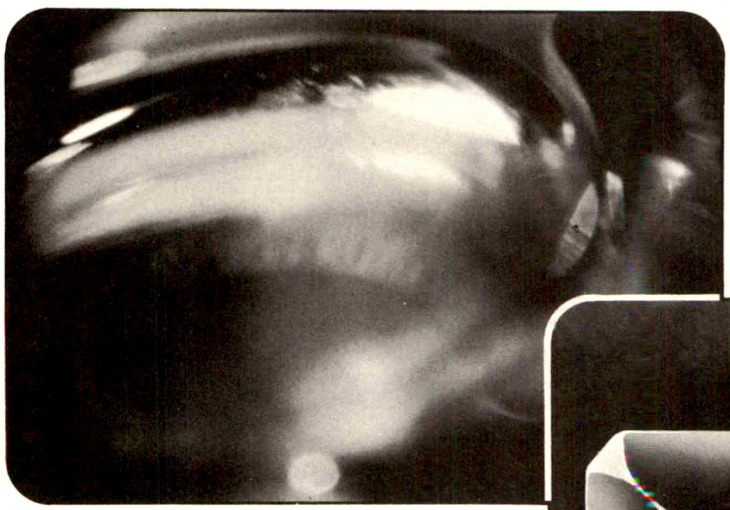
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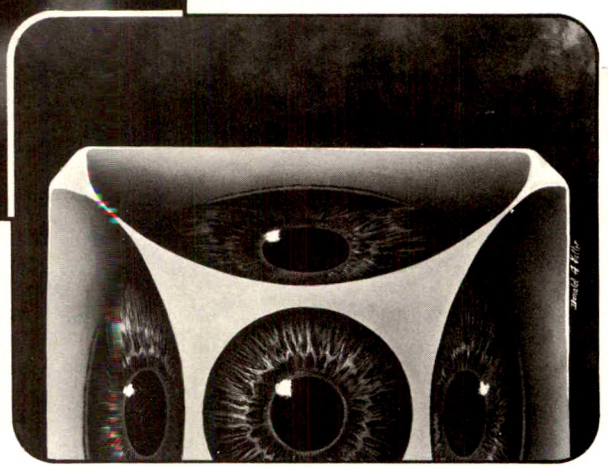
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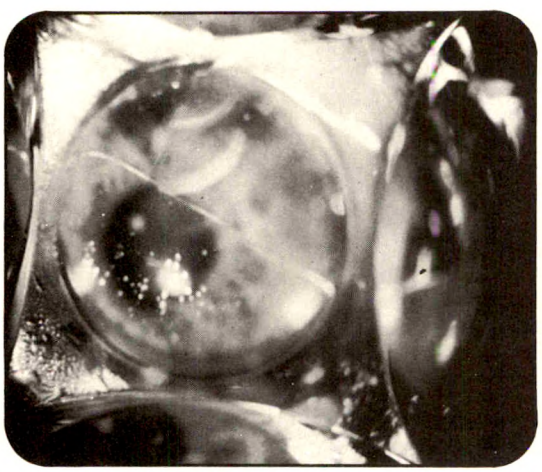
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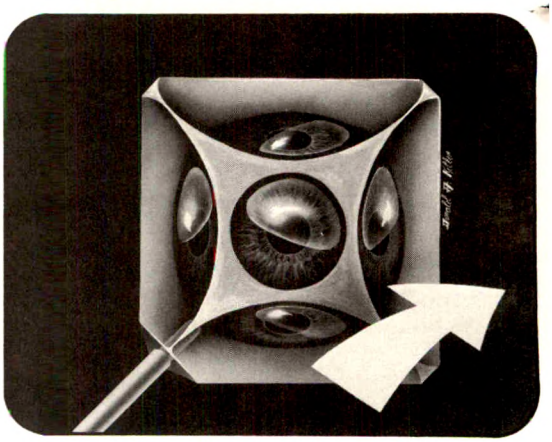


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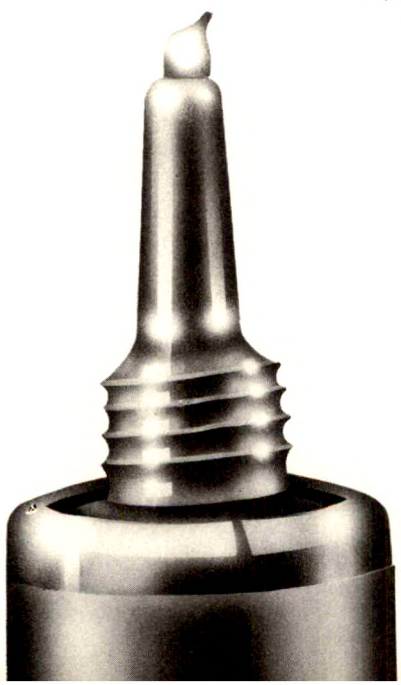
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☐ ***Because of your specialized knowledge and training,*** we believe that only you should prescribe the Cortisporin® ophthalmic products—and that's why they are promoted only to ophthalmologists.

☐ ***For your patients*** the Cortisporin ophthalmic products provide a wide therapeutic spectrum. The antibacterials act together against susceptible bacterial pathogens commonly involved in the ophthalmic infections accessible to topical therapy.* The hydrocortisone component gently and rapidly relieves signs and symptoms of inflammation and inhibits corneal neovascularization.

☐ ***Economical, too,*** with the two sterile forms providing convenient day and night coverage at lower cost than most other major brands of similar activity.

Cortisporin® Ophthalmic Suspension Sterile

(Polymyxin B–Neomycin–Hydrocortisone)

Each cc. contains: Aerosporin® brand Polymyxin B Sulfate, 10,000 Units; neomycin sulfate (equivalent to 3.5 mg. neomycin base), 5 mg.; hydrocortisone, 10 mg. (1%); thimerosal (preservative), 0.001%.
The vehicle contains the inactive ingredients cetyl alcohol, glyceryl monostearate, liquid petrolatum, polyoxyl 40 stearate, propylene glycol and purified water.

Cortisporin® Ophthalmic Ointment Sterile

(Polymyxin B–Bacitracin–Neomycin–Hydrocortisone)

Each gram contains: Aerosporin® brand Polymyxin B Sulfate 5,000 Units; zinc bacitracin 400 Units; neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base); hydrocortisone 10 mg (1%); special white petrolatum qs.

***INDICATIONS:** Based on a review of these drugs by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective: For the treatment of nonpurulent bacterial infections of the eye due to organisms sensitive to the antibiotic ingredients of the drugs and when the anti-inflammatory action of the hydrocortisone is indicated as in nonpurulent bacterial, allergic, vernal and phlyctenular conjunctivitis; nonpurulent blepharitis; interstitial, sclerosing, postoperative or acne rosacea keratitis; superficial chemical and thermal burns of the cornea.

Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS: These products are contraindicated in acute purulent conjunctivitis and blepharitis; tuberculous, fungal or viral lesions of the skin or eye, including herpes simplex, dendritic keratitis, vaccinia or varicella; and conditions involving the posterior segment of the eye. They are also contraindicated in those individuals who have shown hypersensitivity to any of their components.

PRECAUTIONS: Extended ophthalmic use of topical steroid therapy may result in glaucoma with possible damage of the optic nerves, posterior subcapsular cataract formation, or may aid in the establishment of secondary ocular infections from fungi or viruses liberated from ocular tissues. It is advisable that intraocular pressure be checked frequently. In those diseases causing thinning of the cornea, perforation has been known to occur with the use of topical steroids. As with any antibiotic preparation, prolonged use may result in the overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Articles in the current medical literature indicate an increase in the prevalence of persons allergic to neomycin. The possibility of such a reaction should be borne in mind.

Complete literature available on request from Professional Services Dept. PML.

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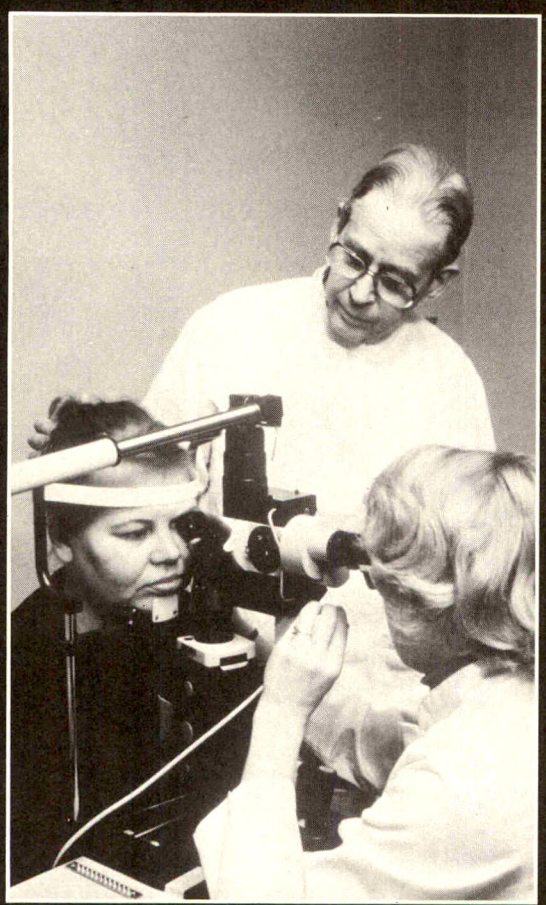
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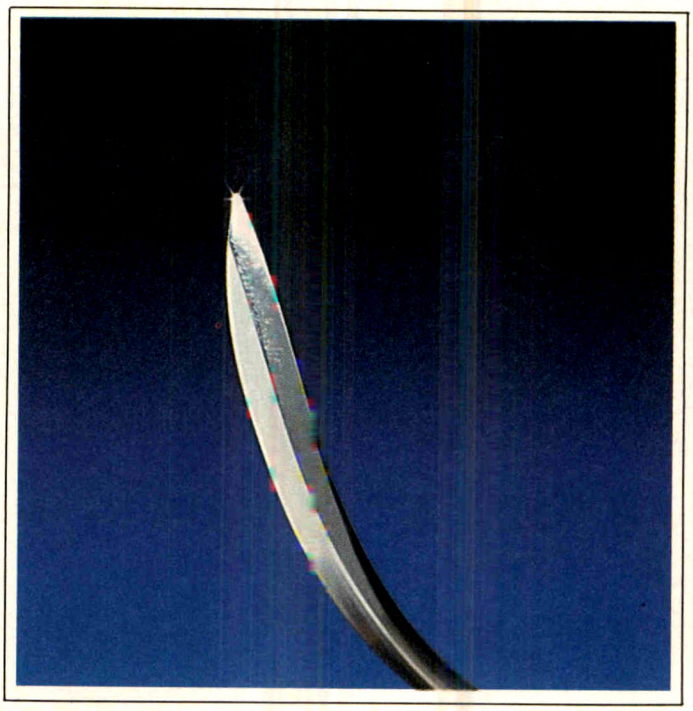
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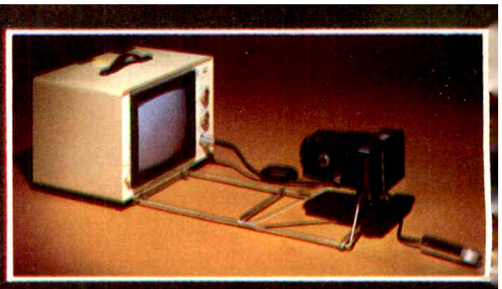
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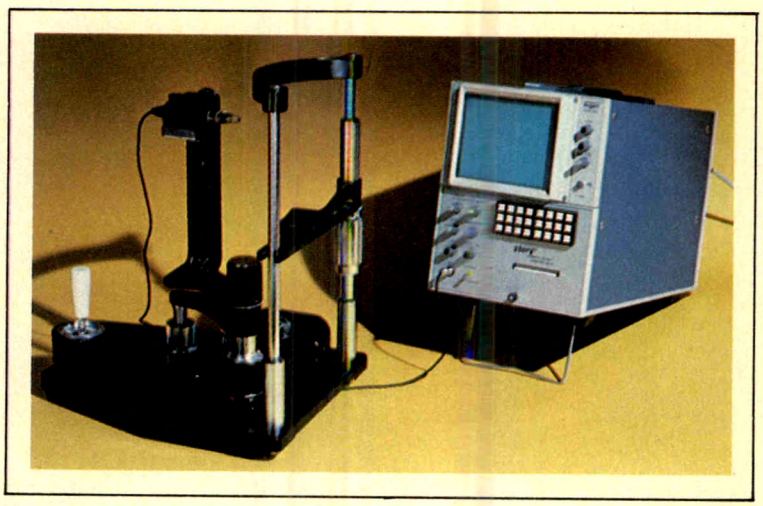
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Think of your next patient as 15 pieces of paperwork.

You may not realize it, but every patient who enters your office creates as many as 15 separate pieces of paperwork.

And no matter how small your practice is, handling those ledger cards, bills, files, and insurance forms (to name only a few) takes thousands of staff hours a year, as well as your own time. Time you could be putting to better use.

But the Acuity Systems office computer drastically cuts the time spent on paperwork. Because it keeps complete patient accounts, types up insurance forms, and schedules appointments. It also produces itemized bills and daily bank deposit slips. And it performs a variety of other time-consuming daily record-keeping chores quickly and accurately.

It also functions as a word processor. In this mode, it can search your records for patients who need to be recalled, and then type labels for reminder postcards. Plus it can generate personalized form letters for insurance inquiries, referral replies, and other repetitive correspondence.

What's more, the Acuity Systems office computer stores all its files electronically on magnetic disks. This allows your staff instant access to records, speeding answers to patient inquiries, and improving patient relations.

You get a better cash flow, plus vital financial data.

Because Acuity Systems' office computer does everything with incredible speed, patients can receive bills on the spot, and insurance claims can be processed the same day.

And, it also keeps track of aging accounts for you. At the touch of a button, it can produce a list of past-due patients your staff can utilize for an on-going collection program.

As a result, collectibles are greatly reduced. So you'll have a steadier, more reliable cash flow that will allow you to manage your funds better.

In addition, the Acuity Systems office computer automatically prepares a variety of important financial reports, including trial balance, profit and loss statements, and balance sheets. These reduce your need for costly accounting services. And they give you a day-to-day understanding of your financial position you may never have had before.



The Acuity Systems' office computer, part of the Intelligent Office™ system of practice management. Shown are the display terminal with keyboard, high-speed printer, and disk drive. The disk drive can also be placed in a closet, allowing the terminal and printer to be placed on a desk top.

It puts your finger on the pulse of your practice.

In addition to performing all your daily bookkeeping and billing functions, Acuity Systems' office computer also compiles a variety of practice management reports, including:

- practice income by doctor, procedure, day, or any of a variety of other variables.
- revenue analysis, allowing you to monitor your sources of income.
- inventory control (for practitioners who dispense).

With these reports, you'll be able to monitor your own workload, or that of other members of your practice. You'll be able to see where your income comes from, and where you may be able to reduce your overhead. And you'll have the information you need to manage your practice effectively.

What's more, since the computer is completely contained in your office, only you have access to this confidential patient and practice information.

It speaks plain English, so it's easy to operate.

You might think that a computer system this sophisticated would

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PRACTICE PERFECT.

need its own programmer. But the Acuity Systems office computer doesn't, because it's preprogrammed. In fact, it's so easy to use that your secretary can be trained to run it in just a few hours. There's no special computer language to learn, because it actually prompts your secretary with simple, step-by-step instructions. Which means it's almost impossible to make a mistake.

What all this means is that, whether yours is a solo or a multi-doctor practice, the Acuity Systems office computer has the features and functions you need for eye care, programmed in the way that's most convenient to use them.

With Acuity Systems behind it, you can be sure it'll work.

And that's a feeling of security you won't get elsewhere.

Because most other "computer companies" sell you the computer and the program, and then kiss you goodbye. They don't help you through the transition period, when the computer is new. While some of them do train your employees, they make no provision for training new employees later on. None of them follow up to make sure the system's working right. And few of them actually service both the hardware and software.

But at Acuity Systems, we give you all the services the other companies don't. Like complete planning for the transition to computerization by our Practice Management Analyst. Complete training, plus follow-up and new employee training by our Customer Service representatives. And 24-hour service of any problem by our own nationwide service network.

Why do we do all this for you? Because in the past 10 years, we've acquired an excellent reputation from our other products, the Auto-Refractor® and the Auto-Lensmeter™. And making sure our new office computer works perfectly in every practice is the only way we're going to keep that reputation.

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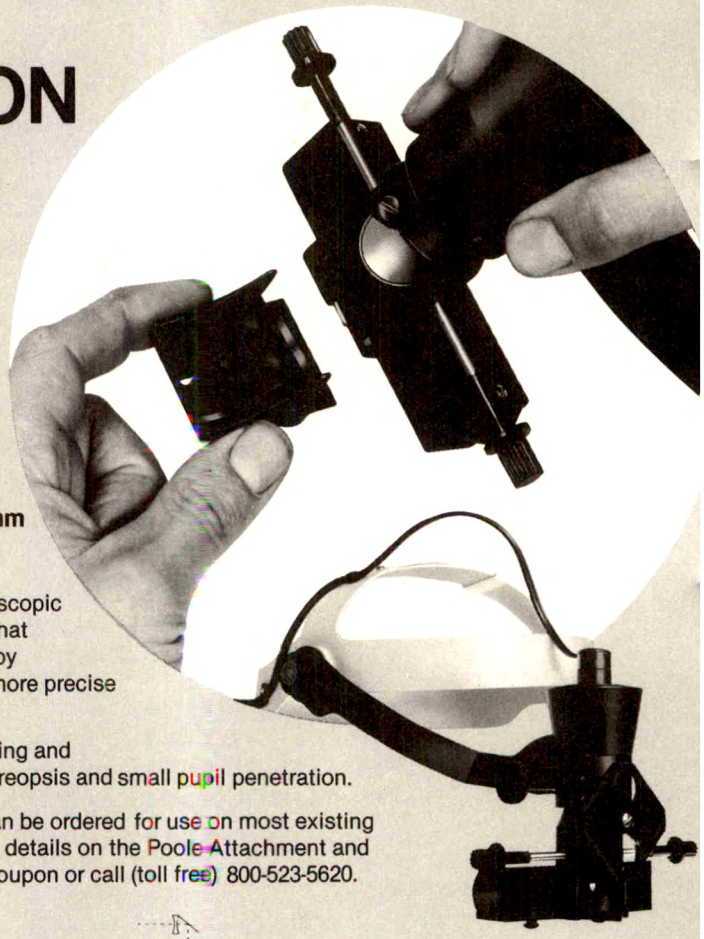
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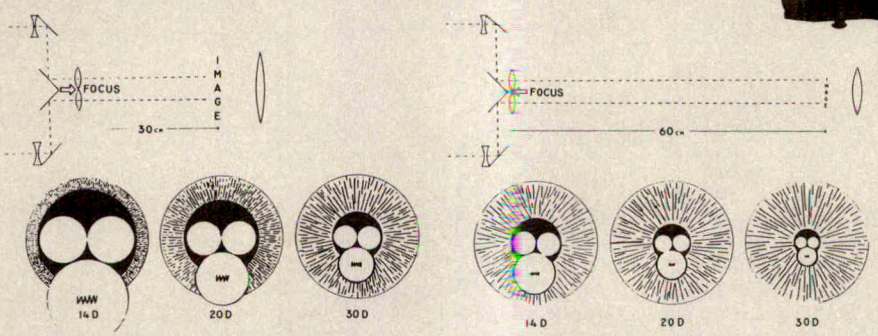


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(Poole, Poler, and Sudarsky). Schematic representation of the use of viewing lens distance and power variation to optimize stereopsis and magnification for a given pupillary aperture. Left, Variable focus telescopic ophthalmoscope focused on the viewing lens image at 30cm (pupillary diameters: 8mm, 6mm, and 4mm). Right, Variable focus telescopic ophthalmoscope focused on the viewing lens image at 60cm (pupillary diameters: 4mm, 3mm, and 2mm). Fundus Magnifications 1.5x with 30D at 60cm to 8.5x with 14D at 30cm.

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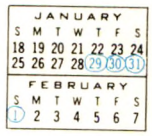
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MID WINTER NATIONAL MEETING

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JANUARY 29 - FEBRUARY 1, 1981

(ending 12 noon, Sunday)



CONTACT LENS ASSOCIATION OF OPHTHALMOLOGISTS, INC.

CONRAD BERENS LECTURER
John A. Dyer, M.D.
Corneal Contact Lens in Perspective—1948-1981

DISTINGUISHED VISITING LECTURER
Thomas F. Spring, M.D.
Contact Lenses, Endothelium and Keratoconus

PRACTICE SEMINAR
(9:00 a.m. - 12:00 noon Thursday)

Scientific Program begins at 2:00 p.m. Thursday

Thursday Afternoon (January 29)

Symposium: EXTENDED WEAR OF CONTACT LENSES IN APHAKIA ..... Jack Hartstein, M.D., *Moderator*
Patient Selection ..... Joseph A. Baldone, M.D.
Cooper Lenses ..... Richard G. Lembach, M.D.
Hydrocurve Lenses ..... Perry S. Binder, M.D.
Sauflon Lenses ..... Anthony B. Nesburn, M.D.
Silicone Lenses ..... Robert T. Blackhurst, M.D.
CSI Lenses ..... H. Dwight Cavanagh, M.D.
Complications of Extended Wear Lenses ..... G. Peter Halberg, M.D.
Panel Discussion

THE USE OF EXTENDED WEAR LENSES IN MYOPIA ..... Louis A. Wilson, M.D.
Symposium: THE USE OF EXTENDED WEAR LENSES FOR THERAPEUTIC PURPOSES ..... Richard H. Keates, M.D., *Moderator*
Cooper Lenses ..... Richard G. Lembach, M.D.
Bausch & Lomb Lenses ..... John Brodrick, M.D.
Sauflon Lenses ..... Bruce Bodner, M.D.
Softsight Therapeutic Lenses ..... Henry Gelender, M.D.
PRACTICAL WORKSHOPS ..... *Topics to be announced*

Friday Morning (January 30)

Symposium: TORIC SOFT LENSES ..... Barry A. Maltzman, M.D., *Moderator*
Lens Availability and Design ..... Fitting Techniques and Wear Expectations (Dura-Soft—Hydrocurve)
Fitting Techniques and Wearing Expectations (Hydromere—Miracon—Hydron)...Pitfalls to Avoid in Fitting Toric Lenses
Panel Discussion - Questions and Answers. ***Participants:*** Herschell H. Boyd, M.D., Paul R. Honan, Jr., M.D., Bernard J. Slatt, M.D., Harold A. Stein, M.D.
Symposium: HYGIENIC CARE OF SOFT LENSES ..... Ellis Gruber, M.D., *Moderator*
OPHTHALMIC DEVICES: A Review of Three Years of FDA Involvement ..... Max Talbott, Ph.D.
Symposium: CONTACT LENSES IN INFANTS AND CHILDREN ..... Harold P. Koller, M.D., *Moderator*
Indications and Available Materials for Contact Lenses in Infants and Children ..... Judith E. Gurland, M.D.
Pathophysiology of Contact Lens Therapy in Infants and Children ..... I. Matthew Rabinowicz, M.D.
Contact Lens Fitting Techniques Unique to the Pediatric Population ..... Harold P. Koller, M.D.
Indications and Complications of Intraocular Lenses vs. Contact Lenses in Infants and Children ..... Jerry W. Maida, M.D.
Results of Extended and Daily Wear Contact Lenses in Infants and Children ..... Harold P. Koller, M.D.
DISTINGUISHED VISITING LECTURER ..... **Thomas F. Spring, M.D.**

Spouse Luncheon at Don the Beachcomber Compliments of CLAO

Friday Afternoon (January 30)

CONTACT LENS COURSES - Section I ..... F. Linsy Farris, M.D., *Director, Course Program*
1. Current Concepts in the Correction of Myopia—Extended Wear Lens, Orthokeratology and Refractive Corneal Surgery ..... Perry S. Binder, M.D.
2. Management of Corneal & Contact Lens Problems ..... G. Peter Halberg, M.D., Robert A. D'Amico, M.D.
3. How to Select the Best Soft Lens for Your Patient in 1981 ..... Ellis Gruber, M.D.

CLAO MID-WINTER MEETING

4. Astigmatism Correction with Hard & Soft Contact Lenses ..... Paul R. Honan, M.D., Herschell H. Boyd, M.D.
5. Specialized Techniques of Lens Fitting ..... Joseph W. Soper
6. Hard Lens Modification—Basic Lens Evaluation and Modification Techniques ..... Paul R. Honan, Jr., M.D., Marvin A. Garland, M.D.
7. Fitting of CAB and Silicone Lenses for Daily and Extended Wear .. Jay I. Lippman, M.D., Daniel Sigband, M.D.
8. Contact Lens Fitting in Infants & Children ..... Harold P. Koller, M.D.
9. Introduction to Refractive Keratoplasty ..... Miles H. Friedlander, M.D., Herbert E. Kaufman, M.D.
10. Contact Lens—Lacrimal System Interaction ..... David Lamberts, M.D., Frank J. Holly, Ph.D.
11. Extended Wear of Contact Lenses ..... Rao N. Gullapalli, M.D.
12. Contact Lens Equipment and Solutions ..... Ronald H. Akashi, M.D.

INTRAOCULAR LENS COURSES—Section I ..... Herve M. Byron, M.D., *Director, Course Program*

13. Intracapsular Extractions with Implants—I ..... Stephen Obstbaum, M.D., Marvin Kwitko, M.D.
14. Extracapsular Extractions with Implants—I ..... William Harris, M.D., Robert F. Azar, M.D.
15. Techniques for the Advance Implant Surgeon—I ..... Richard P. Kratz, M.D., Charles Kelman, M.D.
16. Special Instrumentation & Therapeutic Agents ..... Buol K. Heslin, M.D., David J. McIntyre, M.D.

CONTACT LENS COURSES—Section II ..... R. Linsy Farris, M.D., *Director, Course Program*

1. Extended Contact Lens Wear ..... Anthony B. Nesburn, M.D.
2. Update on Therapeutic Contact Lenses ..... Edward L. Shaw, M.D.
3. Keratoconus ..... Frank B. Hoefle, M.D.
4. Soft Lens Fitting Technique ..... Joseph W. Soper
5. Bifocal & Multifocal Contact Lenses for the Presbyope and Aphake ..... Frank J. Weinstock, M.D., Abraham Schlossman, M.D.
6. The Beginning Training & Utilization of a Contact Lens Technician ..... Robert J. Crossen, M.D.
7. Starting from Scratch—Contact Lens Fitting ..... Paul R. Honan, M.D., Herschell H. Boyd, M.D.
8. Hard Lens Modification—Advanced Techniques ..... Marvin A. Garland, M.D., Paul R. Honan, M.D.
9. Soft Toric Lenses ..... Barry A. Maltzman, M.D.
10. Fitting Contact Lenses by Piggy Back Technique ..... Joseph A. Baldone, M.D.
11. Contact Lens Fitting in Difficult Cases ..... Donald J. Doughman, M.D.
12. Ocular Allergy Problems in Contact Lens Wearers ..... Mark B. Abelson, M.D.

INTRAOCULAR LENS COURSES—Section II ..... Herve M. Byron, M.D., *Director, Course Program*

13. Intracapsular Extraction with Implants—II ..... Stephen Obstbaum, M.D., Marvin Kwitko, M.D.
14. Extracapsular Extraction with Implants—II ..... William Harris, M.D., Robert F. Azar, M.D.
15. Techniques for the Advanced Implant Surgeon—II ..... Richard Kratz, M.D., Charles Kelman, M.D.
16. Keratorefractive Surgery ..... James V. Aquavella, M.D., Dennis D. Shepherd, M.D.

Wine and Cheese Tasting Party Compliments of Barnes-Hind Pharmaceuticals, Inc.

Saturday Morning (January 31)

Symposium: INTERACTION OF THE CORNEAL EPITHELIUM, TEAR FILM, AND THE CONTACT LENS ..... James V. Aquavella, M.D., *Moderator*

Stimulation and Tear Flow ..... Jules L. Baum, M.D.
The Contact Lens Surface ..... Rao N. Gullapalli, M.D.
Immunological Aspects ..... Mark B. Abelson, M.D.
Tear Film ph ..... Michael A. Lemp, M.D.
Tear Film Tonicity ..... R. Linsy Farris, M.D.
Summary and Discussion ..... James V. Aquavella, M.D.

Symposium: PROTECTION OF ENDOTHELIUM DURING IOL SURGERY .. Herbert E. Kaufman, M.D., *Moderator*

OVERVIEW—Problem of Cell Loss During Surgery ..... Herbert E. Kaufman, M.D.
Quantitation of Cell Loss and Its Prevention by Altering Surgical Technique ..... William M. Bourne, M.D.
The Use of Viscose Agents to Minimize Surgical Cell Loss ..... David Miller, M.D.
Lens Coatings and Other Non-Viscose Type Approaches to the Prevention of Cell Loss . Herbert E. Kaufman, M.D.

ISSUES FACING OPHTHALMOLOGY ..... George Edward Garcia, M.D., *President American Association of Ophthalmology*

CONRAD BERENS LECTURER ..... **John A. Dyer, M.D.**
OPHTHALMIC TECHNICIANS COURSE ..... Topics to be announced

Saturday Afternoon (January 31)

THEODORE E. OBRIG MEMORIAL LECTURER ..... To Be Announced

Symposium: WHAT'S NEW AND INTERESTING IN LENS IMPLANTATION ..... Harold A. Stein, M.D., *Moderator*

Patient Selection—Extended Wear of IOL? ..... Harold A. Stein, M.D.
Instrumentation in Surgery ..... Aziz J. Anis, M.D., F.A.C.S.
Newer Intraocular Lens Designs ..... Ronald W. Barnet, M.D.
Sutures for Cataracts, Implant Surgery and Astigmatism Control ..... J. Elliott Blaydes, Jr., M.D.
Statistics on Implant Results ..... James V. Aquavella, M.D.
Some Mechanical Considerations ..... C. William Simcoe, M.D.
Preventing Complications with IOL's ..... Stanley C. Becker, M.D.
Managing Complications ..... Richard P. Kratz, M.D.

Symposium: IS EXTRACAPSULAR CATARACT SURGERY WORTH IT? .... C. William Simcoe, M.D., *Moderator*

Three panelists (Richard P. Kratz, M.D., Richard H. Keates, M.D., and K. Buol Heslin, M.D.) will each give a 10 minute

presentation of their personal experiences regarding: Prognosis for a Successful Repair of Retinal Detachment; Cystoid Macular Edema; Quality of Vision. A Panel Discussion and Questions from the Audience will follow.
MOVIES ..... Robert F. Azar, M.D., *Moderator*

International Party Sponsored by Alcon/BP

Sunday Morning (February 1)

SURGICAL CORRECTION OF REFRACTIVE ERRORS
Jack Hartstein, M.D., *Chairman*

Symposium: SURGICAL CORRECTION OF ASTIGMATISM ..... Richard P. Kratz, M.D., *Moderator*
Prevention: Cataract Wound Closure ..... Clifford Terry, M.D.
Comparison of Keratometers ..... Dennis Shepherd, M.D.
Correction: Reconstruction of Previous Incision ..... Thomas Cravy, M.D.
Corneal Tuck ..... Robert F. Azar, M.D.
Symposium: SURGICAL CORRECTION OF MYOPIA—PRO's AND CON's ..... Walter J. Stark, M.D., *Moderator*
History of Surgical Correction of Myopia and Early Clinical Results ..... Leeds Katzen, M.D.
Surgical Correction of Myopia—Studies in Laboratory Animals and Human Cadaver Eyes ..... Ronald Smith, M.D.
Complications of Radial Keratotomy ..... J. James Rowsey, M.D.
Update of Clinical Experience with Surgical Correction of Myopia ..... Leo D. Bores, M.D.
A Prospective, Multicenter, Clinical Trial of Radial Keratotomy for Myopia ..... George Waring, M.D.
Summary and Conclusions ..... Walter J. Stark, M.D.
Symposium: SURGICAL CORRECTION OF APHAKIA ..... Perry S. Binder, M.D., *Moderator*
Keratophakia and Hypermytropic Keratomileusis ..... Cass Swinger, M.D.
Hypermytropic Keratomileusis ..... Jorge Krumeich, M.D.
Keratophakia & Keratomileusis Hyperopia Using Precarved Frozen Material ..... Miles H. Friedlander, M.D.
Epikeratophakia ..... Herbert E. Kaufman, M.D.
Panel Discussion—Summary and Conclusions

Contact & Intraocular Lens Presentations—Thursday afternoon, Friday morning and Saturday morning

---

An optional Practice Management Seminar will be conducted on Thursday, January 29, 1981 immediately preceding the scientific session. There is no registration fee for the Practice Management Seminar; the registration fee for the Scientific Session is the total fee for all sessions.

Registration for the Scientific Session will take place on Wednesday, January 28 from 12:00 noon to 5:00 p.m. and Thursday, January 29 from 7:30 a.m. to 5:30 p.m. Use the registration form below to pre-register and save time at the meeting.

Refund Policy. Full refund if cancellation is received by January 1, 1981. A $25.00 service charge per person for cancellations from January 2, 1981 to January 28, 1981. No refunds for cancellations received after January 29, 1981.

A special room rate has been arranged with the Sahara Hotel. For room reservations only, write to the Sahara Hotel, Las Vegas, Nevada 89114.

PRE-REGISTRATION FORM—CLAO MID-WINTER NATIONAL MEETING
January 29 - February 1, 1981 Sahara Hotel Las Vegas, Nevada

Name _____ M.D. _____ Other _____

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☐ Physician Member of CLAO—US$100.00 ☐ Physician Non-Member—US$250.00

☐ Resident Physician in Ophthalmology—US$50.00

☐ Technician* employed by Ophthalmologist (Physician Member of CLAO). Must be identified by letter by the employing ophthalmologist—US$75.00

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Please make check payable to: CLAO—Mid Winter Meeting and forward to R. Linsy Farris, M.D.
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New Orleans, LA 70115

*Technicians. This fee includes the entire meeting (3-1/2 days), in addition to the Ophthalmic Assistants and Technicians Course.

In chronic open-angle glaucoma

NEW 40-MONTH STUDY REPORTS LONG-TERM EFFICACY AND TOLERABILITY

Timoptic

(TIMOLOL MALEATE | MSD)
OPHTHALMIC SOLUTION

In an open, long-term maintenance study* of the effect of continuous therapy (up to 40 months) of chronic open-angle glaucoma with TIMOPTIC, 96% of the patients exhibited IOP values of ≤ 21 mm Hg at each measurement during treatment with TIMOPTIC...and the drug was generally well tolerated. (See graphs and data on facing page.)

A better quality of life for many patients

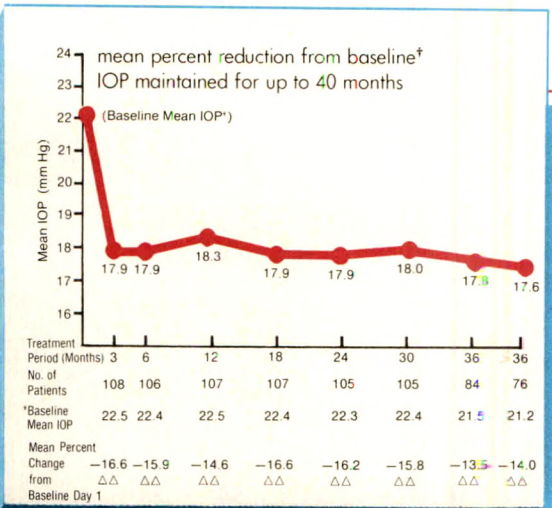
Patients on TIMOPTIC generally do not experience the following therapy-limiting side effects: night blindness/blurred vision/dimming of vision.**

Patients on TIMOPTIC have significantly fewer and less severe side effects than those on pilocarpine or epinephrine, i.e., a much lower incidence of ocular discomfort—such as less burning, smarting, itching, browache, and headache.**

TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product. As with other topically applied ophthalmic drugs, this drug may be absorbed systemically. As with the use of other antiglaucoma drugs, diminished responsiveness to TIMOPTIC after prolonged therapy has been reported in some patients.

*Data on file, Merck Sharp & Dohme Research Laboratories. Additional information available on request.

**Based on controlled multiclinic studies. Data on file, Merck Sharp & Dohme Research Laboratories. Additional information available on request.

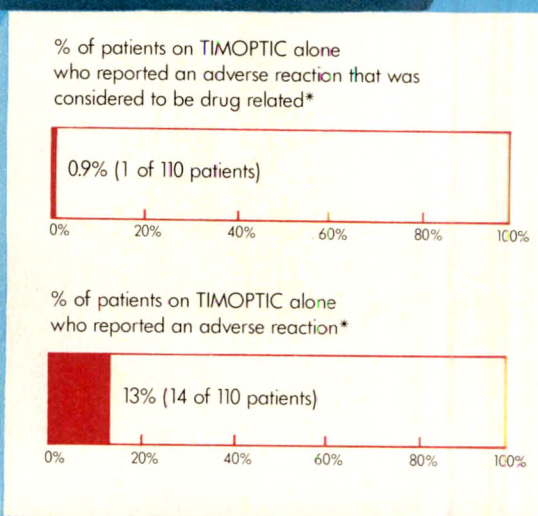


long-term efficacy of Timoptic® (Timolol Maleate) MSD)

There was virtually no loss of therapeutic effect in patients treated for up to 40 months with TIMOPTIC alone—i.e., the IOP of almost all patients was lowered to 21 mm Hg or below throughout the study.*

†Of the 110 patients on TIMOPTIC alone, 91 were on previous antiglaucoma treatment and had a treated baseline mean IOP of 21.4 mm Hg. The 19 patients with no previous antiglaucoma treatment had an untreated baseline mean IOP of 27.5 mm Hg. Thus, the mean IOP at baseline for the combined group is 22.5 mm Hg.

ΔΔSignificant percent change from baseline, $p < 0.01$.



long-term tolerability of Timoptic® (Timolol Maleate) MSD)

During the 40-month study,* 14 of 110 patients (13%) on TIMOPTIC alone reported an adverse reaction, but only one of these, an allergic reaction, was considered to be drug related.

Visual disturbances including refractive changes (due to withdrawal of miotic therapy in some cases) have been infrequently associated with therapy with TIMOPTIC. Rarely, aphakic cystoid macular edema has been reported, but a causal relationship to therapy with TIMOPTIC has not been established.

*Data on file, Merck Sharp & Dohme Research Laboratories. Additional information available on request.

For a brief summary of prescribing information, please see following page.

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b.i.d.

Timoptic®

(TIMOLOL MALEATE | MSD)
OPHTHALMIC SOLUTION

a beta-adrenergic blocking agent in ophthalmic solution

a significant advance for the reduction of IOP in:

- chronic open-angle glaucoma
- aphakic glaucoma
- some patients with secondary glaucoma
- patients with elevated intraocular pressure at sufficient risk to require lowering of the ocular pressure
- some patients who respond inadequately to multiple antiglaucoma drug therapy

How to start patients on TIMOPTIC:
Usual starting dosage: one drop 0.25% TIMOPTIC in each eye twice a day.

How to transfer from current therapy to TIMOPTIC:
1. On the first day: continue with the agent(s) already being used and add one drop 0.25% TIMOPTIC in each eye b.i.d.

2. On the second day: discontinue the previously used agent(s) and continue with TIMOPTIC in each eye b.i.d.

If clinical response is not adequate:
Dosage may be increased (from the 0.25% solution) by changing to one drop 0.5% TIMOPTIC twice a day in each eye. Dosages above one drop of 0.5% TIMOPTIC twice a day generally have not been shown to produce further reduction of IOP. If the intraocular pressure is maintained at satisfactory levels, the dosage schedule may be changed to one drop once a day in each eye.

Because of diurnal variations in intraocular pressure, satisfactory response to the once-a-day dose is best determined by measuring the intraocular pressure at different times during the day.

In patients with a history of severe cardiac disease, pulse rates should be checked.

CONTRAINDICATIONS: TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product.
WARNINGS: As with other topically applied ophthalmic drugs, this drug may be absorbed systemically.
PRECAUTIONS: Should be used with caution in patients with known contraindications to systemic use of beta-adrenergic receptor blocking agents. These include bronchospastic disease; sinus bradycardia and greater than first-degree block; cardiogenic shock; cardiac failure; concomitant use with adrenergic-augmenting psychotropic drugs. In patients with a history of severe cardiac disease, pulse rates should be checked. Patients who are already receiving a beta-adrenergic blocking agent orally and who are given TIMOPTIC should be observed for a potential additive effect either on the intraocular pressure or on the known systemic effects of beta blockade.

Sufficient clinical data have not been obtained to show safety or efficacy in narrow-angle or angle-closure glaucoma.

As with the use of other antiglaucoma drugs, diminished responsiveness to TIMOPTIC after prolonged therapy has been reported in some patients. However, in one long-term study in which 56 patients have been followed for at least 3 years, no significant difference in mean intraocular pressure has been observed after initial stabilization.
Pregnancy: TIMOPTIC has not been studied in human pregnancy. The use of TIMOPTIC in pregnant women requires that the anticipated benefit be weighed against possible hazards.
Use in Children: Since clinical studies in children have not been done, TIMOPTIC is not recommended for use in children at this time.
Animal Studies: No adverse ocular effects were observed in rabbits and dogs administered TIMOPTIC topically in studies lasting one and two years respectively.

Lifetime studies with timolol have been completed in rats at oral doses of 25, 100, and 300 mg/kg/day and in mice at oral doses of 5, 50, and 500 mg/kg/day. In male and female rats and male mice at all dose levels and in female mice at dose levels of 5 and 50 mg/kg/day, timolol demonstrated no carcinogenic effect. There was a slight increase in the incidence of mammary adenocarcinomas in female mice that received 500 mg/kg/day (about 500 times the maximum recommended human oral dose on a mg/kg basis). Timolol caused dose-related elevations of serum prolactin in female mice at doses of 100 mg/kg or more, but only very slight transient elevations were found in male mice at doses of 500 mg/kg. Since numerous studies have demonstrated that drugs which cause elevations of serum prolactin are associated with mammary tumors in rodents, the mammary tumors in the female mice in the highest dosage group of this study were considered to have resulted from an increased serum prolactin. In humans, no such association between serum prolactin and mammary carcinoma has been established. Furthermore, in adult human female subjects who received oral dosages of up to 60 mg of timolol, the maximum recommended human oral dosage, there were no clinically meaningful changes in serum prolactin.
ADVERSE REACTIONS: TIMOPTIC Ophthalmic Solution is usually well tolerated. The following adverse reactions have been reported either in clinical trials of up to 3 years duration prior to release in 1978 or since the drug has been marketed:

Signs and symptoms of ocular irritation, including conjunctivitis, blepharitis, and keratitis, have been reported occasionally.

Hypersensitivity reactions, including localized and generalized rash, and urticaria have been reported rarely.

Visual disturbances including refractive changes (due to withdrawal of miotic therapy in some cases) have been infrequently associated with therapy with TIMOPTIC. Rarely, aphakic cystoid macular edema has been reported, but a causal relationship to therapy with TIMOPTIC has not been established.

Aggravation or precipitation of certain cardiovascular and pulmonary disorders has been reported, presumably related to effects of systemic beta blockade (see PRECAUTIONS). These include bradyarrhythmia, hypotension, syncope, and bronchospasm (predominantly in patients with pre-existing bronchospastic disease). In clinical trials, slight reduction of the resting heart rate in some patients (mean reduction 2.9 beats/minute, standard deviation 10.2) has been observed.

The following adverse effects have been reported rarely, and a causal relationship to therapy with TIMOPTIC has not been established: headache, anorexia, dyspepsia, nausea, dizziness, CNS effects (fatigue, confusion, depression, somnolence, and anxiety), palpitation, and hypertension.
HOW SUPPLIED: TIMOPTIC Ophthalmic Solution, 0.25% and TIMOPTIC Ophthalmic Solution, 0.5%. Both are available in 5-ml and 10-ml plastic OCUMETER® Ophthalmic Dispensers with a controlled drop tip.
980(DC7)(6011)

MSD
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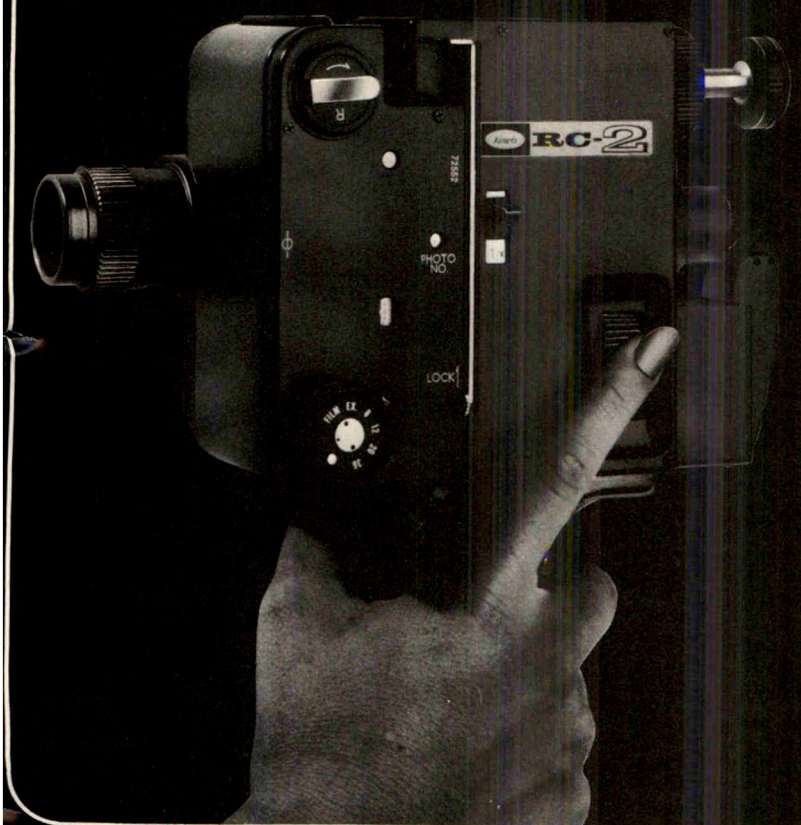
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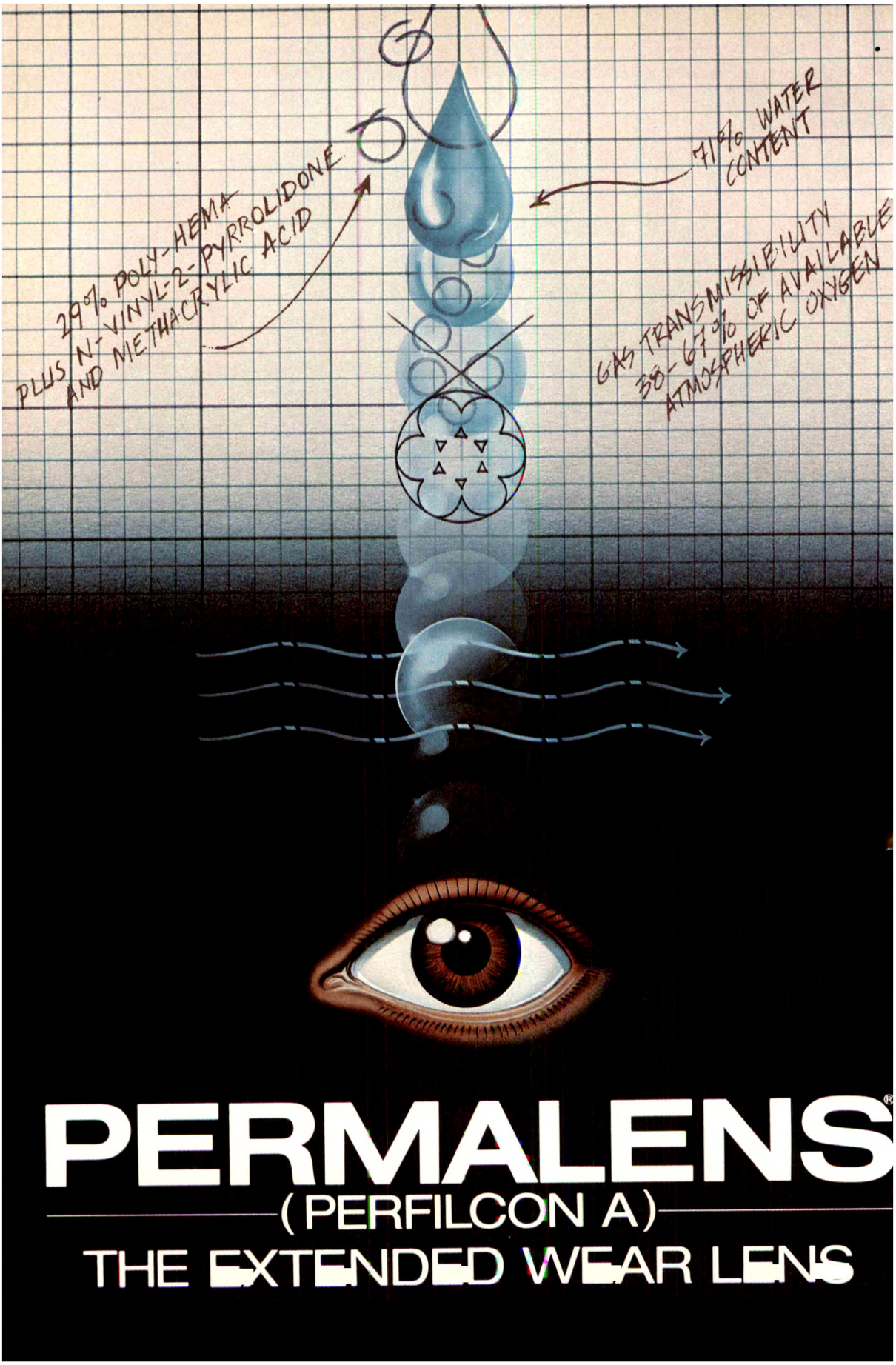
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PERMALENS®

(PERFILCON A)

THE EXTENDED WEAR LENS

Permalens® Contact Lenses: not just extended wear of a lens, but a lens **designed specifically for extended wear.**

That's why Permalens® Contact Lenses are rapidly changing the field of vision correction. Now your aphakic patients can again experience near-normal vision from the moment they wake up in the morning. Without the bother of daily insertion and removal.

And because of their advanced design, Permalens® Contact Lenses exhibit a physiological response comparable to daily-wear soft lenses. Even though your patients wear them 24 hours a day, for weeks at a time. The long-term acceptance of Permalens® Contact Lenses has been proven through extensive clinical trials.

This response is partly due to the Permalens® material, perfilcon A, which combines high water content, high oxygen permeability, and dimensional stability. These properties were designed into the lens to facilitate oxygen transfer directly through the lens, so the cornea can receive an oxygen supply adequate to maintain itself in a healthy state.

These properties also make Permalens® Contact Lenses more supple, and therefore comfortable on the patient's eyes. Patients have reported, in fact, they're the next best thing to wearing no lens at all.

Easy to fit, Permalens® Contact Lenses are available in a range of powers from +11.00 to +17.00 diopters. And with just four parameters, the lens fits most corneal sizes and curvatures.

Maintenance, too, is uncomplicated. Since Permalens® Contact Lenses usually need only be removed once a month, cleaning and disinfection are simplified. The preferred regimen is Preflex®/Flexsol®/Normol.® (Thermal regimens can also be used.) For re-hydration during lens wear, Clērz® or Adapettes® eye drops are available.

Patient comfort and convenience . . . clinically proven safety . . . easy fitting . . . simple maintenance. Good reasons why thousands of patients and practitioners alike have accepted Permalens® as **the** extended-wear lens.

Permalens®
(perfilcon A)

Hydrophilic Contact Lens
For Extended Wear
Post-Cataract (Aphakic) Use

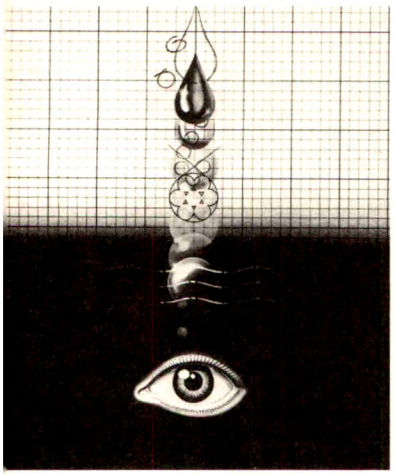
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CooperVision® Inc.
OPTICS DIVISION

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Permalens®
(perfilcon A)

Hydrophilic Contact Lens for Extended Wear Post-Cataract (Aphakic) Use

DESCRIPTION

The **Permalens**® (perfilcon A) Hydrophilic Contact Lens is a hemispherical shell which covers the cornea and may cover a portion of the adjacent sclera. The lens material, perfilcon A, is a terpolymer of 2-hydroxyethyl methacrylate, N-vinyl-2-pyrrolidone, and methacrylic acid, with ethyleneglycol dimethacrylate as a crosslinking agent. The hydrated lens consists of 29% perfilcon A and 71% water by weight. The gas permeability of **Permalens**® Hydrophilic Contact Lenses, using the Fatt method,[1] has been determined to be: 42.0 x 10^{-11} $cm^2 \cdot ml\ O_2/sec \cdot ml \cdot mm\ Hg$ at 36°C.

ACTIONS

When placed on the human cornea, the hydrated **Permalens**® Hydrophilic Contact Lens acts as a refracting medium to compensate spherical ametropias.

INDICATIONS AND USAGE

Aphakia

Permalens® (perfilcon A) Hydrophilic Contact Lenses are indicated for extended-wear aphakic vision correction in persons with non-diseased eyes.

Note: Supplemental spectacles may be required, in some conditions, to provide near vision or to compensate for uncorrected refractive astigmatism.

CONTRAINDICATIONS

Permalens® Hydrophilic Contact Lenses are contraindicated by the presence of any of the following conditions:

1. Acute and subacute inflammations of the anterior segment of the eye.
2. Any eye disease which affects the cornea or conjunctiva.
3. Insufficiency of lacrimal secretion.
4. Corneal hypoesthesia.
5. Any systemic disease which may affect the eye or be exaggerated by wearing contact lenses.

WARNINGS

Abrasions and Infections

As a lens becomes less comfortable than it was when it was first placed on the wearer's cornea, the lens should be removed and inspected for the presence of a foreign body, lens coating, deposits, or lens damage. If examination of the patient's eye reveals any eye abrasion, ulceration, irritation or infection, or any other abnormal eye condition is observed concurrently with lens wear, the lens should be removed immediately and a physician consulted.

Patients should not be fitted with Permalens® (perfilcon A) Hydrophilic Contact Lenses during the post-operative period until the opinion of the surgeon, the eye has healed completely.

Medications and Eye Drops

Permalens® Hydrophilic Contact Lenses must be stored in an appropriate solution when off the eyes. The solution used is dependent on the system selected for disinfection. When the lenses are disinfected with the thermal disinfection system, they may be stored in PERMASOL™ Rinsing and Storage Solution (sterile buffered balanced salt solution preserved with edetate sodium 0.1% and thimerosal 0.001%) or BOILnSOAK® Sterile Saline Solution (sterile buffered isotonic solution containing boric acid, sodium borate, sodium chloride 0.7%, preserved with Thimerosal (Lilly) 0.001% and edetate disodium 0.1%). When lenses are disinfected with the chemical disinfection system, they may be stored in FLEXSOL® Sterile Disinfection and Storage Solution (sterile buffered isotonic solution of sodium chloride, sodium borate, boric acid, polyvinylpyrrolidone, polyoxyethylene and polyoxypropylene, preserved with Thimerosal (Lilly) 0.001%, chlorhexidine 0.005% and edetate disodium 0.1%).

Only the above solutions and the following may be used with **Permalens**® Hydrophilic Contact Lenses:

1. PREFLEX® Sterile Cleaning Solution (sterile solution of a nonionic cleaner preserved with Thimerosal (Lilly) 0.004% and edetate disodium 0.2%).
2. NORMOL® Sterile Rinsing Solution (sterile buffered isotonic aqueous solution of sodium chloride, sodium borate and boric acid, preserved with Thimerosal (Lilly) 0.001%, edetate disodium 0.1% and chlorhexidine 0.005%).
3. CLERZ® Lubricating and Rewetting Eye Drops (sterile solution preserved with edetate disodium 0.1% and Thimerosal (Lilly) 0.001%).
4. ADAPETTES® Sterile Lubricating Solution (buffered isotonic aqueous solution containing Adsorbobase® (polyvinylpyrrolidone with other water-soluble polymers), Thimerosal (Lilly) 0.004%, and edetate disodium 0.1%).

No conventional (hard) contact lens solution should be used. Only those solutions specifically recommended by your eye-care practitioner should be used during wear.

WEARING RESTRICTIONS

Permalens® (perfilcon A) Hydrophilic Contact Lenses should not be worn while swimming or in the presence of noxious or irritating vapors.

Lens-Care Regimen

Patients must adhere to the recommended lens-care regimen of **Permalens**® Hydrophilic Contact Lenses. Failure to follow this procedure may result in the development of serious ocular infections which might result in corneal ulcers.

PRECAUTIONS

Storage

Permalens® Hydrophilic Contact Lenses must be stored in the appropriate storage solution: PERMASOL™ Rinsing and Storage Solution, BOILnSOAK® Sterile Saline Solution, or FLEXSOL® Sterile Disinfection and Storage Solution, depending on disinfecting methods used. If left exposed to air, the lenses will dehydrate, become brittle and break readily. If a lens dehydrates, it should be soaked in one of the following until it returns to a hydrated (soft) pliable state:

1. PERMASOL™ Rinsing and Storage Solution
2. BOILnSOAK® Sterile Saline Solution
3. FLEXSOL® Sterile Disinfection and Storage Solution

PERMA-case™ Contact Lens Storage and Carrying Case should be used for storing lenses.

Hygiene

Hands must be washed and rinsed thoroughly, and dried before handling the lenses. Cosmetics, lotions, soaps and creams must not come in contact with the lenses since eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until the spray has settled.

Cleaning and Disinfecting

Prior to wearing, **Permalens**® (perfilcon A) Hydrophilic Contact Lenses must be both cleaned and disinfected.

Cleaning: Monthly removal for cleaning is recommended. However, at the discretion of the eye-care practitioner, the lenses may be removed at a frequency adjusted to the needs of each patient. Each time the lenses are removed from the wearer's eyes, both surfaces of the lenses must be cleaned using several drops of PREFLEX® Sterile Cleaning Solution. Between regular cleaning periods, CLERZ® Lubricating and Rewetting Eye Drops or ADAPETTES® Sterile Lubricating Solution may be used to maintain lens hydration while being worn.

Routine Thermal Disinfection Method: Permalens® Hydrophilic Contact Lenses may be effectively disinfected using a low-heat disinfecting unit approved for use with hydrophilic contact lenses. After cleaning with PREFLEX® Sterile Cleaning Solution, rinse lenses with PERMASOL™ Rinsing and Storage Solution or BOILnSOAK® Sterile Saline Solution, and disinfect in a RINCON® low-heat Thermal Disinfection Unit or other approved unit. The lens storage containers must be emptied and filled with fresh PERMASOL™ Rinsing and Storage Solution or BOILnSOAK® Sterile Saline Solution just prior to disinfecting the lenses.

Alternate Thermal Disinfection Method: If a thermal unit is not available, the following disinfecting system can be used temporarily. After cleaning the lenses according to instructions, fill each chamber of the PERMA-case™ with PERMASOL™ or BOILnSOAK® Bring a pan of water to a rolling boil. Remove the pan from heat. Place the sealed carrying case into hot water and allow to cool. When the water is cool, lenses have been disinfected.

NOTE: USE OF THE RINCON® THERMAL DISINFECTION UNIT SHOULD BE RESUMED AS SOON AS POSSIBLE.

Chemical Disinfection Method: Disinfection with PREFLEX® Sterile Cleaning Solution, FLEXSOL® Sterile Disinfection and Storage Solution and NORMOL® Sterile Rinsing Solution has been shown to be an effective disinfection system. Following each removal, **Permalens**® Hydrophilic Contact Lenses must be cleaned with PREFLEX® Sterile Cleaning Solution and rinsed with NORMOL® Sterile Rinsing Solution. Each time the lens is stored, the PERMA-case™ Contact Lens Storage and Carrying Case must be emptied and refilled with fresh FLEXSOL® Sterile Disinfection and Storage Solution. Fresh FLEXSOL® Sterile Disinfection and Storage Solution must also be used when disinfecting a lens.

WARNING: FLEXSOL® STERILE DISINFECTION AND STORAGE SOLUTION SHOULD *NEVER* BE USED WITH HEA

LENS EJECTION

Occasionally, a lens may become partially dehydrated during sleep. This may result in the lens being dislodged from the eye by lid action or rubbing the eyelids. Dislodged or routinely removed lenses should be thoroughly rehydrated in one of the following solutions:

1. PERMASOL™ Rinsing and Storage Solution
2. NORMOL® Sterile Rinsing Solution
3. BOILnSOAK® Sterile Saline Solution

After the lens returns to a soft, supple state it should be cleaned and disinfected prior to replacement on the eye.

ADVERSE REACTIONS

Serious corneal damage may result from wearing lenses which have been soaked in conventional hard contact lens solutions. Eye irritation may occur within a short time after putting on a lens stored in a solution of improper pH or tonicity. Removal of the lens will relieve the irritation.

Excessive tearing or redness, unusual eye secretions, reduced visual acuity, and photophobia are not normal; if these symptoms occur, the patient should be examined by an eye-care practitioner to determine the cause.

DOSAGE AND ADMINISTRATION

Fitting

Conventional methods of fitting contact lenses do not apply to **Permalens**® (perfilcon A) Hydrophilic Contact Lenses. For a detailed description of the fitting technique, refer to the **Permalens**® Hydrophilic Contact Lens Professional Fitting Guide, copies of which are available from Cooper Laboratories, Inc., 265 N. Whisman Road, Mountain View, California 94043.

WEARING SCHEDULE

Permalens® Hydrophilic Contact Lenses are indicated for extended-wear (24-hour) use, without a gradual daily increase in wearing time required. Cleaning is recommended at monthly intervals. However, at the discretion of the eye-care practitioner, the lenses may be removed at a frequency adjusted to the needs of each patient.

Lens Care and Handling

Care must be taken on the initial visit to ensure that the patient is supplied with all necessary accessory products and fully understands all care and handling instructions for the lenses. Regular post-fitting visits are necessary to assure patient health and compliance with instructions.

HOW SUPPLIED

Each lens is supplied sterile in a sealed glass vial containing a buffered isotonic salt solution. The vial is marked with the dioptric power, base curve, diameter, lot number, and expiration date of the lens.

Accessories

Patient Care Kit	Cat. #80230
Wearer's Manual	Cat. #80244
PERMA-case™ Contact Lens Storage and Carrying Case	Cat. #80231
FLEXSOL® Sterile Disinfection and Storage Solution*	Cat. #90110
NORMOL® Sterile Rinsing Solution*	Cat. #90127
PREFLEX® Sterile Cleaning Solution*	Cat. #90131
CLERZ® Lubricating and Rewetting Eye Drops	Cat. #80104
PERMASOL™ Rinsing and Storage Solution	Cat. #80131
RINCON® Thermal Disinfection Unit**	Cat. #90238
BOILnSOAK® Sterile Saline Solution*	Cat. #90103
ADAPETTES® Sterile Lubricating Solution	Cat. #90100

CAUTION: Federal Law Prohibits Dispensing Without Prescription

[1]Fatt I, Morris JA: A survey of gas-permeable contact lenses. *The Optician*: 27-36 (November 4, 1977).

*A product of Burton, Parsons & Company, Inc.
**A product of Rincon Industries, Inc.

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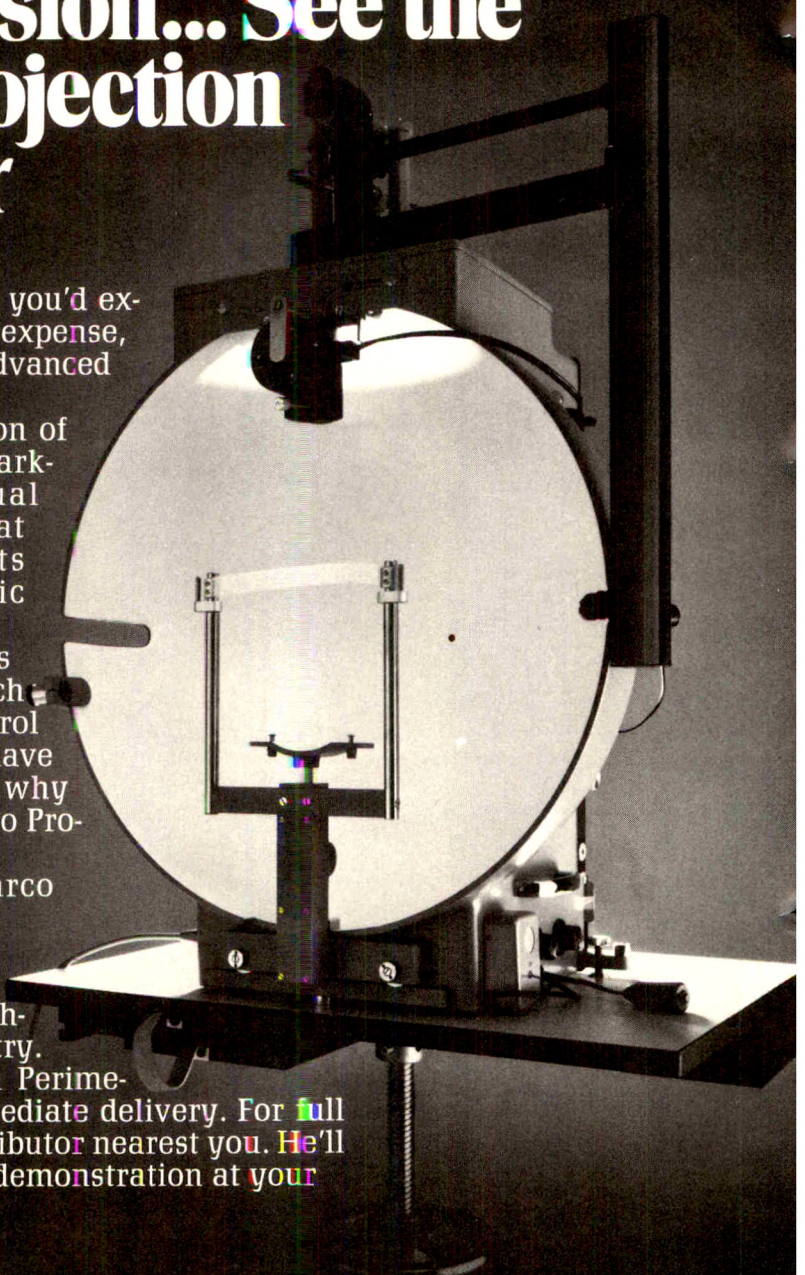
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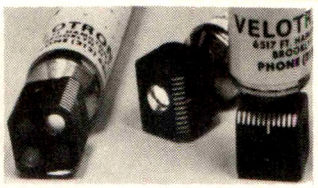
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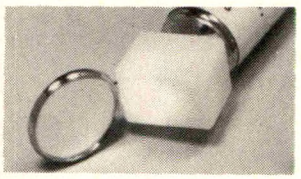
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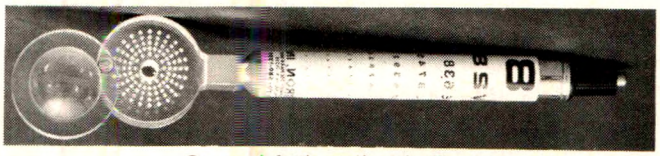
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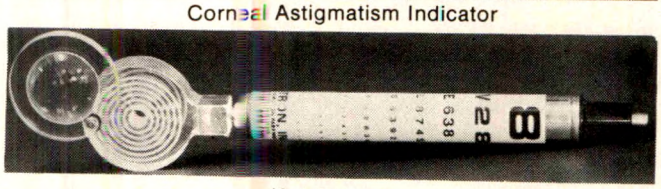
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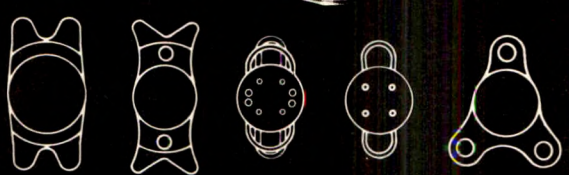


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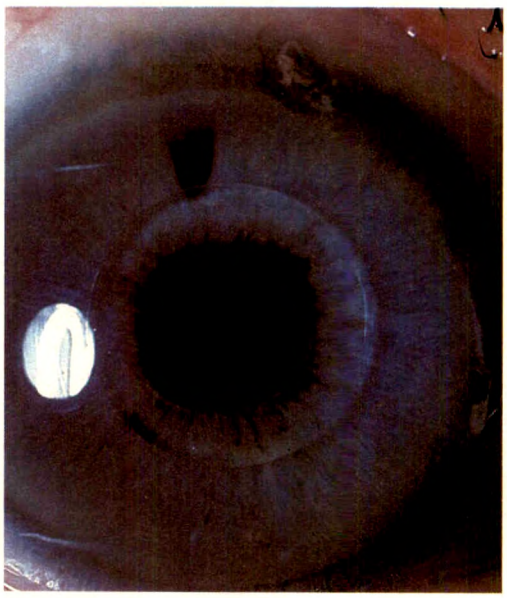
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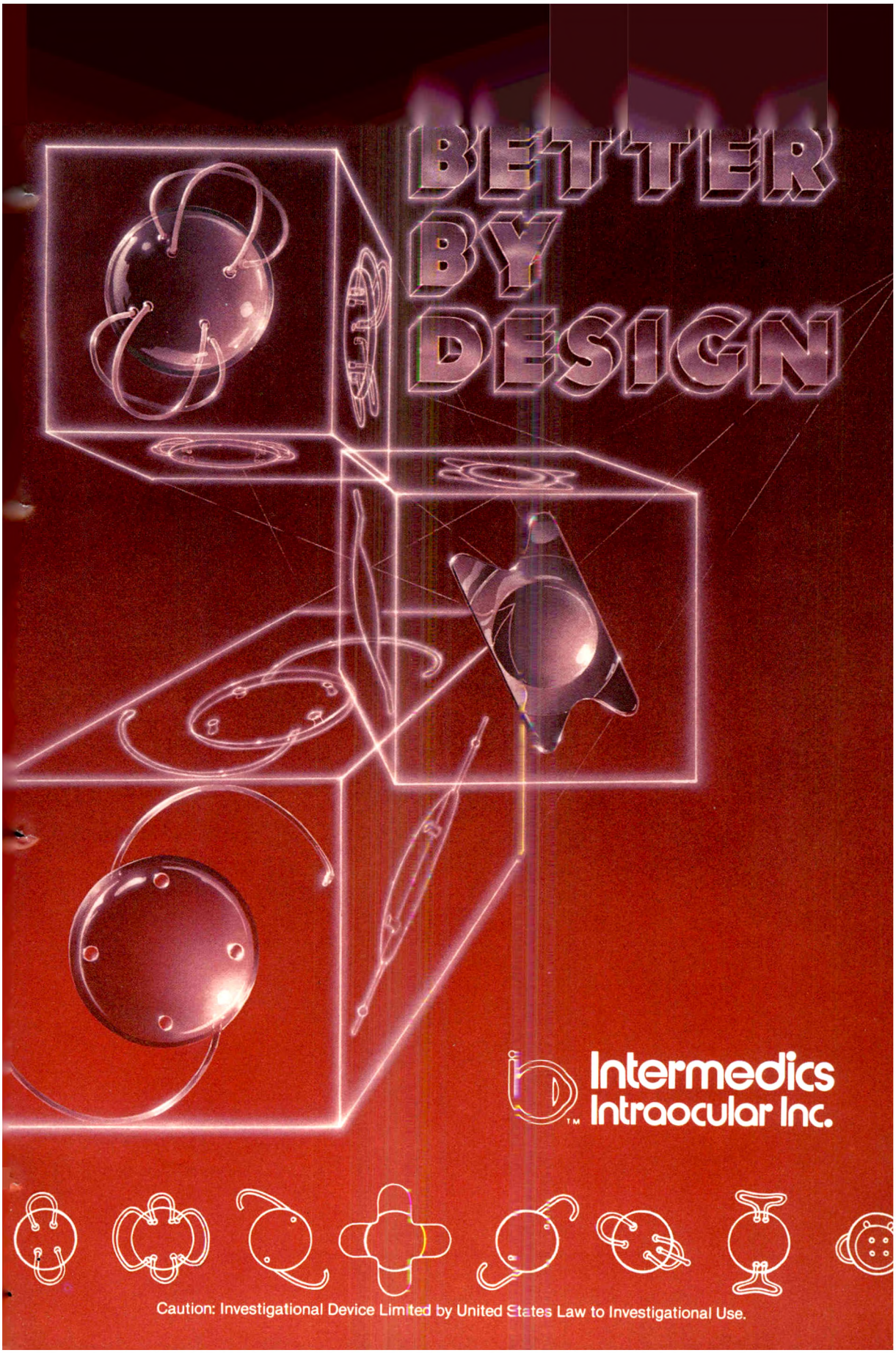
References:

1. Nelson, EL. Ophthalmic Steroids in Ocular Anti-Inflammatory Therapy. HE Kaufman, Editor, Springfield, IL. Thomas, 1970.
2. Hull, FW and Epstein, WA. Comparison of the intraocular pressure effects of topical dexamethasone and fluorometholone. Allergan Report Series No. 74, 1972.

INDICATIONS—For steroid responsive inflammation of the palpebral and bulbar conjunctiva, cornea and anterior segment of the globe. CONTRAINDICATIONS—Acute superficial herpes simplex keratitis. Fungal diseases of ocular structures. Vaccinia, varicella and most other viral diseases of the cornea and conjunctiva. Tuberculosis of the eye. Hypersensitivity to the constituents of this medication. WARNINGS—Steroid medication in the treatment of herpes simplex keratitis (involving the stroma) requires great caution; frequent slit-lamp microscopy is mandatory. Prolonged use may result in glaucoma, damage to the optic nerve, defects in visual acuity and fields of vision, posterior subcapsular cataract formation, or may aid in the establishment of secondary ocular infections from fungi or viruses liberated from ocular tissue. In those diseases causing thinning of the cornea or sclera, perforation has been known to occur with use of topical steroids. Acute purulent untreated infection of the eye may be masked or activity enhanced by presence of steroid medication. Safety and effectiveness have not been demonstrated in children of the age group 2 years or below. *Use in Pregnancy:* Safety of the use of topical steroids during pregnancy has not been established. PRECAUTIONS—As fungal infections of the cornea are particularly prone to develop coincidentally with long-term local steroid applications, fungus invasion must be suspected in any persistent corneal ulceration where a steroid has been used or is in use. Intraocular pressure should be checked frequently. ADVERSE REACTIONS—Glaucoma with optic nerve damage, visual acuity or field defects, posterior subcapsular cataract formation, secondary ocular infection from pathogens liberated from ocular tissues, perforation of the globe.

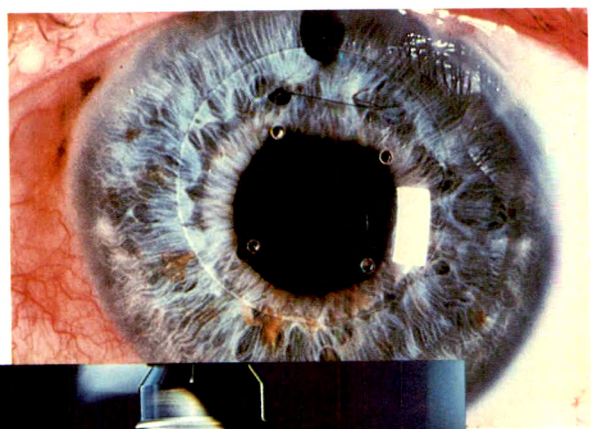
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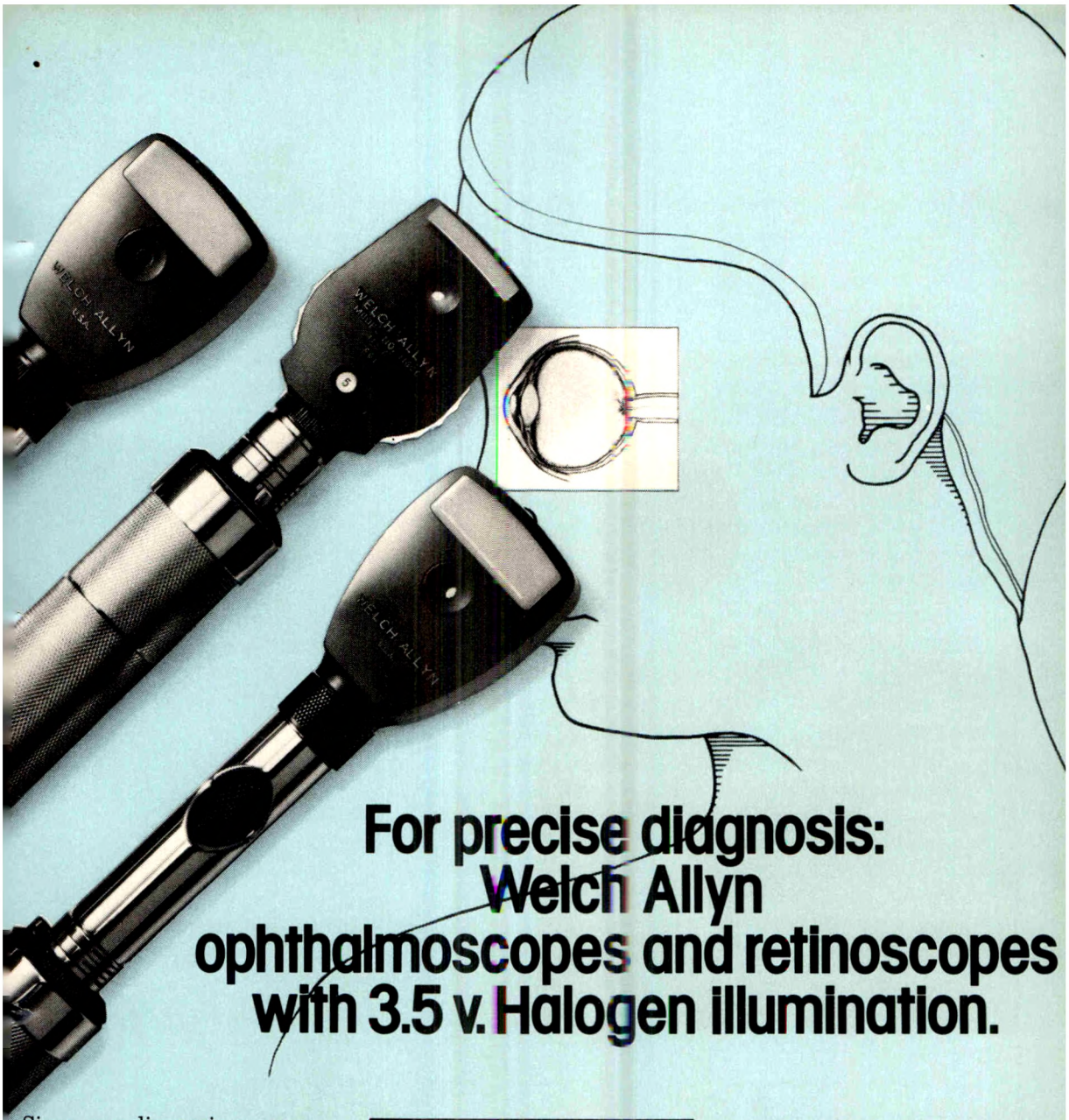
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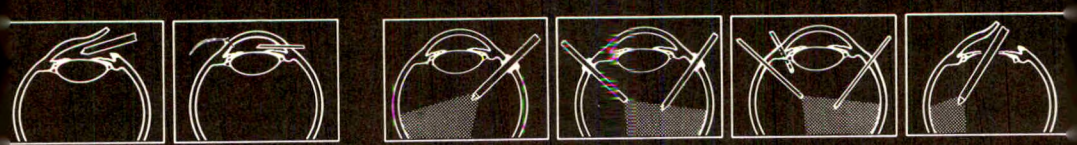
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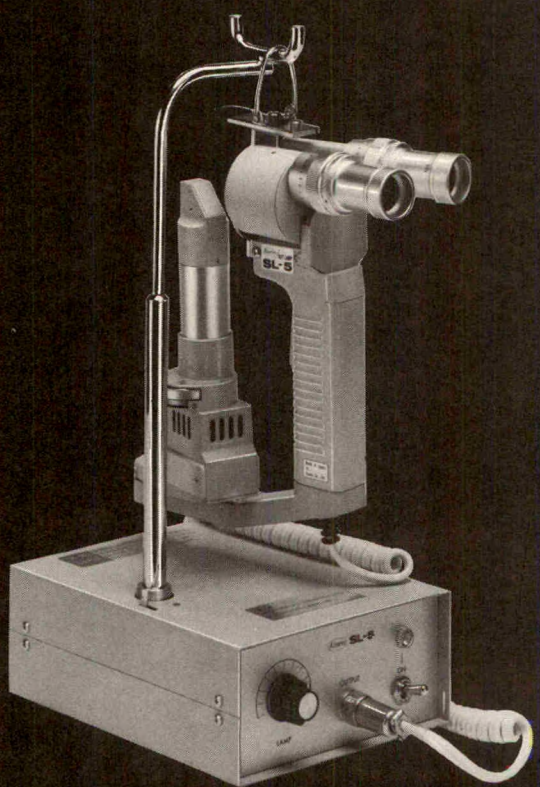
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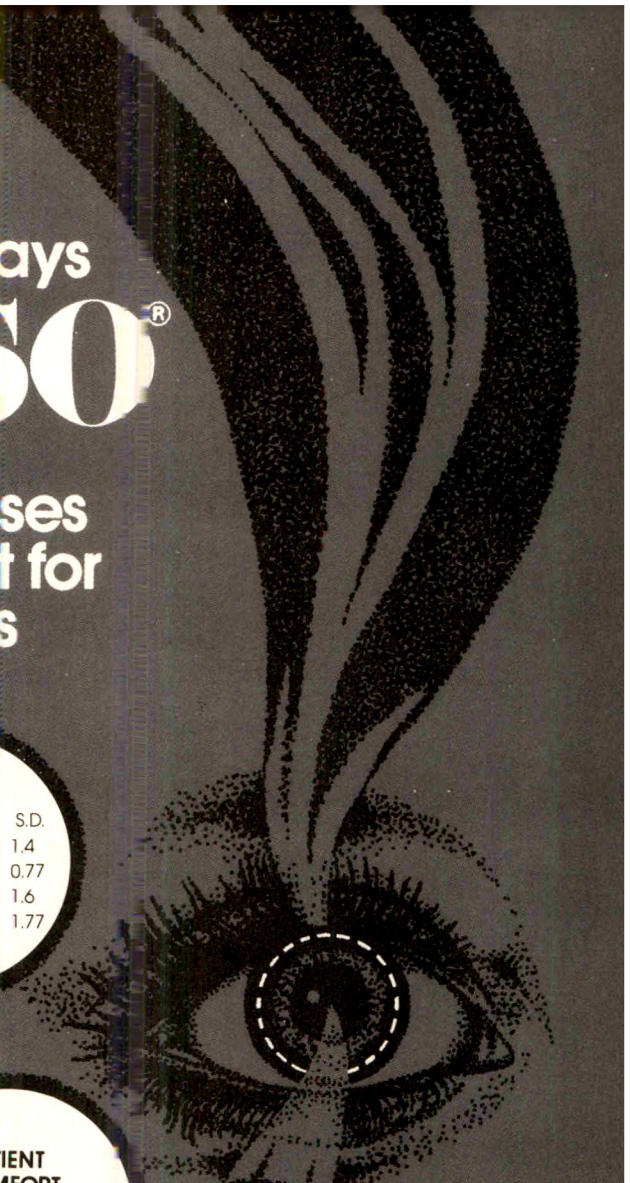
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PMMA – 0.01

WETTING ANGLE

	W.A.	S.D.
MESO –	21.1	1.4
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BP Flex –	22.3	1.6
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MESO – 4.3
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PATIENT COMFORT

MESO best –	16.5%
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No difference –	75.3

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	MESO	Polycon
>0.00 to 0.05mm –	19.2%	27.4%
>0.05 to 0.10 –	2.7	15.1
>0.10 –	–	4.1

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Polycon –	no	no	yes
PMMA –	yes	yes	yes
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MESO (cabufocon) Contact Lenses

The MESO (cabufocon) contact lens is fabricated from cellulose acetate butyrate which is a thermoplastic polymer derived from cellulose.

Description: The MESO (cabufocon) contact lens is composed of the polymer cellulose acetate butyrate.

It is manufactured in a range of base curves (6.0-9.0mm) and diameters (6.0-12.5mm) to permit custom fitting of the cornea.

It has the following physical characteristics:

- Gas permeability (@ 25° C: 4.5 x 10^{-11} cm^2/sec ml O_2/ml soln-mm Hg (Oxygen Flux Meter, Schemae Versatae)
- Thermal conductivity: 4.0-8.0 x 10^{-4} cal/sec/cm^2/1° C/cm (ASTM C177)
- Wetting angle: 46.4° dry, and 26.9° after soaking for 24 hours. (measured using the Poster Bubble Chamber technique)
- Water absorption: 1.8% by weight (ASTM D570)

The water content of the MESO (cabufocon) material when fully hydrated is 1.8% due to the polymer's free hydroxyl and carbonyl content.

Action: The MESO (cabufocon) contact lens, when placed on the human cornea, acts as a refracting medium to correct myopia, hyperopia, astigmatism and aphakia.

Indication: The MESO (cabufocon) contact lens is indicated for the correction of visual acuity in non-diseased eyes and aphakia.

Contra-indication: MESO (cabufocon) contact lenses are contraindicated in the presence of any of the following conditions:

1. Active conjunctivitis.
2. Active or chronic trachoma infection.
3. Active herpes infection.
4. Any condition which has led to extensive corneal neovascularization.
5. Corneal hypothesia except that resulting from aphakia.
6. Any situation in which the wearer is unable to maintain good lens care regimen.

Warnings: The MESO (cabufocon) contact lens must be stored only in Flex-Care or Flexsol solution.* No other storage solutions are recommended. Patients must adhere to the recommended daily care regimen. Failure to do so may result in ocular infection.

If the lens becomes more uncomfortable to the wearer than when first placed on the eye the lens should be removed, checked for dirt or scratches, and cleaned. If this condition persists, or if the eye becomes red or painful, the lens should be removed immediately. If symptoms persist, your eye-care practitioner should be consulted.

The MESO (cabufocon) contact lens should be removed before sleeping or swimming and should not be worn in the presence of noxious fumes or vapors. If unusual blurring occurs, the lens should not be worn until the condition subsides.

Precautions:

Lens care – Storage: When the MESO (cabufocon) contact lens is removed from the eye, it should be stored only in Flex-Care or Flexsol sterile soaking solution after cleaning with Preflex* and rinsing with Flex-Care or Normol solution.* The lens should be completely covered with the storage solution. Fill the lens case receptacle till just below the rim.

Before placing the MESO (cabufocon) lens on the eye, rinse the lens with Flex-Care or Normol solution. Discard the old rinsing solution in the case, rinse the case with Flex-Care or Normol, and dry the case. Refill with fresh storage solution each time the lens is stored.*

The MESO (cabufocon) lens, unlike the hydrophilic soft lens, does not dry out or become brittle if exposed to air. If the lens does become dry, it should be soaked in storage solution for a minimum of four hours before reinsertion on the eye.

Fluorescein may be used with the lens.

Lens cleaning: Ordinary mucous and oily deposits are removed with daily cleaning of the lens with Preflex as described in your patient booklet.

Adverse reactions: As with any contact lens, corneal edema and corneal abrasions may occur if the lens is not properly fit, or if the wearing time is excessive.

Dose and administration: A gradual build-up of wearing time is recommended. A recommended schedule is as follows:

A) On the day of the initial fitting, two to four hours continuous wear.
B) Increase one hour each day for one week until eight continuous hours have been achieved.
C) If checkup is normal, wearing time is again increased one hour a day until full time (12 hours or more) is achieved.

Wearing schedule should be determined by the eye care practitioner.

Studies have not been performed to support safety for extended wear of this lens.

How supplied: Each lens is supplied in a sealed sterile packet. The packet is identified with the dioptric power, base curve, center thickness and manufacturer's lens number.

When the lens is removed from the packet, it should be stored in Flex-Care or Flexsol solution for at least four hours prior to dispensing to ensure maximum patient comfort.

CAUTION: Federal law prohibits dispensing without prescription.

Complete list of recommended solutions for Cleaning, Rinsing and Soaking MESO lenses:

Cleaning:	Preflex* Cleaning Solution Soft Mate* Cleaning Solution
Rinsing:	Flex-Care* Rinsing, Storing and Disinfecting Solution Soft Mate* Rinsing Solution Normol* Rinsing Solution
Soaking:	Flex-Care* Rinsing, Storing and Disinfecting Solution Flexsol* Soaking Solution Soft Mate* Disinfection & Storage Solution

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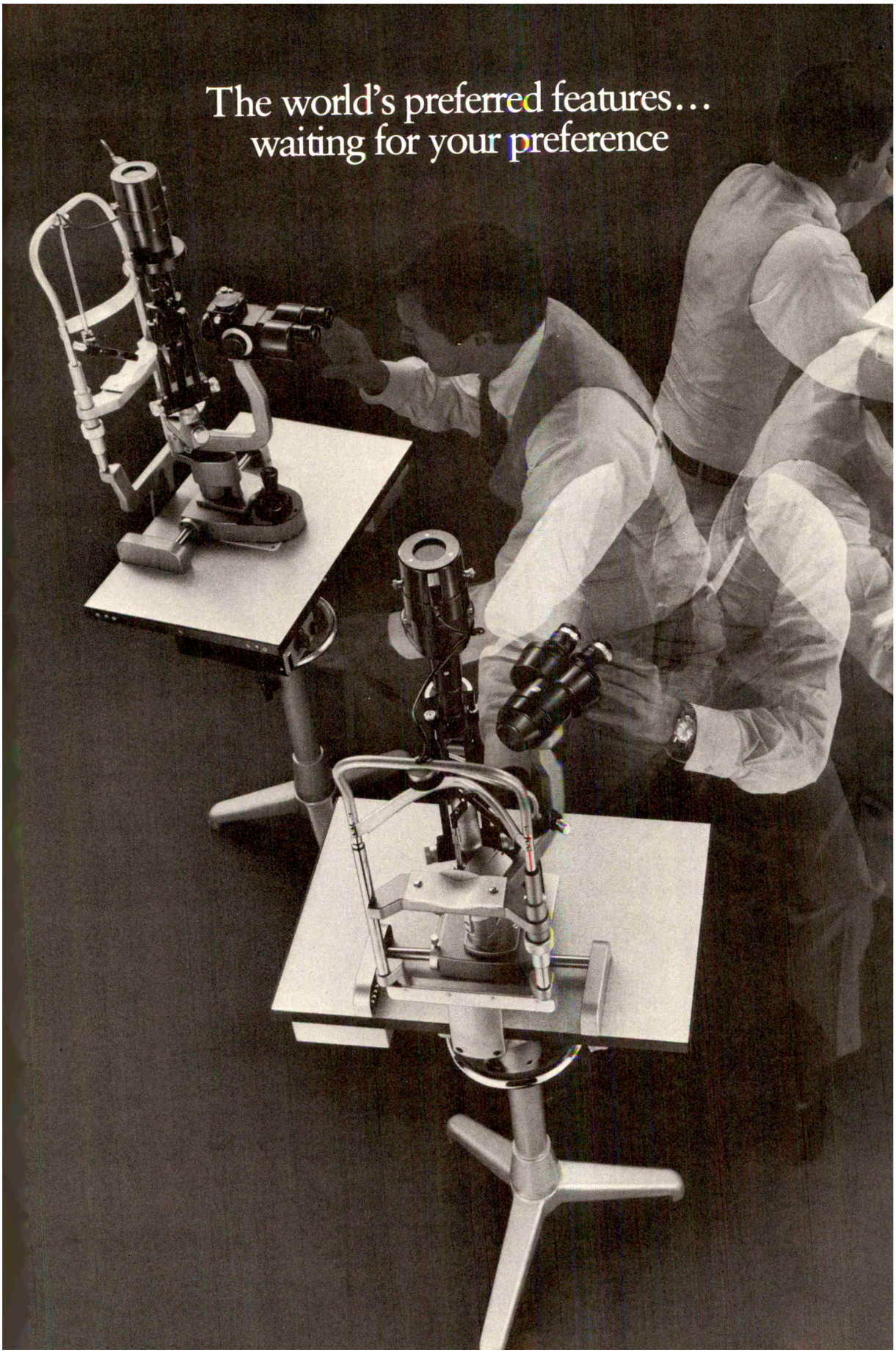
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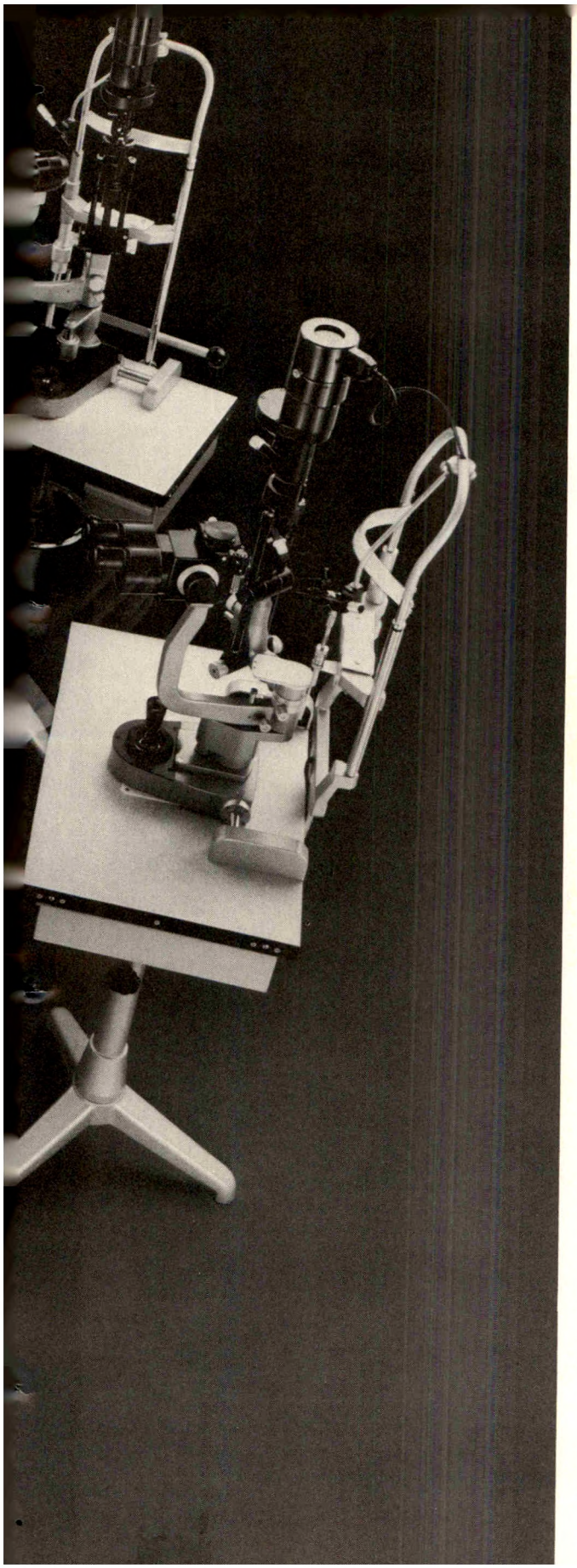
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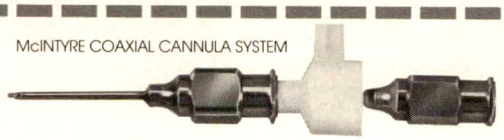
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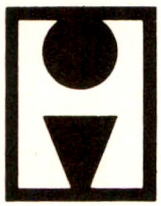
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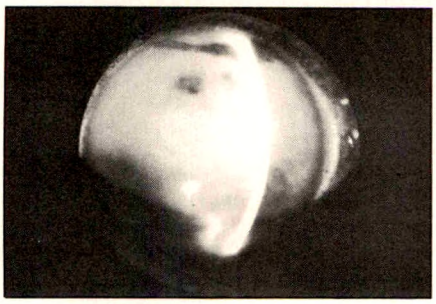
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Contents and Contributors

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Unit 3—GRANULOMATOUS INFLAMMATION OF THE EYE—Daniel M. Albert, MD, 31 slides
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Unit 13—RETINOBLASTOMA AND PSEUDOGLIOMA—Daniel M. Albert, MD, and Delia N. Sang, MD, 30 slides
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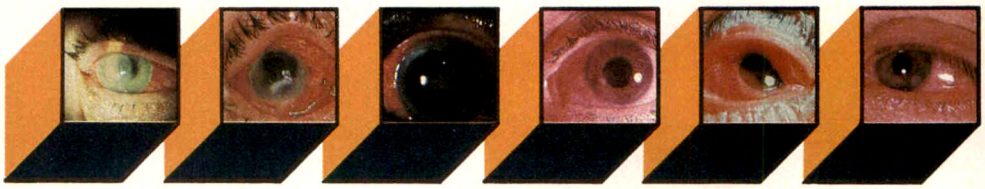
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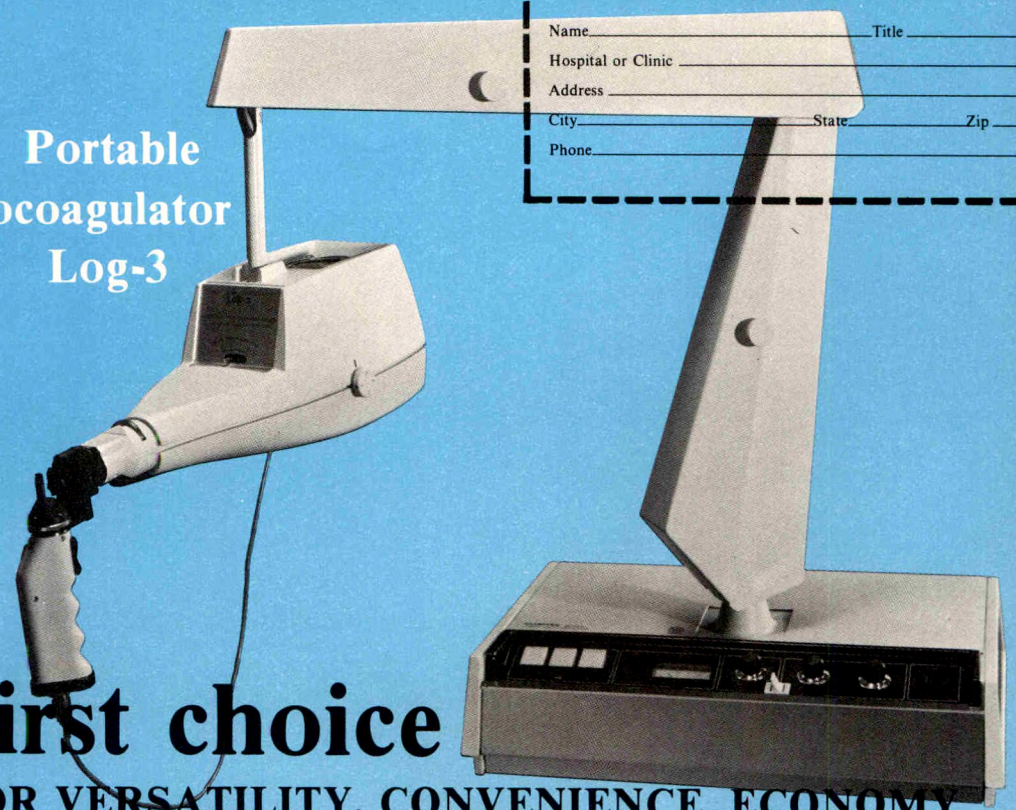
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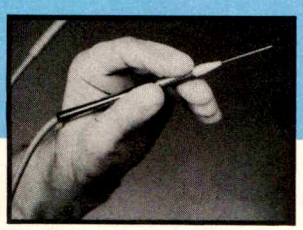
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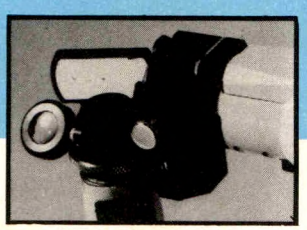


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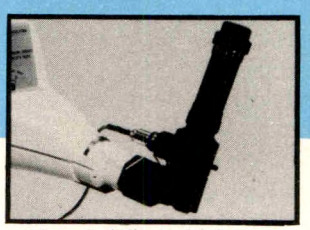
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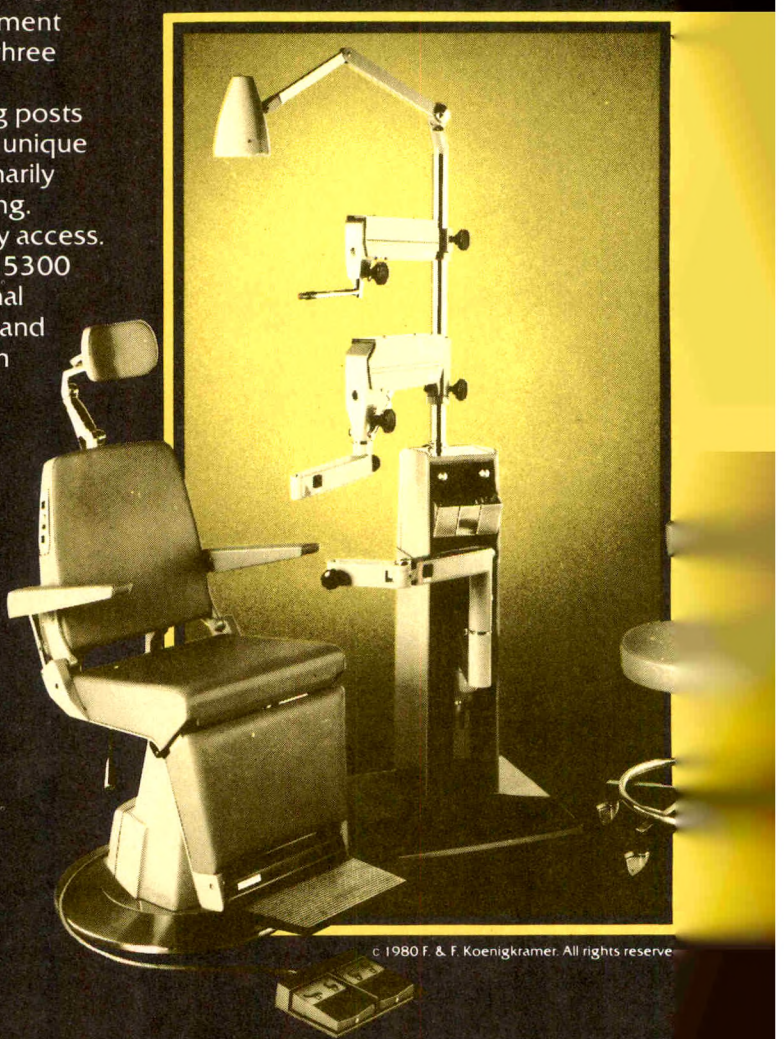
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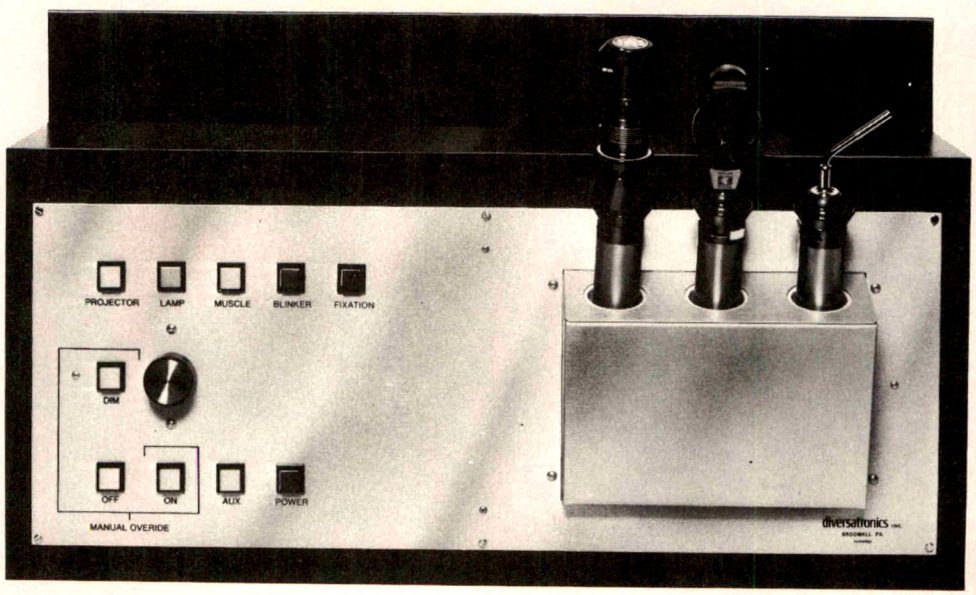
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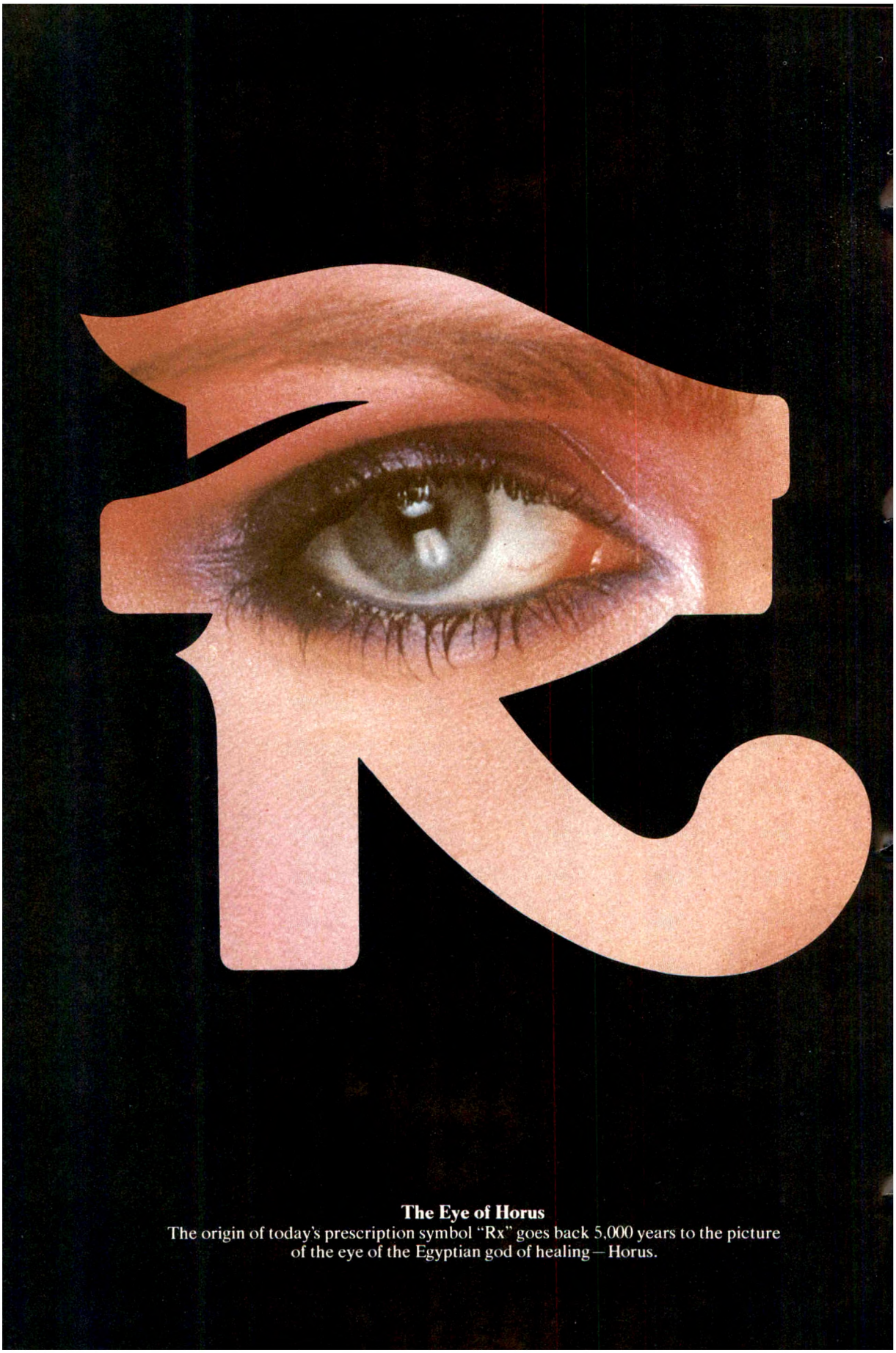


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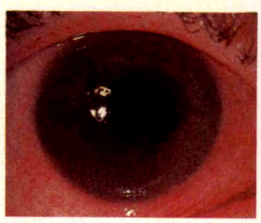
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Intraocular penetration of trifluridine occurs after topical instillation of VIROPTIC into human eyes. Decreased corneal integrity or stromal or uveal inflammation may enhance the penetration of trifluridine into the aqueous humor. Unlike the results of ocular penetration of trifluridine *in vitro,* 5-carboxy-2′-deoxyuridine was not found in detectable concentrations within the aqueous humor of the human eye.

INDICATIONS AND USAGE: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% is indicated for the treatment of primary keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus, types 1 and 2. VIROPTIC is also effective in the treatment of epithelial keratitis that has not responded clinically to the topical administration of idoxuridine or when ocular toxicity or hypersensitivity to idoxuridine has occurred. In a smaller number of patients found to be resistant to topical vidarabine, VIROPTIC was also effective.

The clinical efficacy of VIROPTIC in the treatment of stromal keratitis and uveitis due to Herpes simplex or ophthalmic infections caused by vacciniavirus and Adenovirus has not been established by well-controlled clinical trials. VIROPTIC has not been shown to be effective in the prophylaxis of Herpes simplex virus keratoconjunctivitis and epithelial keratitis by well-controlled clinical trials. VIROPTIC is not effective against bacterial, fungal or chlamydial infections of the cornea or nonviral trophic lesions.

During controlled multicenter clinical trials, 92 of 97 (95%) patients (78 of 81 with dendritic and 14 of 16 with geographic ulcers) responded to VIROPTIC therapy as evidenced by complete corneal re-epithelialization within the 14 day therapy period. In these controlled studies, 56 of 75 (75%) patients (49 of 58 with dendritic and 7 of 17 with geographic ulcers) responded to idoxuridine therapy. The mean time to corneal re-epithelialization for dendritic ulcers (6 days) and geographic ulcers (7 days) was similar for both therapies. In other clinical studies, VIROPTIC was evaluated in the treatment of Herpes simplex virus keratitis in patients who were unresponsive or intolerant to the topical administration of idoxuridine or vidarabine. VIROPTIC was effective in 138 of 150 (92%) patients (109 of 114 with dendritic and 29 of 36 with geographic ulcers) as evidenced by corneal re-epithelialization. The mean time to corneal re-epithelialization was 6 days for patients with dendritic ulcers and 12 days for patients with geographic ulcers.

CONTRAINDICATIONS: VIROPTIC brand Trifluridine Ophthalmic Solution, 1%, is contraindicated for patients who develop hypersensitivity reactions or chemical intolerance to trifluridine.

WARNINGS: The recommended dosage and frequency of administration should not be exceeded (see Dosage and Administration).

PRECAUTIONS:

General: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% should be prescribed only for patients who have a clinical diagnosis of herpetic keratitis.

VIROPTIC may cause mild local irritation of the conjunctiva and cornea when instilled but these effects are usually transient.

Although documented *in vitro* viral resistance to trifluridine has not been reported following multiple exposure to VIROPTIC, the possibility exists of viral resistance development.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Mutagenic Potential. Trifluridine has been shown to exert mutagenic, DNA damaging and cell transforming activities in various standard *in vitro* test systems, and clastogenic activity in *Vicia faba* cells.

Although the significance of these test results is not clear or fully understood, there exists the possibility that mutagenic agents may cause genetic damage in humans.

Oncogenic Potential. The oncogenic potential of trifluridine is unknown at this time. The oncogenic potential of trifluridine in rodents is being evaluated.

Pregnancy: The drug should not be prescribed for pregnant women unless the potential benefits outweigh the potential risks.

Nursing Mothers: It is unlikely that trifluridine is excreted in human milk after ophthalmic instillation of VIROPTIC because of the relatively small dosage ($\leq$5.0 mg/day), its dilution in body fluids and its extremely short half-life (approximately 12 minutes). The drug should not be prescribed for nursing mothers unless the potential benefits outweigh the potential risks.

ADVERSE REACTIONS: The most frequent adverse reactions reported during controlled clinical trials were mild, transient burning or stinging upon instillation (4.6%) and palpebral edema (2.8%). Other adverse reactions in decreasing order of reported frequency were superficial punctate keratopathy, epithelial keratopathy, hypersensitivity reaction, stromal edema, irritation, keratitis sicca, hyperemia, and increased intraocular pressure.

OVERDOSAGE: Overdosage by ocular instillation is unlikely because any excess solution should be quickly expelled from the conjunctival sac.

Acute overdosage by accidental oral ingestion of VIROPTIC has not occurred. However, should such ingestion occur, the 75 mg dosage of trifluridine in a 7.5 ml bottle of VIROPTIC is not likely to produce adverse effects. Single intravenous doses of 15-30 mg/kg/day in children and adults with neoplastic disease produce reversible bone marrow depression as the only potentially serious toxic effect and only after 3-5 courses of therapy. The acute oral LD_{50} in the mouse and rat was 4379 mg/kg or higher.

DOSAGE AND ADMINISTRATION: Instill one drop of VIROPTIC Ophthalmic Solution, 1% onto the cornea of the affected eye every two hours while awake for a maximum daily dosage of nine drops until the corneal ulcer has completely re-epithelialized. Following re-epithelialization, treatment for an additional seven days of one drop every four hours while awake for a minimum daily dosage of five drops is recommended.

If there are no signs of improvement after seven days of therapy or complete re-epithelialization has not occurred after 14 days of therapy, other forms of therapy should be considered. Continuous adminsitration of VIROPTIC for periods exceeding 21 days should be avoided because of potential ocular toxicity.

HOW SUPPLIED: VIROPTIC Ophthalmic Solution, 1% is supplied as a sterile ophthalmic solution in a plastic Drop-Dose® dispenser bottle of 7.5 ml.

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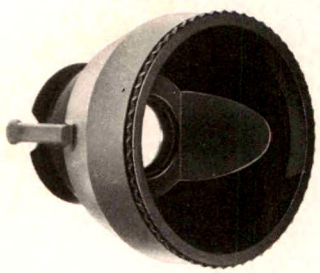
Large Three-mirror lens



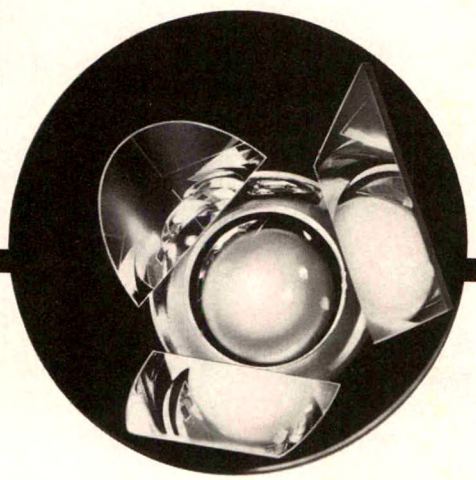
Three-mirror lens for babies, 10 mm



Three-mirror lens for children, 11 mm



Ora one-mirror lens with depressor

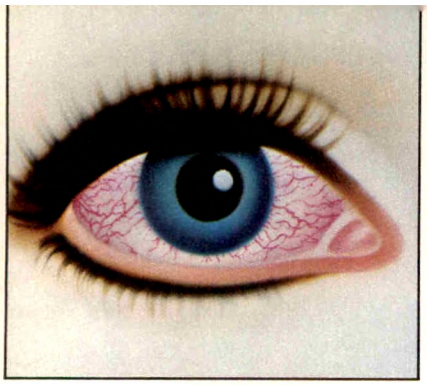


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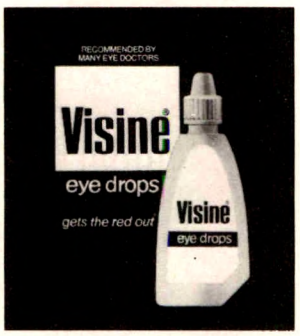
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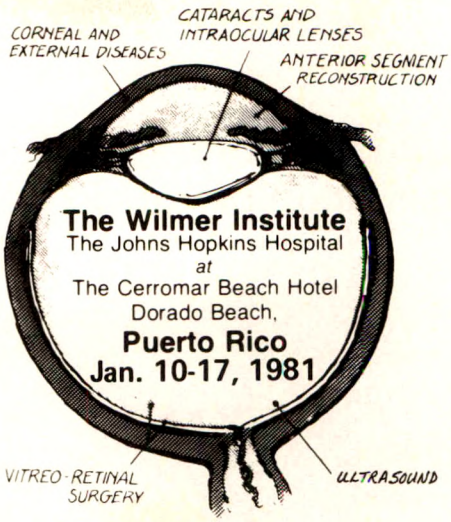
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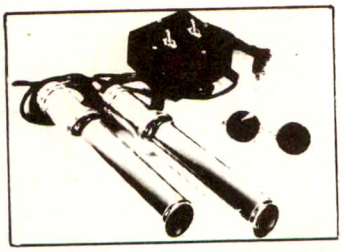
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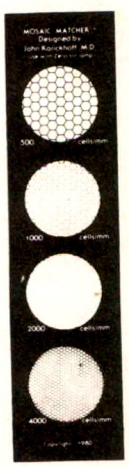
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- **Single-entity antibiotic.**
- **Generally low potential for sensitivity reactions and irritation.**

DESCRIPTION Gentamicin sulfate is a water-soluble antibiotic of the aminoglycoside group active against a wide variety of pathogenic gram-negative and gram-positive bacteria.

GARAMYCIN **Ophthalmic Solution** is a sterile, aqueous solution buffered to approximately pH 7 for use in the eye. Each ml. contains gentamicin sulfate (equivalent to 3.0 mg. gentamicin), disodium phosphate, monosodium phosphate, sodium chloride, and benzalkonium chloride as a preservative.

GARAMYCIN **Ophthalmic Ointment** is a sterile ointment, each gram containing gentamicin sulfate (equivalent to 3.0 mg. gentamicin) in a bland base of white petrolatum, with methylparaben and propylparaben as preservatives.

ACTIONS The gram-positive bacteria against which gentamicin sulfate is active include coagulase-positive and coagulase-negative staphylococci, including certain strains that are resistant to penicillin; Group A beta-hemolytic and non-hemolytic streptococci; and *Diplococcus pneumoniae*. The gram-negative bacteria against which gentamicin sulfate is active include certain strains of *Pseudomonas aeruginosa*, indole-positive and indole-negative *Proteus* species, *Escherichia coli*, *Klebsiella pneumoniae* (Friedlander's bacillus), *Haemophilus influenzae* and *Haemophilus aegyptius* (Koch-Weeks bacillus), *Aerobacter aerogenes*, *Moraxella lacunata* (diplobacillus of Morax-Axenfeld), and *Neisseria* species, including *Neisseria gonorrhoeae*. Although significant resistant organisms have not been isolated from patients treated with gentamicin at the present time, this may occur in the future as resistance has been produced with difficulty *in vitro* by repeated exposures.

INDICATIONS GARAMYCIN **Ophthalmic Solution** and **Ointment** are indicated in the topical treatment of infections of the external eye and its adnexa caused by susceptible bacteria. Such infections embrace conjunctivitis, keratitis and keratoconjunctivitis, corneal ulcers, blepharitis and blepharoconjunctivitis, acute meibomianitis, and dacryocystitis.

CONTRAINDICATIONS GARAMYCIN **Ophthalmic Solution** and **Ointment** are contraindicated in patients with known hypersensitivity to any of the components.

WARNINGS GARAMYCIN **Ophthalmic Solution** is not for injection. It should never be injected subconjunctivally, nor should it be directly introduced into the anterior chamber of the eye.

PRECAUTIONS Prolonged use of topical antibiotics may give rise to overgrowth of nonsusceptible organisms, such as fungi. Should this occur, or if irritation or hypersensitivity to any component of the drug develops, discontinue use of the preparation and institute appropriate therapy.

Ophthalmic ointments may retard corneal healing.

ADVERSE REACTIONS Transient irritation has been reported with the use of GARAMYCIN **Ophthalmic Solution.**

Occasional burning or stinging may occur with the use of GARAMYCIN **Ophthalmic Ointment.**

DOSAGE AND ADMINISTRATION GARAMYCIN **Ophthalmic Solution:** instill one or two drops into the affected eye every four hours. In severe infections, dosage may be increased to as much as two drops once every hour.

GARAMYCIN **Ophthalmic Ointment:** apply a small amount to the affected eye two to three times a day.

HOW SUPPLIED GARAMYCIN **Ophthalmic Solution—Sterile,** 5-ml. plastic dropper bottle, sterile, boxes of one and six. Store away from heat.

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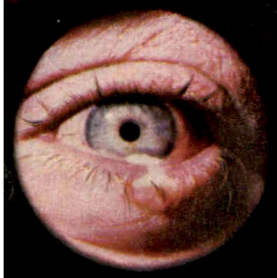
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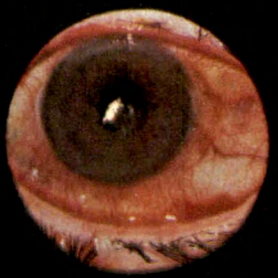
Acute catarrhal conjunctivitis with episcleritis. Etiology: S. aureus. Before treatment.



7th day post treatment with GARAMYCIN Ophthalmic. Culture negative.



Corneal ulcer. Etiology: S. aureus. Before treatment.



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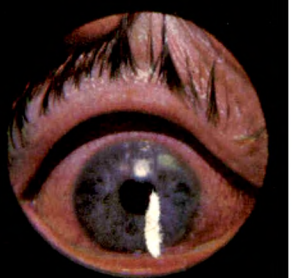
Acute hemorrhagic conjunctivitis. Etiology: Pneumococcus. Before treatment.



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Corneal marginal ulcer. Etiology: S. aureus. Before treatment.



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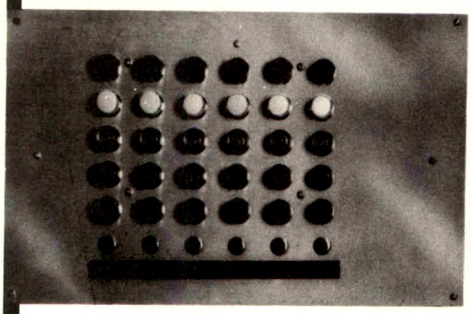
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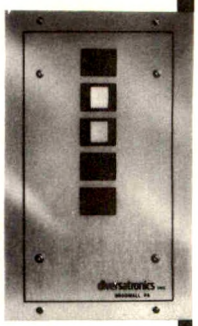
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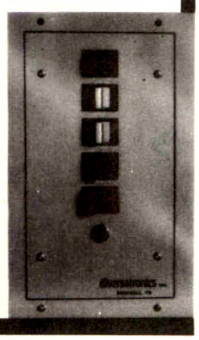
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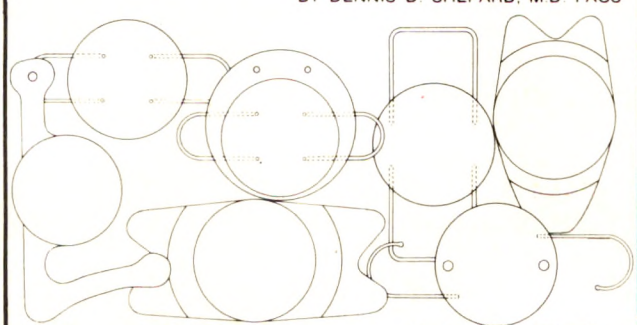
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11:15 - 12:15	Lens, Aqueous Biochemistry	Leo T. Chylack, Jr.
1:15 - 3:15	Glaucoma	Richard J. Simmons, Bernard Schwartz
3:30 - 5:30	Pharmacology and Therapeutics	Bernard Schwartz
6:00 - 8:00	Cocktail Party	

Sunday, December 7, 1980

7:45 - 9:45	Plastic Surgery	Arthur S. Grove, Jr.
10:00 - 12:00	Lacrimal Surgery, Orbital Disease	Arthur S. Grove, Jr.
12:45 - 2:45	Corneal, External Disease	Claes H. Dohlman, Kenneth R. Kenyon
3:00 - 5:00	Microbiology, Immunology	Mathea R. Allansmith

Friday, December 12, 1980

7:30 - 9:30	Neuro-Ophthalmology	Simmons Lessell
9:45 - 11:30	Neuro-Ophthalmology	Simmons Lessell
12:30 - 1:45	Emergency Medicine and Anesthesia	John V. Donlon, Jr.
1:45 - 3:15	Strabismus	Lloyd Wilcox
3:30 - 5:30	Strabismus	Lloyd Wilcox

Saturday, December 13, 1980

7:45 - 8:30	Embryology	Film
8:30 - 9:45	Developmental Anomalies	Richard M. Robb
10:00 - 11:00	Pediatric Inflammation	William P. Boger III
11:00 - 12:00	Genetics	Robert A. Petersen
12:45 - 2:45	Pathology	Daniel M. Albert
3:00 - 5:00	Pathology	Daniel M. Albert

Sunday, December 14, 1980

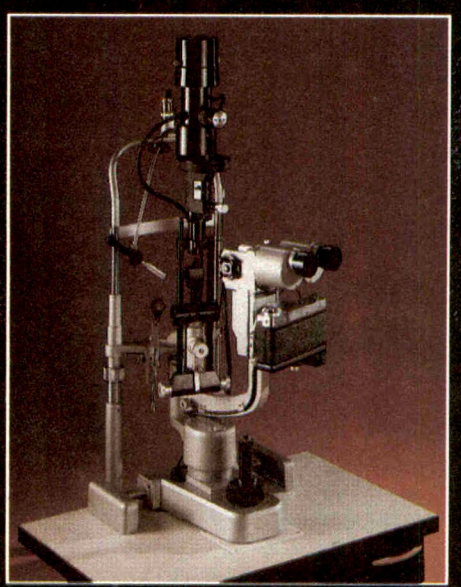
7:30 - 8:45	Contact Lenses, Optics	David Miller
9:00 - 11:00	Retinal Electrophysiology, psychophysics	Eliot L. Berson
11:00 - 11:45	Retina, Vitreous I	John Weiter
12:45 - 3:15	Retina, Vitreous II	Edward A. Ryan
3:30 - 5:00	Retina, Vitreous III	John Weiter

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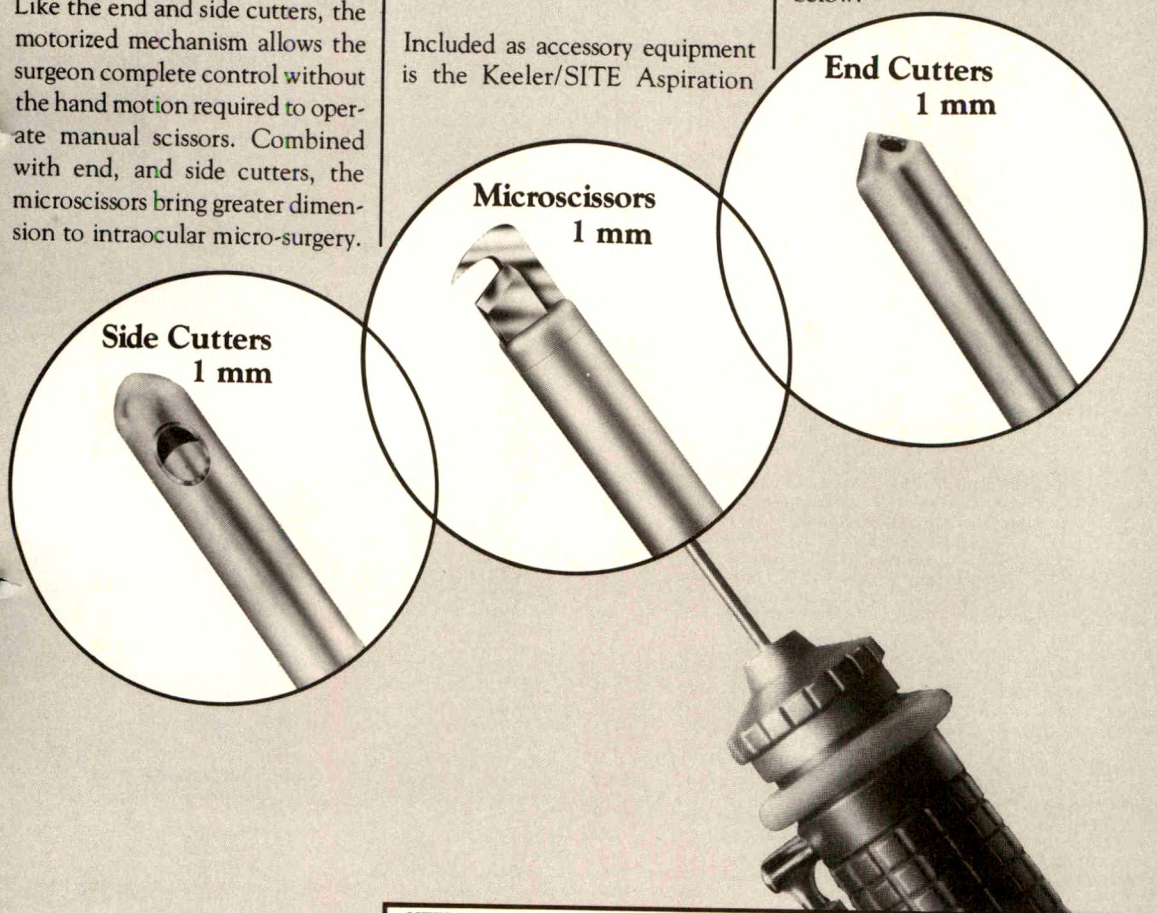
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Description. Vira-A (vidarabine, adenine arabinoside, or Ara-A) is an antiviral drug for topical ophthalmic administration. The chemical name is 9-β-D-arabinofuranosyladenine. Each gram of the ophthalmic ointment contains 30 mg of vidarabine monohydrate equivalent to 28.11 mg of vidarabine in a sterile, inert, petrolatum base.

Clinical Pharmacology. Vira-A is a purine nucleoside obtained from fermentation cultures of *Streptomyces antibioticus*. Vira-A possesses *in vitro* and *in vivo* antiviral activity against Herpes simplex types 1 and 2, Varicella-Zoster, and Vaccinia viruses. Except for Rhabdovirus and Oncornavirus, Vira-A does not display *in vitro* antiviral activity against other RNA or DNA viruses, including Adenovirus.

The antiviral mechanism of action has not been established. Vira-A appears to interfere with the early steps of viral DNA synthesis. Vira-A is rapidly deaminated to arabinosylhypoxanthine (Ara-Hx), the principal metabolite. Ara-Hx also possesses *in vitro* antiviral activity but this activity is less than that of Vira-A. Because of the low solubility of Vira-A, trace amounts of both Vira-A and Ara-Hx can be detected in the aqueous humor only if there is an epithelial defect in the cornea. If the cornea is normal, only trace amounts of Ara-Hx can be recovered from the aqueous humor.

Systemic absorption of Vira-A should not be expected to occur following ocular administration and swallowing lacrimal secretions. In laboratory animals, Vira-A is rapidly deaminated in the gastrointestinal tract to Ara-Hx.

In contrast to topical idoxuridine, Vira-A demonstrated less cellular toxicity in the regenerating corneal epithelium of the rabbit.

Indications and Usage. Vira-A Ophthalmic Ointment, 3%, is indicated for the treatment of acute keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus types 1 and 2. Vira-A is also effective in superficial keratitis caused by Herpes simplex virus which has not responded to topical idoxuridine or when toxic or hypersensitivity reactions to idoxuridine have occurred. The effectiveness of Vira-A Ophthalmic Ointment, 3%, against stromal keratitis and uveitis due to Herpes simplex virus has not been established.

The clinical diagnosis of keratitis caused by Herpes simplex virus is usually established by the presence of typical dendritic or geographic lesions on slit-lamp examination. In controlled and uncontrolled clinical trials, an average of seven and nine days of continuous Vira-A Ophthalmic Ointment, 3%, therapy was required to achieve corneal re-epithelialization. In the controlled trials, 70 of 81 subjects (86%) re-epithelialized at the end of three weeks of therapy. In the uncontrolled trials, 101 of 142 subjects (71%) re-epithelialized at the end of three weeks. Seventy-five percent of the subjects in these uncontrolled trials had either not healed previously or had developed hypersensitivity to topical idoxuridine therapy.

The following topical antibiotics: gentamicin, erythromycin, chloramphenicol; or topical steroids: prednisolone or dexamethasone, have been administered concurrently with Vira-A Ophthalmic Ointment, 3%, without an increase in adverse reactions.

Normally, corticosteroids alone are contraindicated in Herpes simplex virus infections of the eye. If Vira-A Ophthalmic Ointment, 3%, is administered concurrently with topical corticosteroid therapy, corticosteroid-induced ocular side effects must be considered. These include corticosteroid-induced glaucoma or cataract formation and progression of a bacterial or viral infection.

Vira-A is not effective against RNA virus or adenoviral ocular infections. Vira-A is also not effective against bacterial, fungal, or chlamydial infections of the cornea or nonviral trophic ulcers.

Contraindication. Vira-A Ophthalmic Ointment, 3%, is contraindicated in patients who develop hypersensitivity reactions to it.

Warnings. ***Use in Pregnancy:*** Vira-A (vidarabine) parenterally is teratogenic in rats and rabbits. Ten percent Vira-A ointment applied to 10% of the body surface during organogenesis induced fetal abnormalities in rabbits. When 10% Vira-A ointment was applied to 2 to 3% of the body surface of rabbits, no fetal abnormalities were found. This dose greatly exceeds the total recommended ophthalmic dose in humans. The possibility of embryonic or fetal damage in pregnant women receiving Vira-A (vidarabine ophthalmic ointment), 3%, is remote. The topical ophthalmic dose is small, and the drug relatively insoluble. Its ocular penetration is very low. However, a safe dose for a human embryo or fetus has not been established. Consequently, Vira-A should be used only when clearly indicated.

It is not known whether Vira-A is excreted in human milk. As a general rule, because many drugs are excreted in human milk, nursing should not be undertaken while a patient is under treatment. However, excretion of vidarabine in breast milk is unlikely because Vira-A is rapidly deaminated in the gastrointestinal tract.

Mutagenic Potential: Results of *in vitro* experiments indicate that vidarabine can be incorporated into mammalian DNA and can induce mutation in mammalian cells (mouse L5178Y cell line). Thus far, *in vivo* studies have not been as conclusive, but there is some evidence (dominant lethal assay in mice) that vidarabine may be capable of producing mutagenic effects in male germ cells.

It has also been reported that vidarabine causes chromosome breaks and gaps when added to human leukocytes *in vitro*. While the significance of these effects in terms of mutagenicity is not fully understood, there is a well-known correlation between the ability of various agents to produce such effects and their ability to produce heritable genetic damage.

Oncogenic Potential: Chronic parenteral (IM) studies of vidarabine have been conducted in mice and rats.

In the mouse study, there was a statistically significant increase in liver tumor incidence among the vidarabine-treated females. In the same study, some vidarabine-treated male mice developed kidney neoplasia. No renal tumors were found in the vehicle-treated control mice or the vidarabine-treated female mice.

In the rat study, intestinal, testicular, and thyroid neoplasia occurred with greater frequency among the vidarabine-treated animals than in the vehicle-treated controls. The increases in thyroid adenoma incidence in the high-dose (50 mg/kg) males and the low-dose (30 mg/kg) females were statistically significant.

Hepatic megalocytosis, associated with vidarabine treatment, has been found in short- and long-term rodent (rat and mouse) studies. It is not clear whether or not this represents a preneoplastic change.

The recommended frequency and duration of administration should not be exceeded (see Dosage and Administration).

Precautions. The diagnosis of keratoconjunctivitis due to Herpes simplex virus should be established clinically prior to prescribing Vira-A Ophthalmic Ointment, 3%.

Patients should be forewarned that Vira-A Ophthalmic Ointment, 3%, like any ophthalmic ointment, may produce a temporary visual haze.

Although viral resistance to Vira-A has not been observed, this possibility may exist.

Adverse Reactions. Lacrimation, foreign-body sensation, conjunctival injection, burning, irritation, superficial punctate keratitis, pain, photophobia, punctal occlusion, and sensitivity have been reported with Vira-A Ophthalmic Ointment, 3%. The following have also been reported but appear disease-related: uveitis, stromal edema, secondary glaucoma, trophic defects, corneal vascularization, and hyphema.

Overdosage. Acute massive overdosage by oral ingestion of the ophthalmic ointment has not occurred. However, the rapid deamination to arabinosylhypoxanthine should preclude any difficulty. The oral LD_{50} for vidarabine is greater than 5,020 mg/kg in mice and rats. No untoward effects should result from ingestion of the entire contents of a tube.

Overdosage by ocular instillation is unlikely because any excess should be quickly expelled from the conjunctival sac. Too frequent administration should be avoided.

Dosage and Administration. Administer approximately one half inch of Vira-A Ophthalmic Ointment, 3%, into the lower conjunctival sac five times daily at three-hour intervals.

If there are no signs of improvement after 7 days, or complete re-epithelialization has not occurred by 21 days, other forms of therapy should be considered. Some severe cases may require longer treatment.

After re-epithelialization has occurred, treatment for an additional seven days at a reduced dosage (such as twice daily) is recommended in order to prevent recurrence.

How Supplied. N 0071-3677-07 (Stock 18-1677-139)

Vira-A Ophthalmic Ointment, 3%, is supplied sterile in ophthalmic ointment tubes of 3.5 g. The base is a 60:40 mixture of solid and liquid petrolatum. **UK**

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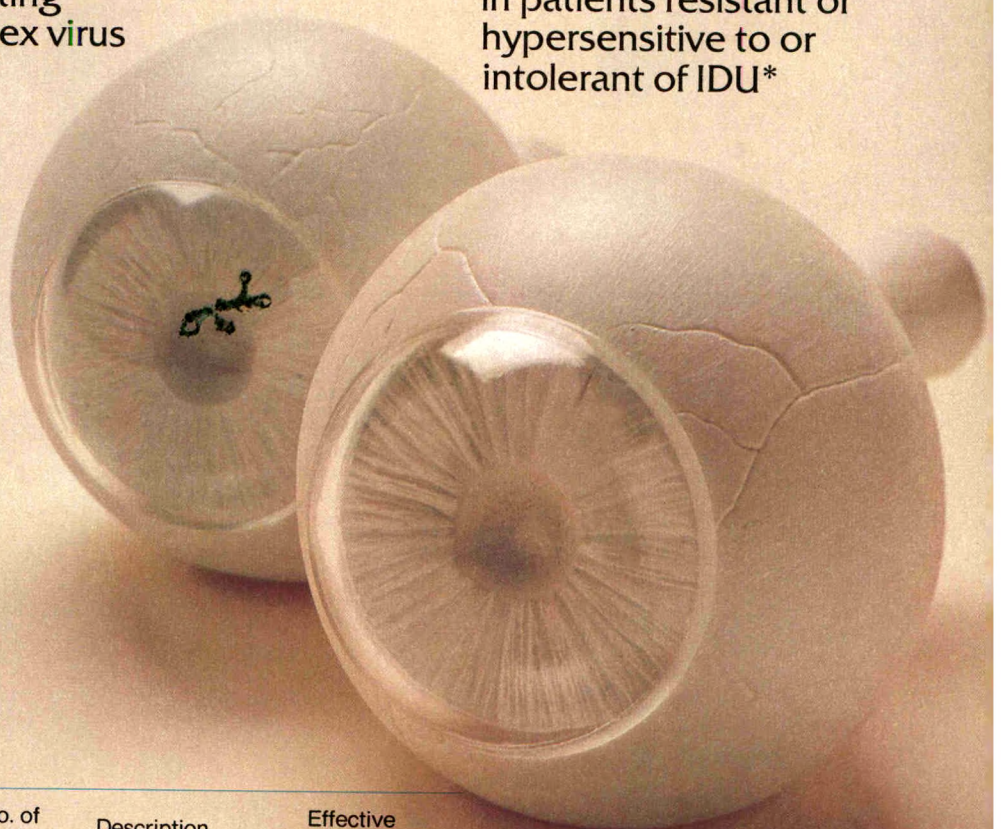
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Clinical Evidence	No. of Subjects	Description	Effective Response
Vira-A Controlled Trials	81	Previously untreated	70 subjects **(86%)**
Vira-A Uncontrolled Trials	142	75% had been resistant or hypersensitive to IDU	101 subjects **(71%)**

NOTE: In the controlled and uncontrolled trials, 70 and 101 subjects, respectively, re-epithelialized at the end of three weeks on Vira-A.

*Data on file, Medical Department, Parke-Davis

Vira-A is preservative-free.

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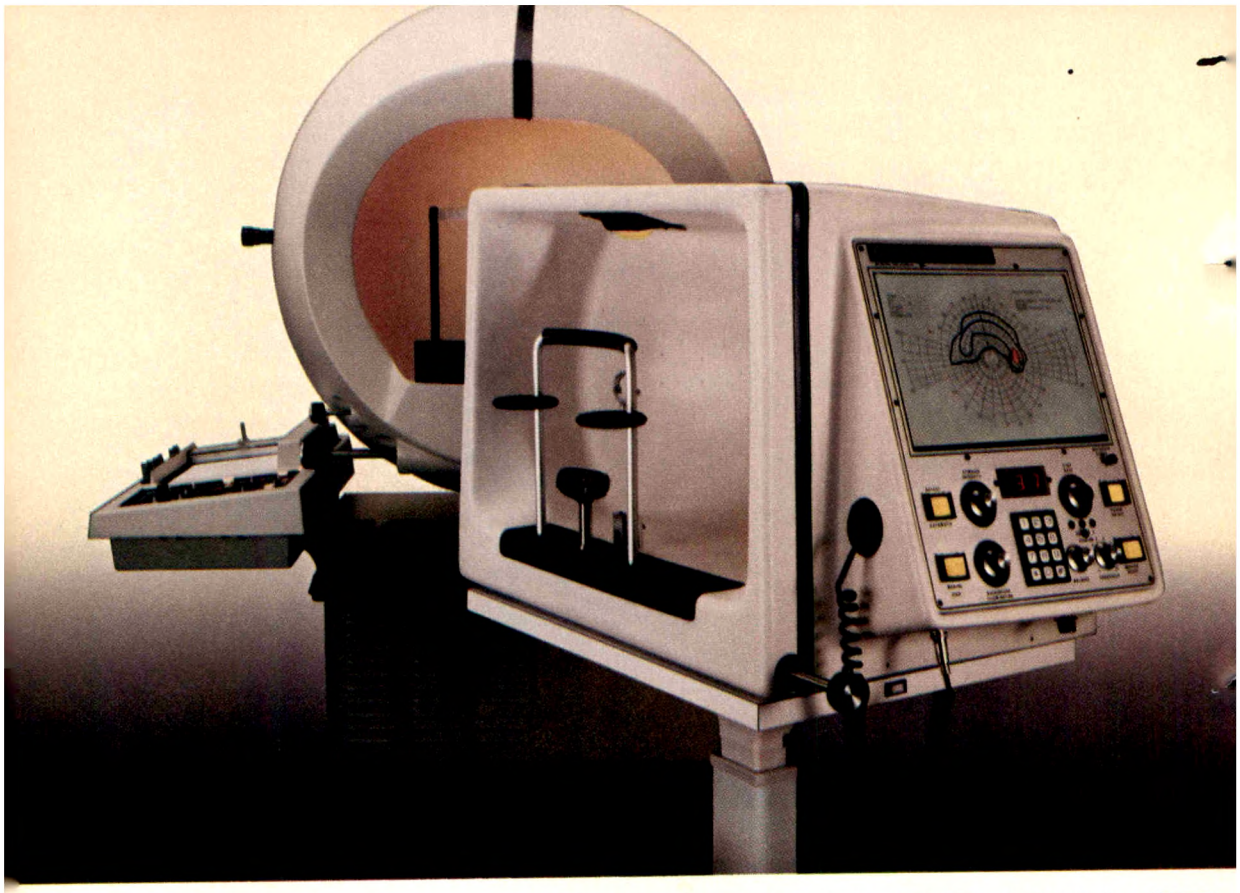
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John Pearce, M.D.
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Saburo Hayano, M.D.
Japan
Svyatoslav Fyodorov, M.D.
USSR
S. Peter Choyce, M.D.
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Karl Jacobi, M.D.
W. Germany

Format:

This year we will offer courses on Wednesday, March 25 only. They will consist of 3 separate 2 hour sessions, giving you the opportunity to attend 3 different courses in one day, as well as view the latest products and scientific developments of our exhibitors. Thursday, Friday, and Saturday will be devoted to lecture & symposia. No old repetitious material or "my lens" panels will be allowed. Abstracts will be accepted for presentations, courses and posters through September 30, 1980.

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Below is a brief sampling of the many and varied courses to be offered this year:

Endothelial Photography Course–
Kenneth Hoffer, M.D.

Kelman Anterior Chamber Lens Course–
Charles Kelman, M.D.

Management of Lens Complications–
Robert Drews, M.D.

Use of Healon in the Anterior Segment–
David Miller, M.D.

Extracapsular Cataract Extraction with Intraocular Lens Implantation–
John Pearce, M.D.

Cataract Extraction and Lens Implantation–
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☐ Nurses $175*
☐ Technicians $175*

☐ Check here if you wish further information on the Ladies' Program

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Each Registrant must fill out a separate form. Cancellations: 90% refund prior to postmark date of Feb. 17, 10% refund a postmark date of Feb. 17. Residents must have a letter from Chief of Department. Make check or money order payable to Intraocular Lens Symposium and send with this form to P.O. Box 3140, Santa Monica, California 90403. For hotel reservatic please contact Century Plaza Hotel directly at Avenue of the Stars, Los Angeles, California 90067. Telephone (213) 277-2(Toll free reservation number (800) 228-3000. When making reservations please indicate that you are with the AIOIS.

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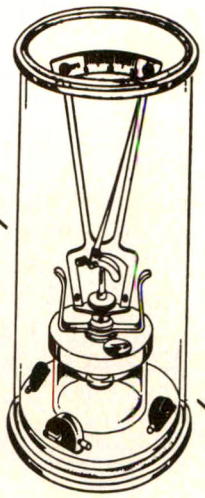
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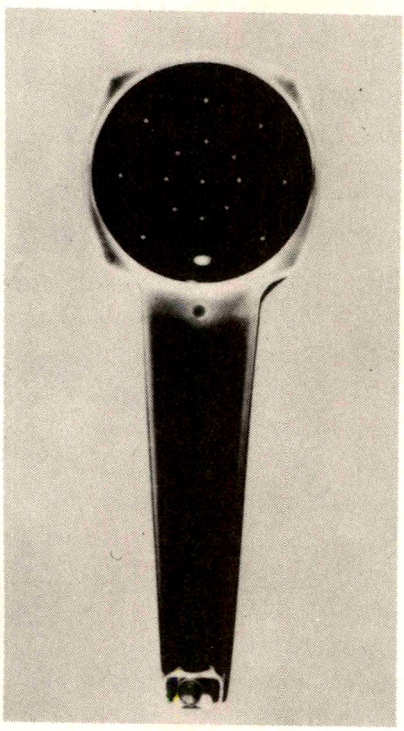
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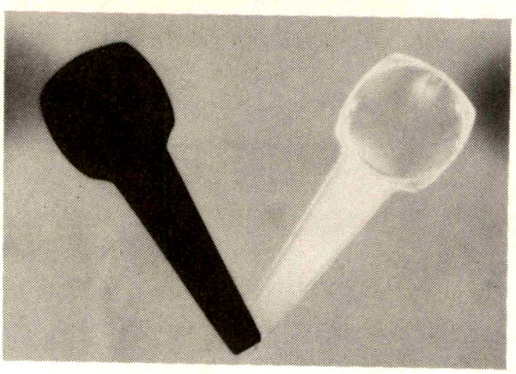
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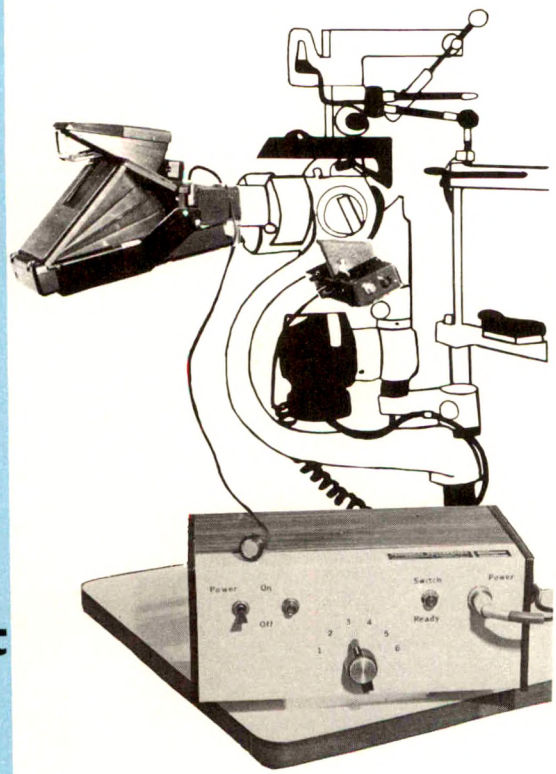
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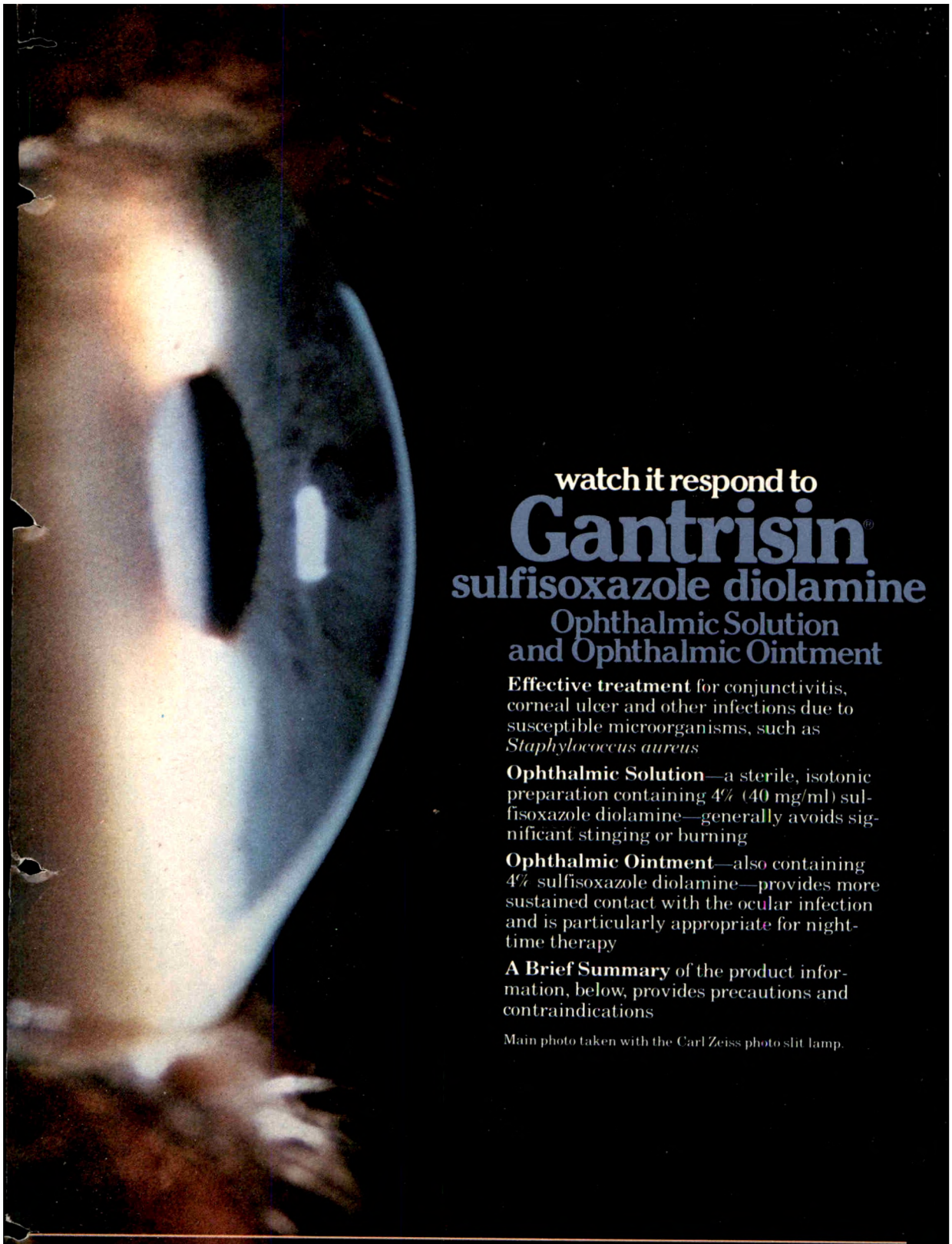
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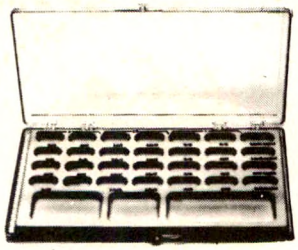
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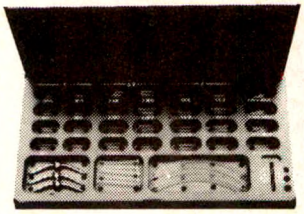
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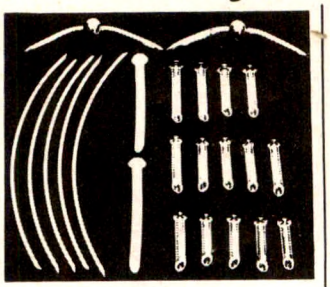
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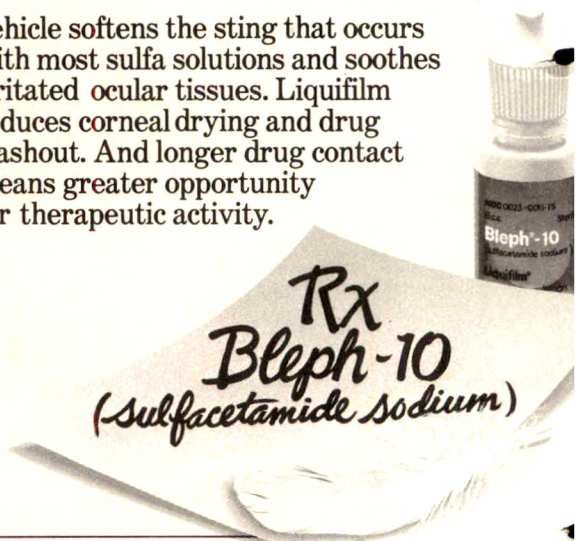


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HEREDITARY FUCHS' DYSTROPHY

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Baltimore, Maryland

Fuchs' dystrophy, originally described in 1910,[1] has been considered a sporadically occurring degenerative disease.[2] Despite its relatively common occurrence, published reports documenting familial transmission are rare.[3] Cross, Maumenee, and Cantolino[4] and, more recently, Krachmer and associates[5] have documented cases of the familial occurrence and autosomal-dominant inheritance of this disorder.

The present paper investigates further the hereditary pattern of Fuchs' dystrophy.

SUBJECTS AND METHODS

We asked 25 patients known to have Fuchs' dystrophy (diagnosed by W. J. S. or A. E. M.) to return with their first-degree relatives (parents, siblings, and children). We asked the relatives who could not come here to make appointments with their ophthalmologists whom we asked to describe the presence and degree of cornea guttata.

We examined the subjects who came here for the presence and severity of cornea guttata with the Haag-Streit slit-lamp, examining the endothelium with direct, specular illumination and retroillumination (Fig. 1). We classified cornea guttata into five grades (Table 1; Fig. 2). We diagnosed Fuchs' dystrophy only when cornea guttata became grouped, as in Grades 2 through 5.

We recorded intraocular pressure with a pneumatonometer, and measured corneal thickness by averaging three readings obtained with the Mishima-Hedbys method of pachymetry.[6] We performed specular microscopy by the method of Bourne and associates,[7] sampling both central and peripheral corneal endothelium.

RESULTS

This study includes a total of 102 patients from 25 families. We partially described two of these families previously.[4] The proband (that member of each family who was first examined here and found to have Fuchs' corneal dystrophy) and at least one first-degree relative in each family were examined by us or by their own ophthalmologists. Of the 77 relatives included, 18 were examined by their own ophthalmologists, whose reports we then used in our analyses.

Of these 25 probands, 22 were women and three men. Their average age was 67.6 years (range, 41 to 87 years); the average age did not differ significantly between men and women ($P > .5$).

From the Wilmer Ophthalmological Institute, Johns Hopkins Hospital, Baltimore, Maryland. This work was supported in part by grants EY010302 (Dr. Stark), EY01773 (Dr. Irene Maumenee), and EY02476 (Dr. Hirst) from the National Eye Institute.

Reprint requests to Walter J. Stark, M.D., Wilmer Institute, Johns Hopkins Hospital, 600 N. Wolfe St., Baltimore, MD 21205.

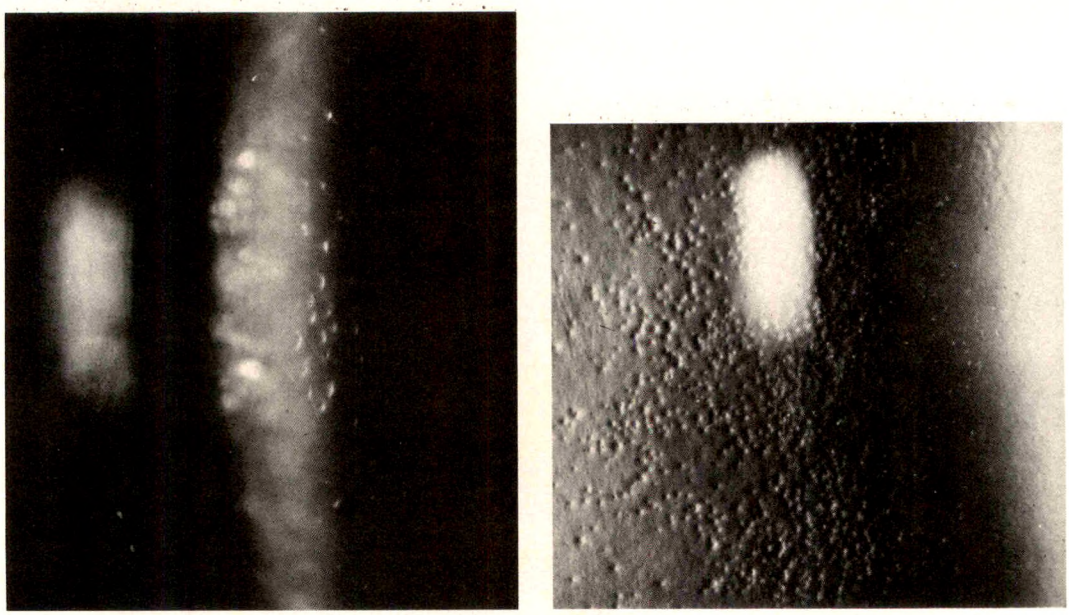


Fig. 1 (Rosenblum and associates). Cornea guttata seen by slit-lamp specular illumination (left) and by retroillumination (right).

We tried to examine as many children of probands as possible, hoping for complete selection. We also examined parents and siblings. We included 77 first-degree relatives; of these, 40 had Grade 2 or worse endothelial cornea guttata and were labeled affected. There was no significant difference in age between the females and males examined ($P > .5$); the average age was 48.3 years (range, 11 to 91 years). Five of eight (63%) of the parents examined were affected, and 21 of 26 siblings (81%). Of the probands' children aged 40 years and older, 11 of 17 (65%) were affected, but only three of 26 (12%) children less than 40 years old were affected. Overall, 14 of 43 (33%) of children of probands had Grade 2 or worse endothelial cornea guttata (Table 2). Nineteen of our 25 families (75%) had at least two affected members. The sex ratio for the total group was 40 males to 37 females; among the secondary cases it was 26 males to 25 females. Male-to-male transmission occurred in two families (Fig. 3).

TABLE 1

CORNEA GUTTATA GRADING SYSTEM

Grade	Findings
0	No central cornea guttata
1	Scattered central cornea guttata
2	1 or 2 mm of grouped central cornea guttata
3	2 to 5 mm of grouped central cornea guttata
4	> 5 mm of grouped central cornea guttata
5	Cornea guttata with epithelial edema

The average corneal thickness in nonaffected relatives (less than Grade 2 cornea guttata) was 0.56 mm. In this group, there was no significant variation of corneal thickness with age. There was a significant difference in the corneal thickness of Grades 0, 1, and 2 vs Grades 3, 4, and 5 ($t = 5.47$; $P < .001$) (Fig. 4).

Endothelial abnormalities, observed through the specular microscope, approximately paralleled the severity of disease

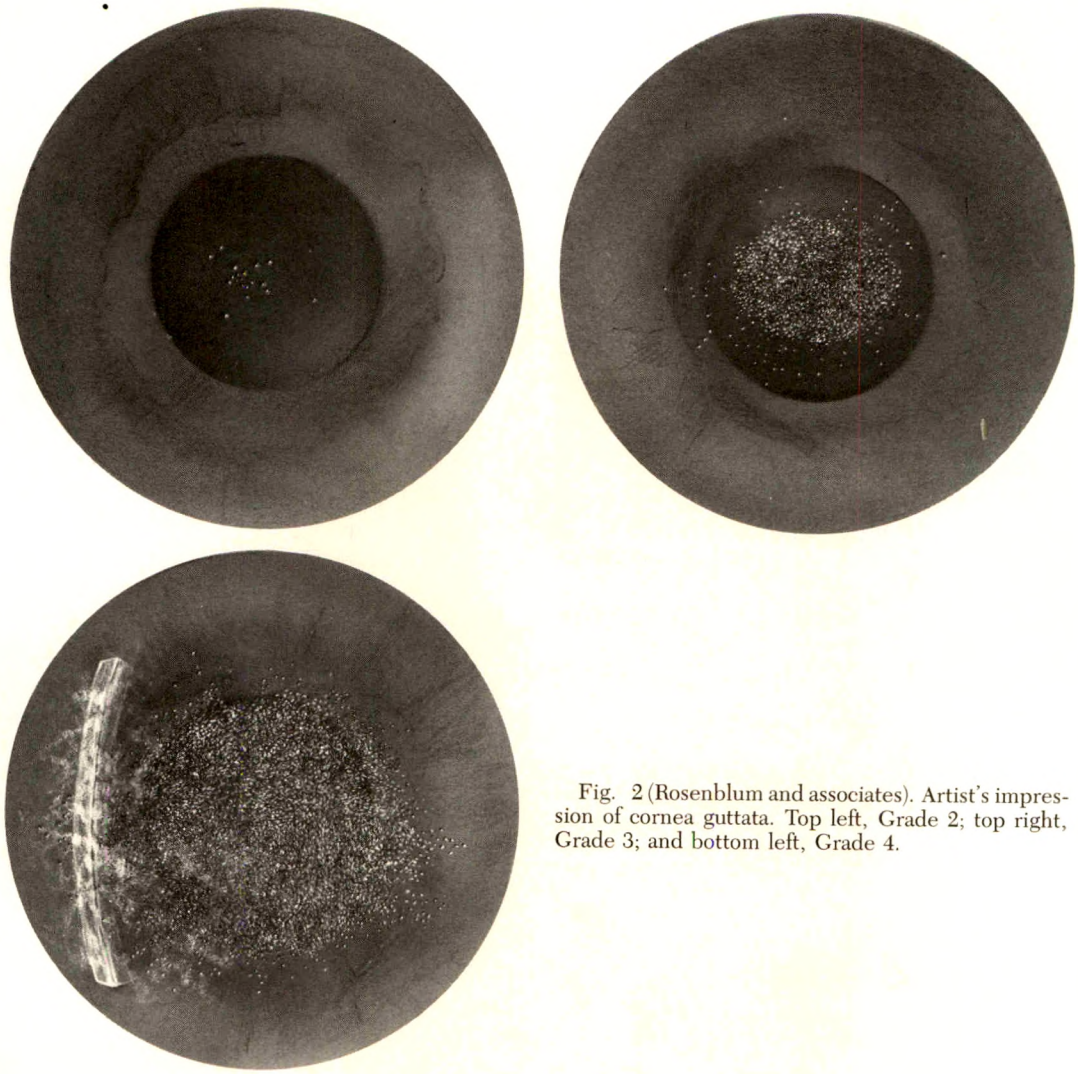


Fig. 2 (Rosenblum and associates). Artist's impression of cornea guttata. Top left, Grade 2; top right, Grade 3; and bottom left, Grade 4.

TABLE 2

DISTRIBUTION OF AFFECTED FIRST-DEGREE RELATIVES

	Parents		Siblings		Children ≥ 40 Years Old		Children < 40 Years Old	
Cornea Guttata	M	F	M	F	M	F	M	F
			Our Patients					
Grade 0	—	—	2	0	2	0	8	8
Grade 1	1	2	1	0	3	1	2	2
Grade 2	—	—	2	2	2	1	3	0
Grade 3	0	1	2	2	1	3	—	—
Grade 4	1	2	0	1	0	2	—	—
Grade 5	0	1	—	—	—	—	—	—
Total	2	6	7	5	8	7	13	10
			Examined Elsewhere					
Normal	—	—	1	1	—	—	1	2
Affected	—	—	7	5	1	1	—	—

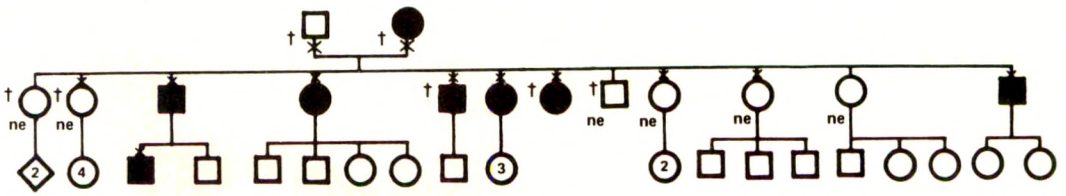


Fig. 3 (Rosenblum and associates). Pedigree of extensive family with Fuchs' corneal dystrophy. Note male-to-male transmission, only seen in autosomal-dominant inheritance. Open squares and circles, unaffected or not examined (ne) males and females; solid squares and circles, affected males and females. Dagger, deceased; diamond shape, sex not specified.

as judged by the degree of cornea guttata by slit-lamp examination. These endothelial cellular changes consisted of loss of normal hexagonal architecture, generalized cell enlargement, and increasing pleomorphism of individual cells (Fig. 5). These changes, however, were not uniform throughout the central cornea, but varied from one location to another. The lesions of cornea guttata appeared as dark shadows obscuring the cell outlines (Fig. 5). They were usually larger than individual cells and were never diagnosed by specular microscopy unless they "crossed" cell borders. In more severe cases, cornea guttata became confluent, and we noted large areas of cornea guttata with no visible cell outline (Fig. 5).

Most reports of Fuchs' dystrophy indicate that females are affected more often than males. There was a female-to-male ratio of approximately 7:1 among probands, but almost a 1:1 ratio among affected first-degree relatives over age 40. To determine whether sampling plays a role in this distribution, we reviewed all reports of penetrating keratoplasty done solely for phakic Fuchs' dystrophy in the records of the pathology laboratory here. Our assumption was that such surgery is done for visually disabling corneal edema and is not likely to be influenced by nonocular factors.

These records contained 79 cases of penetrating keratoplasties performed solely for phakic Fuchs' dystrophy be-

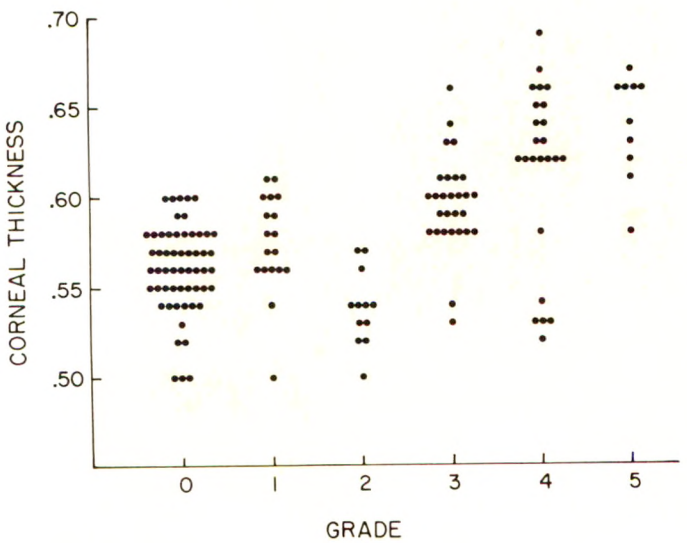


Fig. 4 (Rosenblum and associates). Scattergram of corneal thicknesses by pachymetry, vs grade of cornea guttata.

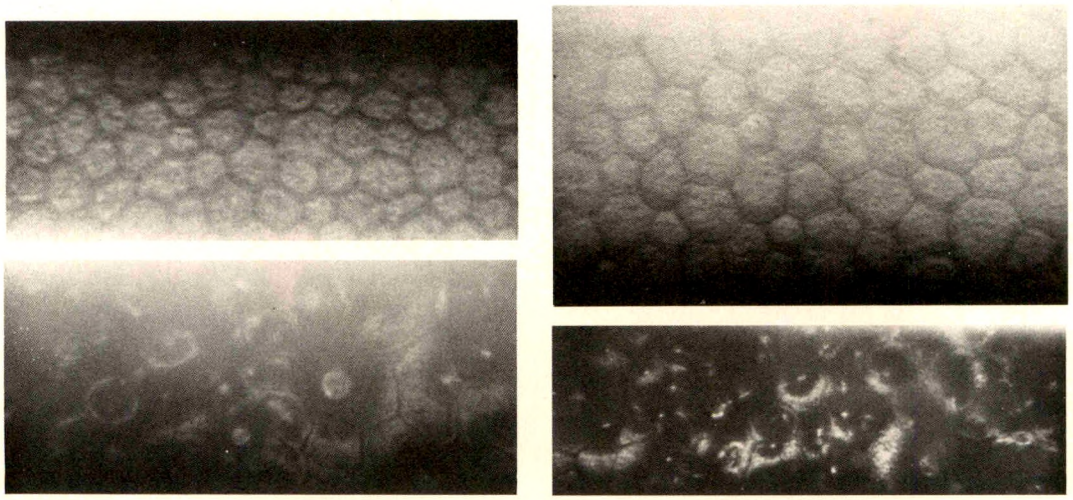


Fig. 5 (Rosenblum and associates). Top left, Normal specular micrograph of corneal endothelium. Top right, Cellular pleomorphism. Bottom left, Cornea guttata. Bottom right, Confluent cornea guttata.

tween 1940 and 1978. Sixty-one patients were female and 18 male, which is slightly more than a 3:1 ratio. The average age at the time of first corneal surgery was 65.3 years and did not differ significantly between the sexes.

Discussion

Fuchs' corneal dystrophy is a common disorder, yet documentation of familial occurrence is rare. In our study, the pattern of distribution of affected members in families is quite consistent with the theoretically expected distribution based on an autosomal-dominant pattern of inheritance (Table 3). Fuchs' dystrophy is only rarely sporadic. Failure to observe this strong familial pattern in the past may be a result of several factors. Ophthalmologists must look for the mild degree of dystrophy with a slit lamp or it will escape detection. Also, cornea guttata seldom becomes visible until the affected individual reaches middle age and

TABLE 3

Expected vs observed number of families with affected first-degree relatives*

Proband's First-Degree Relatives	No. of Families	Families With > 1 Affected Member	
		Expected No.	Observed No.
0	2	0	0
1	11	5.5	9
2	4	3	3
3	4	3.5	3
4	2	1.87	2
5	1	0.97	1
6	0	0	0
7	1	0.99	1
Total	25	15.83	19

*Assuming an autosomal-dominant mode of inheritance.

it remains asymptomatic until advanced age. Thus, affected members in previous generations may often have been unrecognized and be deceased by the time their children become symptomatic.

If cornea guttata is transmitted by autosomal-dominant inheritance, one half of the children of probands should be affected and one half should be normal. When we examined the children of probands, 32% (14 of 43) were affected. In children less than 40 years of age, only 12% (three of 26) had cornea guttata, whereas 65% (11 to 17) of those over 40 years of age were affected. The ascertainment probability (that is, the number of people examined, divided by the total number of people at risk under the given hypothesis) for this group was .531. For all first-degree relatives aged 40 years and over and at risk under the hypothesis of autosomal-dominant inheritance, 72% (37 of 51) were affected. There was no significant difference in age between affected males and females ($P > .5$). The mean age was 60.7 years (range, 41 to 91 years). The sex ratio was 1:1 (26 males:25 females).

The excess of affected patients in our series, above the expected 50% in a dominantly inherited disorder, can probably be attributed to a bias in recruiting relatives who were affected. Our ascertainment rate, in fact, reveals that we examined 56.5% (26 of 46) of all living first-degree male relatives over 40 years old and 59.5% (25 of 42) of all living first-degree female relatives over 40 years old. Since probands were largely responsible for recruiting their own relatives, we cannot assume that the sample was unbiased.

Many investigators have found a marked excess of females, even in the earliest published reports of Fuchs' dystrophy.[2,4,5,8] We also noted this predominance of affected females among our probands and in patients who needed penetrating keratoplasties for corneal dystrophy. However, we did not observe this in the offspring of affected parents, among whom the male-to-female ratio was 11:15. Our observed male-to-male transmission excludes X-chromosome-linked inheritance for two pedigrees. The remaining pedigrees are compatible with, though not conclusive for, autosomal-dominant inheritance. Therefore, an autosomal-dominant mode of inheritance is likely.

We cannot exclude the possibility that there is a sex influence on severity of disease that results in the predominance of women in most series. Similarly, the 3:1 female-to-male ratio we found for patients who underwent corneal surgery measures disease severity rather than disease presence. Increased severity in females, in fact, is certainly consistent with our data (Table 2). Thus, Fuchs' dystrophy, although inherited in an autosomal-dominant fashion, appears to be sex-dependent in its expressivity. In other words, the gene is distributed equally, but its phenotypic expression is more severe in females.

At the outset of our study, we hoped that specular microscopy might demonstrate endothelial changes in Fuchs' dystrophy that could be detected before the development of cornea guttata. Although the cellular pleomorphism and enlargement, along with cornea guttata, form a fairly typical picture in Fuchs' dystrophy, we were unable to detect any changes in cases not diagnosed by slit lamp. Part of the problem may be with the sampling difficulties of specular microscopy. Early cases of Fuchs' dystrophy show only a small area of endothelial morphologic abnormality by slit-lamp examination. Early cases do not appear to have widespread endothelial changes that can be detected with the specular microscope. Unless the ophthalmologist samples the specific region shown to be affected by

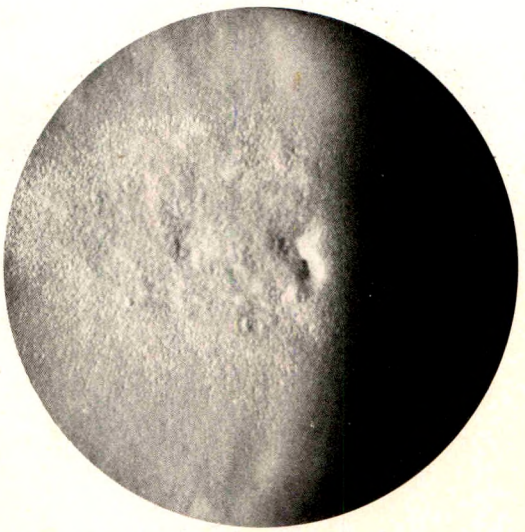


Fig. 6 (Rosenblum and associates). Pattern of Grade 4 cornea guttata. Note that the lesions extend more horizontally than vertically.

slit lamp, he may not note endothelial abnormalities with the specular microscope.

The diagnosis of Fuchs' dystrophy depends on distinguishing cornea guttata in the central cornea from Hassel-Henle warts in the periphery, which are apparently a normal aging change.[9] We were impressed by the strikingly typical manner in which the lesions of cornea guttata occur. They always begin centrally or paracentrally in a small area. As the concentration of lesions and the extent of their distribution increase, this affected region enlarges horizontally more than vertically (Fig. 6). We cannot explain this finding; it may be an effect of aqueous humor circulation, an intrinsic difference between endothelial cells in different areas of the cornea, or an effect of ultraviolet radiation upon endothelial cells in the exposed area of the interpalpebral fissure.

Our findings enable us to draw several conclusions regarding Fuchs' dystrophy. The endothelial dysfunction, clinically manifested as cornea guttata, is a true corneal dystrophy in the large majority of these cases with autosomal-dominant inheritance, a high degree of penetrance, and variable expressivity. There is a sex influence on the course of the disorder, with females being more severely affected. The specular microscope does not detect the endothelial defect earlier than careful slit-lamp examination.

Summary

We studied 102 individuals from 25 families in which at least one member was known to have Fuchs' endothelial dystrophy. We used slit-lamp examination, corneal pachymetry, and specular microscopy. We also examined pathology records which contained 79 cases of penetrating keratoplasties done solely for phakic Fuchs' dystrophy between 1940 and 1978.

Although there was a marked predominance of females in our review of pathology records, our clinical study provided a more even sex distribution. There was a close correlation between the number of expected and observed affected first-degree relatives based on an autosomal-dominant mode of inheritance. We observed a fairly typical corneal pattern, beginning as a circumscribed area of central or paracentral cornea guttata, and thereafter expanding more horizontally than vertically with progression of severity.

Fuchs' dystrophy appears to be a true corneal dystrophy with autosomal-dominant inheritance, a high degree of penetrance, and variable expressivity, with generally increased severity among females.

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Ophthalmic miniature

Near Roholp, close to Strangford Lough, the site of Patrick's landing place in Antrim, are the Wells of Struell, the Drinking well and the Eye well, and here the sailors of St. Patrick replenished the ship's butts.

The waters of the Eye Well were no longer for drinking, but to help the blind once more to see. Legend and faith had endowed the spring with curative properties for diseases of the eye.

Paul Gallico, *The Steadfast Man*
Garden City, New York, Doubleday, 1968

SIMILAR EPITHELIAL HEALING RATES OF THE CORNEAS OF DIABETIC AND NONDIABETIC PATIENTS

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A significant number of problems related to the corneal epithelium are found in diabetes mellitus. Of these, slowly healing abrasions with a propensity for recurrent erosions, a decrease in corneal sensitivity with the occurrence of neurotrophic ulcers, and epithelial healing problems after pars plana vitrectomy are clinically important.[1-6] Brightbill, Myers, and Bresnick[3] found a 60% rate of corneal epithelial clouding at surgery in a group composed primarily of diabetics. Foulks and associates[5] found that 45% of corneas in diabetic patients developed stromal or epithelial edema after vitreoretinal surgery, while only 10% of corneas from nondiabetic patients did.

Between 19% and 45% of corneal problems after vitrectomy involve the corneal epithelium.[3-5] Superficial punctate keratopathy is common and filamentary keratitis less frequent, although neither has great clinical significance. The nonhealing corneal epithelial defect and recurrent erosion, however, are the major concerns in postoperative diabetic patients. A trophic defect can persist for months, requiring repeated patching or a bandage contact lens, topically applied medications, and frequent examinations. Such a defect in the epithelium can lead to an infected corneal ulcer and subsequent scarring.

The cause of such epithelial abnormalities is unknown. Two mechanisms, not mutually exclusive, have been proposed in diabetes mellitus. First, abnormal adherence of the epithelium at the level of the basement membrane may be involved. This theory is supported by the demonstration of structural alterations in the basement membrane complexes of diabetic patients.[7] A second mechanism may be a slowed rate of sliding or mitosis, or both, in epithelial healing. Studies of diabetic rats have demonstrated a relationship between biochemical abnormalities and delayed epithelial healing.[8-14]

Although a similarly slowed rate of corneal epithelial regeneration might be proposed for the human cornea, this has never been measured in humans. We therefore developed a reliable way of measuring corneal regeneration in humans by using commonly available photographic methods. Applying this method prospectively to patients who had undergone surgical removal of their corneal epithelium, we compared the rates of primary corneal regeneration for diabetic and nondiabetic patients.

SUBJECTS AND METHODS

We included 27 patients in this prospective study (Table). We selected the patients consecutively from two groups. The first group included patients with retinal detachments and those who had undergone a pars plana vitrectomy with or without a scleral buckling procedure.

From the Departments of Cornea Research (Drs. Snip and Thoft) and Retina Research (Dr. Tolentino), Eye Research Institute of the Retina Foundation; and the Cornea Service, Massachusetts Eye and Ear Infirmary (Drs. Snip and Thoft), Boston, Massachusetts. Dr. Snip was a Heed Foundation Fellow (1978-1979).

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TABLE

SUMMARY OF CLINICAL DATA

Clincal Data	Patients	
	Diabetics	Nondiabetics
No.	14	13
Age in years		
Mean	50	51
Range	19 to 74	21 to 74
Surgical procedure		
Penetrating keratoplasty	2	7
Scleral buckling	0	5
Vitrectomy	3	1
Scleral buckling and vitrectomy	9	0

If a corneal epithelial scraping was necessary during surgery in order to see retinal details, the surgeon (F.I.T.) gently debrided the epithelium with a knife blade. He carefully left a 1- or 2-mm margin of corneal epithelium at the corneoscleral limbus to serve as a source of regeneration in the postsurgical period.

The second group of patients had undergone corneal transplantation surgery. The donor corneal epithelium was routinely removed by gentle knife debridement before trephining of the donor graft button. This resulted in a reproducible epithelial defect (for example, 50.3-mm^2 defect for an 8.0-mm donor button); a 1- or 2-mm margin of host corneal epithelium always remained to serve as a source of regeneration.

We included no patients with preoperative corneal epithelial problems. During the postoperative period, the medical management of the eyes in both groups was similar: they all received topically applied chloroamphenicol and dexamethasone. We covered all the eyes with light patches and did not use bandage soft contact lenses.

Photographic analysis—We took daily photographs of each patient beginning on the first postoperative day. We obtained several photographs at each sitting with the Zeiss slit-lamp camera with cobalt blue filter. After topical instillation of fluorescein, we took three to five photographs. Photographs taken promptly after the instillation of fluorescein ensured a sharp outline of the defects, and one of us (R.C.S.) confirmed the size of the defect with a slit-lamp examination and made a rough drawing of it at each session. The patients were seen daily while they were in the hospital. If an epithelial defect was present at the time of their scheduled discharge, they were seen again and photographed at their first postoperative visit to the surgeon. We included in this study only patients for whom two or more photographic sessions were available. We took all the photographs at approximately the same time each day (Figure).

The color slides were magnified ten times on a photographic enlarger. We included a millimeter ruler in each photograph to serve as a guide to accurate magnification. We then made pencil tracings of the projected defects on plain white paper, and measured the area encompassed by these irregularly outlined figures with a polar planimeter. This gave a daily measurement of the defects in square millimeters, which we used to calculate daily and average rates of corneal epithelium defect closure. Subsequent analysis of the several read-

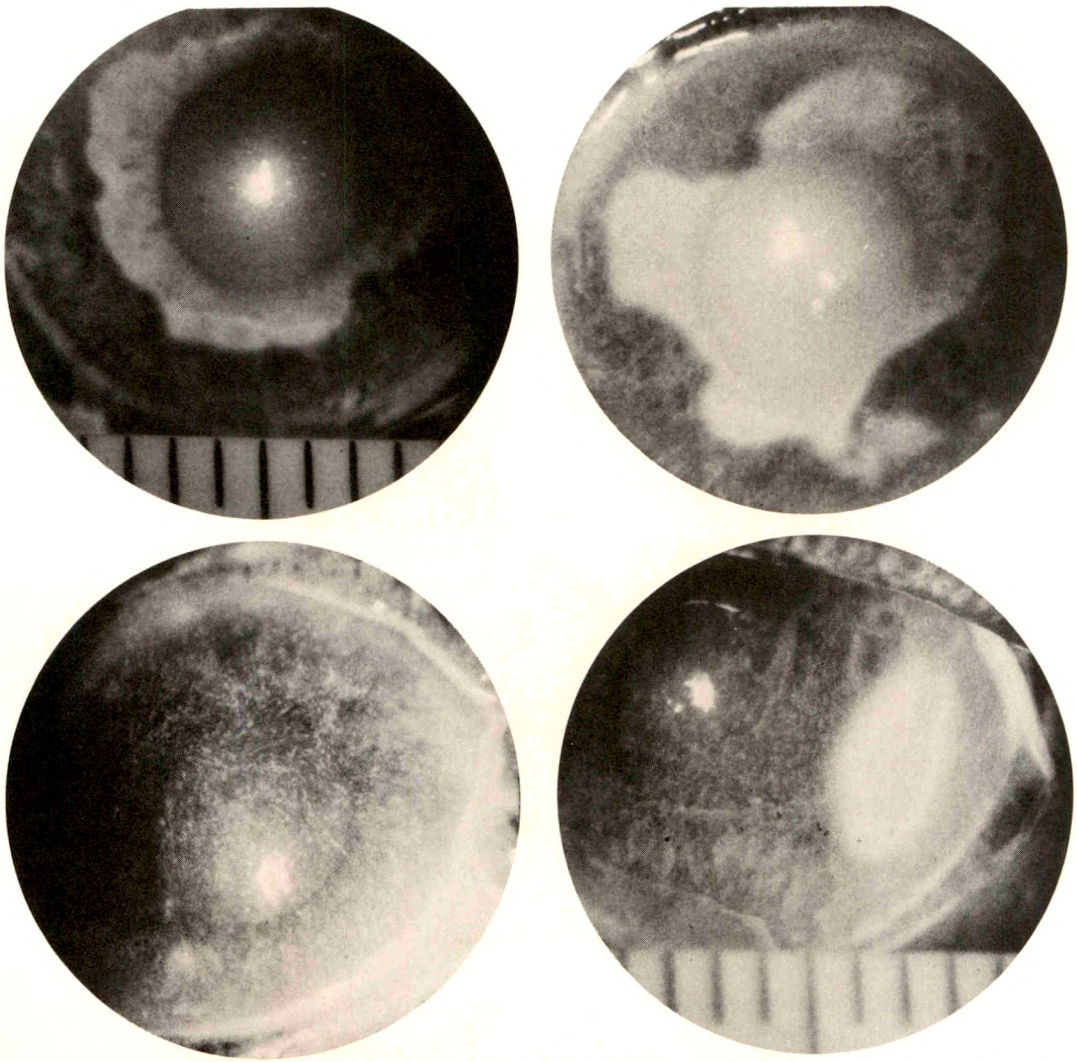


Figure (Snip, Thoft, and Tolentino). Top left, Day 2 (first postoperative day). Fluorescein staining shows the central epithelial defect in a diabetic patient after vitrectomy and scleral buckling. The defect measures 52.7 mm^2. Top right, Day 3. Epithelial defect measures 39.9 mm^2. Bottom left, Day 6. Epithelial defect is completely closed. There is diffuse, irregular surface staining with fluorescein, but the epithelium is intact. Bottom right, Day 15. Fluorescein highlights the epithelial defect. The outline of the defect is blurred and difficult to measure accurately because of a delay in taking the photograph after instillation of fluorescein.

ings taken each day showed good reproducibility for the size of defect calculated on any given day.

Results

Our 27 subjects included 14 diabetic and 13 nondiabetic patients. Their ages were comparable (Table) but the patients were not matched by disease or type of surgical procedure. Thus there were, for example, more patients with corneal transplants in the nondiabetic group and more patients with scleral buckling plus vitrectomy in the diabetic group. However, subsequent analysis of the surgical groups showed that they were similar

from the standpoint of corneal epithelial regeneration, and so we treated them as one group when comparing diabetic to nondiabetic patients.

The 14 diabetic patients in our study had had diabetes for an average of 22 years (range, six to 37 years); all were using insulin (12 by injection and two orally). During the observation period, we carefully monitored every diabetic patient for blood glucose levels and insulin dosages. There were no cases of overt hyperglycemia or hypoglycemia, and no patients had ketosis or acidosis at any time.

The diabetic group healed at an average rate of 18.7 mm^2/day ($\pm$7.8 mm^2/day). The nondiabetic group healed at an average rate of 13.6 mm^2/day ($\pm$5.0 mm^2/day). Subsequent statistical testing showed that these two rates were not significantly different ($P = .05$).

The healing rate for individual patients was linear. In our series, the linear regression co-efficients ranged from 0.78 to 1.00, with an average of 0.95. As the individual healing rate for any patient was similar from day to day, daily measurement may not be necessary. In each of our groups, there were examples of rapid and slow healing, but each individual patient had a consistent linear rate of healing.

Discussion

This study was, to our knowledge, the first to document epithelial healing rates in the human cornea. The method we used was simple, and the photographic documentation provided reproducible data that were subjected to statistical testing and review. This method may be useful clinically for studying the effects of various treatments in the presence of a corneal epithelial defect (for example, the use of aminoglycoside antibiotics). It is well suited for both human and experimental animal studies.[15]

Statistical testing of our data showed no significant difference in the rates of healing of epithelial defects when diabetic patients were compared with nondiabetic patients. The rates we measured were linear and comparable to rates that have been found in the nondiabetic rabbit.[15,16]

It would be misleading, however, to imply that patients with diabetes mellitus heal normally. Several studies have documented corneal epithelial problems in diabetic humans,[1,3,5-7,17] including differences in the healing of abrasions, a tendency toward recurrent erosions, the development of neurotrophic corneal ulcers, and problems in healing after vitrectomy. Diabetic epithelium is known to be vulnerable during surgery, showing earlier clouding and poorer adhesion to the underlying stroma.[6]

This vulnerability does not extend to changes in healing rate, however. The rates of initial sliding and mitosis of the corneal epithelium to cover a surgically created defect are similar for hospitalized patients with controlled diabetes and nondiabetic patients. We did not compare diabetic patients to normal subjects since our nondiabetic patients had undergone major intraocular surgery, but our two groups did undergo comparable surgical stress.

Although a decreased healing rate has been observed in diabetic rats,[8] one must remember that those experiments compared diabetic and normal rats. Also, we should not underestimate the differences at the metabolic level between grossly hyperglycemic rats and carefully monitored, hospitalized humans with controlled diabetes. Since our diabetic patients never had overt hyperglycemia or hypoglycemia, a biochemical effect that depends upon increased levels of intracellular glucose was difficult to demonstrate.

We concluded that abnormal adhesion plays a significant role in some of the

postsurgical abrasion problems in diabetic patients. Although we did not follow up our patients longitudinally to redocument previously published data,[3-5] we did find two patients with previously healed epithelial defects that had broken down. Both of these patients were diabetic; one case occurred after vitreous surgery (Figure) and the other after a corneal transplant. Both patients suffered the breakdown in the early postoperative period, and needed bandage soft contact lenses to heal the defects.

Previous studies have shown that normally regenerating corneal epithelium uses the basement membrane remaining after scraping.[18] Since one of the breakdowns occurred over the donor button of a corneal transplant patient, diabetic patients may fail to utilize the basement membrane and basal cell adhesion complexes that we presume to exist on the donor button. The failure to adhere after regrowth may also reflect primary metabolic abnormalities in the corneal epithelium of diabetic patients. The accumulation of sorbitol has been demonstrated in the corneal epithelium of both diabetic experimental animals and diabetic humans.[5] While the subcellular abnormalities may not be reflected in the initial epithelial coverage rates in our patients, sorbitol accumulation may be only one sign of the epithelial cell biochemical abnormalities caused by diabetes mellitus. Abnormal adherence may be another manifestation of biochemical abnormalities in the synthesis of basement membrane and adhesion complexes, reflected by more frequent recurrent erosions in diabetic patients.

Summary

We developed a reliable method for measuring corneal regeneration in humans with noninvasive, readily available photographic techniques. We then used this method prospectively to compare the rates of primary corneal regeneration after surgery in a series of 27 diabetic and nondiabetic patients.

The rates of corneal epithelial regeneration were not significantly different when diabetic patients were compared with nondiabetic patients. A tendency toward recurrent epithelial defects was confirmed in the diabetic patients, however.

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Ophthalmic miniature

A friend of mine had a servant afflicted with bad eyes. He lost his eyelashes, and his eyes were surrounded with a vermillion disk. They were very disgusting, and a friend advised him to take Grimstone's eye snuff. In three weeks they are as sound as possible. The disorder had lasted some years. Is the fact of any use to you?

Robert Hull, Cursory Notes on *The Morbid Eye*, London, Longman, Orme, Brown, Green and Longman, 1840

ULTRASTRUCTURE AND SUCCESSFUL KERATOPLASTY OF SCLEROCORNEA IN MIETENS' SYNDROME

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AND

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In 1966, Mietens and Weber[1] described a group of anomalies in four of six siblings whose parents were second cousins and apparently healthy; these were growth failure, flexion contracture of the elbows, dislocation of the head of the radii, abnormally short ulnae and radii, mental retardation, bilateral corneal opacities, horizontal and rotational nystagmus, strabismus, and a small pointed nose with a depressed root. The corneas had a diffuse disk-like central opacity localized mainly in the superficial layers. In one case, the corneal haze occupied the pre-Descemet's stroma. There were no chromosomal abnormalities in the leukocytes. The nature of the corneal opacity has not been previously established, and we report here the clinical and histopathologic findings of sclerocornea.

CASE REPORT

A 1,984-g Korean boy was born at term May 7, 1971, to an Rh positive, gravida 1, para 0 mother. The parents were not related. During pregnancy, the mother had no complications, such as infection, fever, toxemia, excessive weight gain, exposure to rubella, or ingestion of unusual medications. One minute after the spontaneous vaginal occipito-anterior delivery without anesthesia, the Apgar score was 9.

The following constellation of abnormalities, noted at birth and confirmed during the neonatal period by clinical and radiographic examination, represented a variant of Mietens' syndrome: bilateral corneal opacities, hypoplastic nose with absence of nasal cartilage, bilaterally absent radii with radial deviation of both hands and flexed thumbs, elbow flexion contractures, and absent left fibula with left club foot.

Results of an intravenous pyelogram and serum IgM test were normal. There were no cytomegalic inclusion bodies in the urine and no intracranial calcifications appeared on skull X-ray. The karyotype from the peripheral blood when the patient was 1 and 2 years old showed normal 46 XY chromosomes. Maternal chromosomes were also normal.

There were no signs of mental retardation and his rate of growth and development were normal, although he remained short for his age and wore casts on both arms and the left leg until the age of 2 years. The parents placed him in a foster home. Psychological and pedagogical tests showed his intelligence to be normal and he developed into a personable, bright child (Fig. 1).

When he was 5 days of age, ophthalmic examination showed bilateral diffuse corneal opacities, denser on the left, with formed anterior chambers, round pupils, no dense cataracts, and normal intraocular pressures by palpation. Annual examinations showed little change in the corneal opacities.

At 5 years of age, he could count fingers at 4 feet with the right eye and see hand motions at 1 foot with the left eye. He fixed with his right eye and had an intermittent searching nystagmus and a left esotropia of about 75 prism diopters by Hirschberg's corneal reflex test.

We examined him while he was under halothane anesthesia. Horizontal corneal diameters were 9.25 mm bilaterally. Both corneas showed diffuse opacification and superficial vascularization with an irregular keratoscope reflection, a clearer periphery, normal thickness by inspection, and no focal posterior corneal defect or iridocorneal adhesions. In the right cornea (Fig. 2, left), nebular anterior stromal opacities formed a ring within a diffuse anterior stromal gray haze. A dense network of subepithelial vessels extended across the nasal one third of the cornea. Most of the posterior stroma was clear. The left cornea (Fig. 2, right) had a moderately dense, central, uniform, white vascularized opacity that occupied the anterior one fourth of the stroma.

From the Corneal Service, Department of Ophthalmology, University of California School of Medicine, Davis, California (Dr. Waring); and Section of Clinical Eye Pathology, Clinical Branch, National Eye Institute, Bethesda, Maryland (Dr. Rodrigues).

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Fig. 1 (Waring and Rodrigues). A 5-year-old boy with Mietens' syndrome has bilateral microsclerocornea with left esotropia, hypoplastic nose, low-set ears, abnormally shaped arms because of absent radii, flexion contractures of elbows, and extreme flexion of thumbs.

Iris architecture appeared normal where the iris could be seen. The lenses were clear, except for a small right anterior cortical cataract. Indirect ophthalmoscopy disclosed bilateral generalized thinning of the retinal pigment epithelium, normal maculas, and superionasal origin of the right optic disk vessels. Intraocular pressure by Schiøtz tonometry and Perkins hand-held applanation tonometry was approximately 12 mm Hg bilaterally.

Our clinical ophthalmic diagnosis was bilateral microsclerocornea with mild right anterior cortical cataract.

On June 16, 1976, we performed a 6.0-mm penetrating keratoplasty and two peripheral iridectomies in the left eye, using a cornea stored in tissue culture (McCarey-Kaufman medium) from an 18-year-old donor. A scleral ring supported the globe while we secured the graft with four interrupted and one running 10-0 nylon suture. On the fifth postoperative day, retinoscopy performed while the child was under general anesthesia disclosed approximate emmetropia.

Postoperatively, he received two subconjunctival injections of triamcinolone acetonide. We prescribed tapering doses of topically applied atropine, a mixture of neomycin, gramicidin, and polymyxin B, and prednisolone acetate. Three to five weeks postoperatively, we removed the sutures in stages because of early graft vascularization. Mackay-Marg tonometry was 14 mm Hg. The center of the graft was clear and compact.

Beginning one month after surgery, the right eye was occluded full-time during waking hours while the child wore spectacles, −2.25 diopters, for the left eye; by eight months after surgery he could see the isolated "E" with the left eye at 1 foot. The intermittent searching nystagmus and left esotropia persisted, but his visual function improved, since he walked with more certainty and perceived smaller objects.

• He rejoined his mother and moved overseas in April 1978.

Materials and Methods

Immediately after removal, the host corneal button was placed epithelial side down on a silicone block, bisected, and one half fixed in Baker's calcium formol solution for light microscopy with the following stains: hematoxylin and eosin, PAS, Masson's trichrome, colloidal iron, and Alcian blue at pH 2.0 and pH 1.4. The remaining one half was trisected, fixed in cold modified Karnovsky's solution and buffered 1% osmium tetroxide, dehydrated through ascending concentrations of ethyl alcohol, embedded, stained with toluidine blue followed by Reynold's lead citrate, and studied by transmission electron microscopy.

Results

Epithelium—The epithelium was five or six layers thick and demonstrated a normal maturation pattern from a single row of basal cells through three layers of polygonal cells to two layers of squamous cells. The basement membrane was irregular (Fig. 3). Ultrastructurally, the epithelial cells displayed normal structure and intercellular attachments. The basal lamina was fragmented and consisted of many layers of fine, granular amorphous material with irregularly spaced hemides-

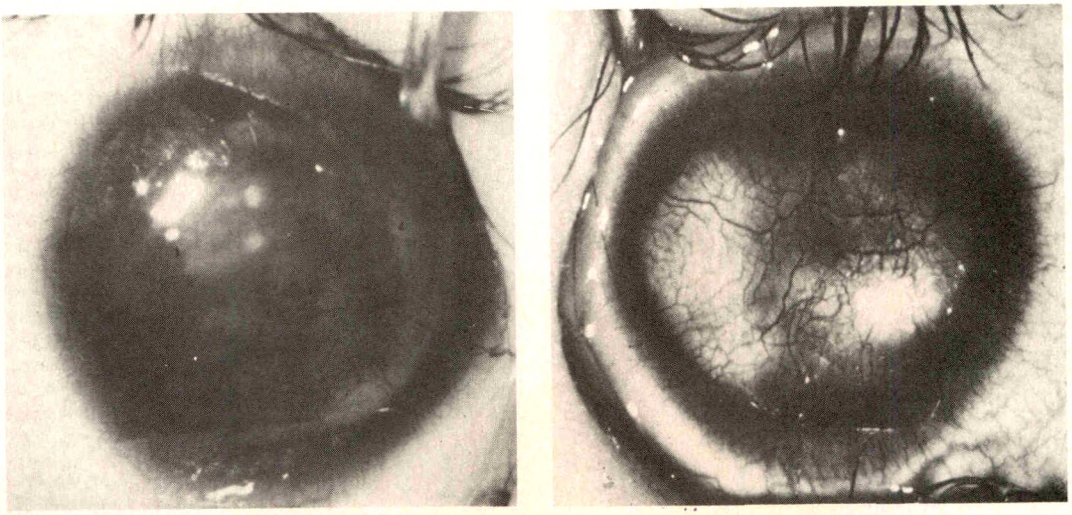


Fig. 2 (Waring and Rodrigues). Sclerocornea in Mietens' syndrome. Left, Right cornea shows superficial vascularization nasally that extends to center of cornea. Anterior stromal opacification consists of diffuse scarring with focal nebular accentuation. Surface is slightly irregular. Right, Left cornea shows total superficial vascularization and scarring, with periphery slightly clearer than center and with opacity more dense temporally than nasally. Epithelial surface is irregular.

mosomal attachment complexes (Fig. 4). Laminations of basement membrane-like material mingled with the superficial collagen fibrils.

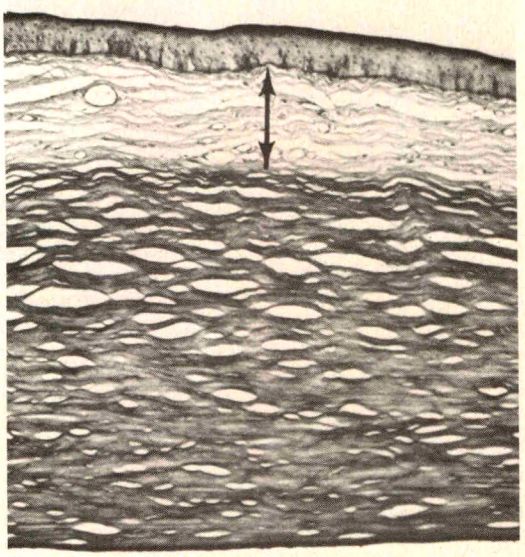


Fig. 3 (Waring and Rodrigues). Vascularized collagenous tissue occupies anterior one fourth of stroma (arrow). Bowman's layer is absent. The endothelium was traumatically removed during surgery (PAS, × 107).

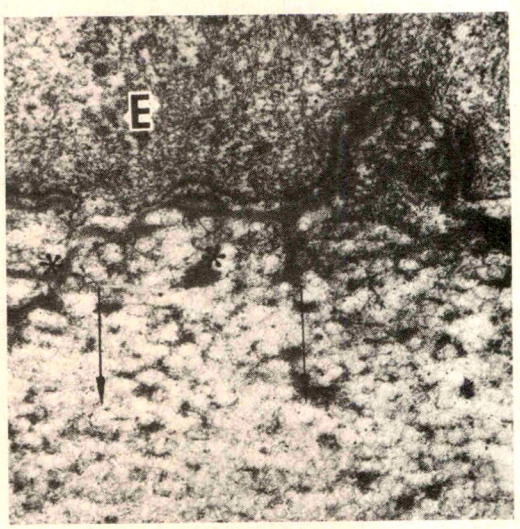


Fig. 4 (Waring and Rodrigues). The subepithelial area shows abnormally large collagen fibrils (arrows) that measure 70 to 90 nm in diameter. The basal epithelial plasma membrane is irregular and there are multiple thin lamellae of basement membrane (asterisk); E, epithelium (× 35,100).

Stroma—Bowman's layer was absent throughout (Fig. 4). A layer of vascularized connective tissue occupied the anterior one fourth of the stroma from corneoscleral limbus to corneoscleral limbus and mixed with the epithelial basal lamina (Fig. 3). The transition between the anterior vascularized connective tissue and the posterior lamellar stroma was gradual. We saw many cells throughout the tissue; most looked like fibroblasts and some were mast cells.

Ultrastructurally, the collagen fibrils were arranged in random bundles and measured from 75 to 90 nm in diameter (Fig. 4) with a periodicity of 50 to 70 nm. Mildly electron-dense, amorphous material surrounded the fibrils. The fibroblasts were enmeshed among the collagen fibrils and showed dilated rough endoplasmic reticulum with the appearance of vacuoles. The mast cells exhibited their scroll-like structures in the cytoplasm.

The posterior three fourths of the stroma showed a lamellar pattern characteristic of normal cornea. Ultrastructurally, the collagen fibrils were 40 to 50 nm in diameter with a periodicity of 58 nm. Morphologically normal keratocytes were present and we saw no abnormal cells.

Descemet's membrane and endothelium—Descemet's membrane was about 6 μ thick. It was homogenous by PAS staining. Ultrastructurally, the anterior one fourth of Descemet's membrane was abnormal (Fig. 5). It had a loose, slightly lamellar pattern that interdigitated with the posterior stromal collagen fibrils. A compact zone with macroperiodicity of about 110 nm occupied the central one half of Descemet's membrane. The posterior one fourth was homogeneous banding without excrescences.

The corneal endothelium was partly absent and where present exhibited autolytic changes with scattered nuclei, mitochondria, free ribosomes, and smooth endoplasmic reticulum.

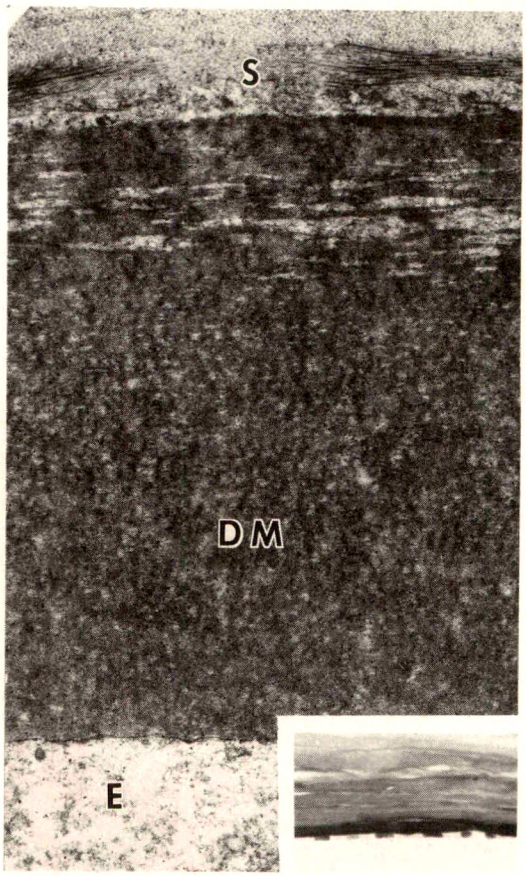


Fig. 5 (Waring and Rodrigues). Inset, Corneal endothelium and Descemet's membrane are intact (PAS, × 120). Descemet's membrane (DM) shows abnormal anterior portion that consists of fine irregular lamellae. Mid-portion shows banding with approximately 110-nm macroperiodicity. Posterior portion becomes more homogeneous. Endothelial cell (E) shows autolytic changes, presumably from fixation; S, stroma (× 27,000).

Discussion

Clinicians often use the term sclerocornea as a nonspecific description for any congenitally opaque, vascularized cornea. Using the term too broadly, however, obscures useful distinctions. Clinically, sclerocornea denotes a cornea with a congenital, peripheral white vascularized opacity that blends with the sclera, obliterates the corneoscleral limbus and scleral sulcus, and leaves the

central cornea somewhat clearer than the periphery.[2] Histopathologically, sclerocornea contains large-diameter collagen fibrils. The lack of inflammation helps distinguish it from postinflammatory scarring.[3,4] (Fig. 6). The congenital corneal abnormality in our patient with Mietens' syndrome does not fit this definition exactly, since the opacity did not obliterate the corneoscleral limbus and since the collagen fibrils were intermediate in diameter (80 nm), between normal cornea (25 to 40 nm) and sclera (200 to 1,500 nm).[5] Thus, sclerocornea is not a discrete diagnostic entity. Howard and Abrahms[6] emphasized this when they reported a variety of associated ocular abnormalities: flattened cornea (cornea plana) in about 77%, shallow anterior chamber in about 39%, iris abnormalities in about 60%, and microphthalmos in about 8%.

We classified sclerocornea into four groups but realize the distinction among them is imprecise: (1) Isolated sclerocornea. Patients in this group have no other ocular abnormalities and show either exaggerated scleral extension across the superior and inferior corneoscleral limbus (scleralization, scleral overriding) or more extensive corneal opacification like the patient described here. (2) Sclerocor-

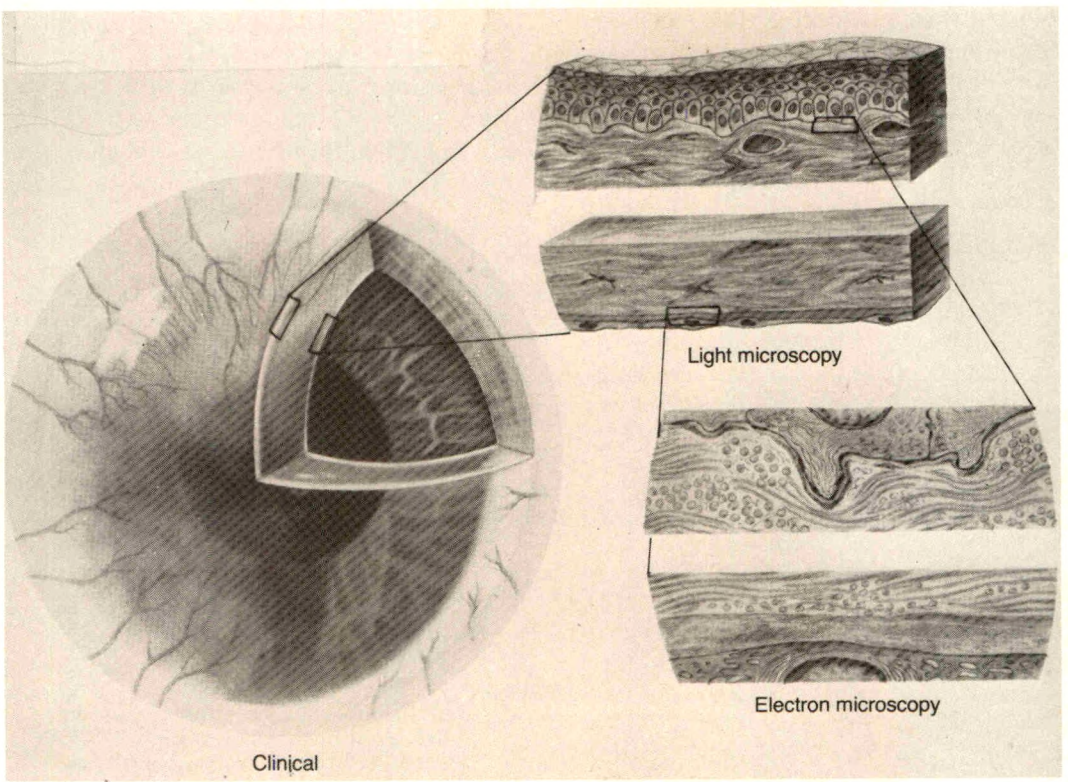


Fig. 6 (Waring and Rodrigues). Composite drawing of sclerocornea emphasizes major features. Clinical (clockwise), peripheral sclerocornea with sharply demarcated edge; mild sclerocornea with finger-like extensions to visual axis; severe full-thickness sclerocornea. Light microscopy, Bowman's layer absent; anterior stroma disorganized with large collagen fibrils and vascularization; posterior stroma less disorganized. Electron microscopy, basal epithelial cells send extensions into anterior stroma; basement membrane irregular; Bowman's layer absent; stroma with large-diameter, disorganized collagen fibrils; Descemet's membrane thin without normal anterior banded layer.

nea plana. Bilateral flat corneas with keratometry readings of less than 38 diopters occur as a sporadic or autosomal dominant anomaly with a shallow anterior chamber, iris abnormalities, and corneal diameters of 10 to 11 mm, on the small side of normal.[7] Our patient with Mietens' syndrome also had microcornea (9.25 mm in diameter), but there was no obvious abnormality in corneal curvature. (3) Anterior chamber cleavage anomalies. A scleralized peripheral cornea is present in 80% of eyes with Rieger's syndrome[8,9] and is common in Peters' anomaly. (4) Total sclerocornea. When the cornea is opaque enough to prohibit visualization of the iris and lens, a precise clinical diagnosis is difficult.[10]

No systemic abnormalities consistently accompany sclerocornea, but a variety have been reported[2,9,11,14-19] (Table).

Our patient resembled those described by Mietens, except for the absence of mental retardation, and we identified the corneal abnormality as microsclerocornea. We have found no cases in published reports other than the original family described by Mietens and Weber.[1]

Histopathologically, the sclerocornea shows an irregular epithelium with variably thick basement membrane, a fragmented or absent Bowman's layer, and disorganized spindles of vascularized stromal collagenous tissue that contains collagen fibrils 60 to 150 nm in diameter.[3] In our case, the architecture of the anterior scleralized cornea resembled sclera, but the collagen fibril diameter was smaller than sclera (Fig. 4). The random structure of these large fibrils and their attendant blood vessels scatters light and gives the cornea its white clinical appearance. Descemet's membrane is abnormal, either present as a thin irregular layer with collagenous tissue behind it[4] or showing focal dehiscences that may contain fibrous tissue.[11] In our case, the

TABLE

SYSTEMIC ABNORMALITIES REPORTED WITH SCLEROCORNEA

Study	Abnormalities
	SKELETAL
Goldstein and Cogan[2]; Bloch[9]	Polydactyly, skull malformations, abnormally shaped external ears, spina bifida, high arched palate, mandibular hypoplasia
	CENTRAL NERVOUS SYSTEM
Goldstein and Cogan[2]; March and Chalkley[14]	Cerebellar dysfunction, defective hearing
	CHROMOSOMAL
Rodrigues, Calhoun, & Weinreb[11]	17p 10q, unbalanced translocation
Ginsberg, Perrin, & Sueoka[15]; Cernea and associates[16]	Trisomy 18
	SYNDROMES
Harben and associates[17]	Smith-Lemli-Opitz (microcephaly, mental and growth retardation, abnormal genitalia, hand and foot anomalies)
Lapri[19]	Beimond's (hexadactyly of hands and feet, lacunae of cranial bones); Lohman's
Desvignes and associates[18]	Lobstein's (fragile bones, blue sclerae, decreased hearing)
Mietens and Weber[1]; present study	Mietens' (abnormal radii, flat nose, elbow flexion contractures, growth and, sometimes, mental retardation)

anterior Descemet's membrane had an abnormal lamellar pattern, but the posterior portions appeared normal, suggesting abnormal development before the fifth month of gestation. The endothelium is usually damaged, precluding detailed description.

Management of sclerocornea is similar to that of other congenital corneal opacities.[12] If the disorder is unilateral and the other eye has good visual acuity, we perform corneal surgery only if ocular structure is favorable. If the disorder is bilateral, we perform penetrating keratoplasty in the early months of life. Although the prognosis for a persistent clear graft is poor, long-term success is possible,[4,13] as our case illustrates.

Summary

A 5-year-old boy with Mietens' syndrome had bilateral microsclerocornea, hypoplastic nose, bilaterally absent radii, elbow flexion contractures, absent left fibula, growth retardation, and normal intelligence. Both corneas measured 9.25 mm in diameter and showed diffuse anterior stromal opacification with focal nebular densities and extensive superficial vascularization. Penetrating keratoplasty in one eye remained clear and compact two years postoperatively and visual acuity improved. Histopathologically, vascularized collagenous tissue occupied the anterior one fourth of the corneal stroma and contained bundles of collagen fibrils 75 to 90 nm in diameter. Descemet's membrane showed abnormal anterior lamination.

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A SURVEY OF *MORAXELLA* CORNEAL ULCERS IN A DERELICT POPULATION

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In the eye clinic population at Bellevue Hospital in New York City, infection with diplobacilli of the *Moraxella* group was the most common cause of bacterial corneal ulcer from 1965 through 1968. Nearly all those infected were derelict alcoholics living in the Bowery district.

Moraxella keratitis has always been rare in the United States[1] and controversy has surrounded the clinical appearance of ulcers caused by *M. lacunata*, *M. liquefaciens*, and *M. nonliquefaciens*. Petit[2] isolated *M. liquefaciens* only from corneal ulcers, while Fedukowicz and associates[3,4] found no difference in the incidence of hypopyon keratitis caused by *M. liquefaciens* and *M. lacunata*.

At the turn of the century, many investigators found the *Moraxella* diplobacillus to be the most commonly diagnosed cause of conjunctivitis.[5,6] More recently, the reported incidence of *Moraxella* conjunctivitis has ranged from 0.1% to 1.0% of all forms of conjunctivitis in the Western world.[7-9]

We attempted to correlate the clinical appearance of *Moraxella* keratitis with the subgroups previously mentioned, and examined those factors that made the Bowery alcoholic particularly susceptible to corneal infection.

SUBJECTS AND METHODS

We studied a series of patients with *Moraxella* corneal ulcers treated at Bellevue Hospital between 1966 and 1968. We recorded the position and severity of corneal ulceration, the degree of liquefaction of Loeffler's medium by the infecting organism, and the patient's alcohol consumption, nutritional status, and sanitary habits. We assessed the possibility that the Bowery, with its alcoholic population, crowded conditions, and poor sanitation, might harbor a reservoir of the *Moraxella* organism. We also obtained cultures from the eyelids, conjunctivae, and nasal cavities of 100 chronic alcoholics, without eye infections, from the Manhattan Bowery Project.

RESULTS

We studied 20 ulcers from 19 patients who ranged in age from 39 to 90 years. The average age was 51 years. Thirteen had an ulcer in the left eye, five in the right eye, and one had a bilateral infection. All but one of the patients admitted to alcohol abuse. Eleven called them-

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selves "heavy" drinkers, and four "moderate" (four to six drinks per day) drinkers. We noted that 14 had poor dietary habits and impaired nutrition. Fifteen had poor sanitary habits. Five said they slept outdoors frequently. Four had had recent trauma to the affected eye. No social history was available for one patient.

We correlated ulcer type and hypopyon with species of *Moraxella* (Table 1). We found no correlation when we combined *M. lacunata* and *M. liquefaciens* as one species and compared the clinical features of the ulcer to the organism's ability to liquify Loeffler's medium. Both species induced either aggressive central hypopyon ulcers or shallow ulcers without hypopyon.

We obtained antibiotic sensitivities of *Moraxella* organisms isolated from 12 ulcers. Included in this group were organisms from six *M. liquefaciens* ulcers, one *M. lacunata* ulcer, and five *M. nonliquefaciens* ulcers. All but one of the *Moraxella* isolates were sensitive to penicillin as well as to the other antibiotics tested. The resistant strain, an *M. liquefaciens*, was resistant to sulfa, streptomycin, and lincomycin.

We also compared the incidence of *Moraxella* keratitis to other causes of bacterial corneal ulcers at Bellevue Hospital for the ten-year period from 1965 to 1974 (Table 2). A dramatic decline in the incidence of *Moraxella* ulcers was evident. The trend began in 1969, but was most marked after 1970. *Moraxella* accounted for 46% of all ulcers seen at Bellevue from 1965 to 1970 but only 5% of the ulcers encountered from 1971 to 1974.

The results of the cultures of the eyelids, conjunctivae, and nasal cavities of the 100 Bowery alcoholics from the Manhattan Bowery Project are shown in Table 3. *Moraxella* was second only to *Staphylococcus* as the most frequently cultured organism.

Discussion

The eighth edition of Bergey's Manual[10] classes *Moraxella* with the Neisseriaceae. The five species are *M. lacunata* (including the subspecies *liquefaciens*), *M. nonliquefaciens*, *M. bovis*, *M. phenylpyruvica*, and *M. osloensis*. The clinically important species are *M. lacunata* and *M. nonliquefaciens*. A positive oxidase reaction distinguishes the Neisseriaceae from Acinetobacter (formerly Mimeae). This taxonomic classification is in accord with prior clinical findings that the severity of keratitis is unrelated to the degree of liquefaction of Loeffler's medium.[3,4] What we have noted in our series of cases is that ulcers produced by *M. nonliquefaciens* cannot be distinguished clinically from ulcers induced by *M. lacunata* or *M. lacunata*, subspecies *liquefaciens*.

TABLE 1

Clinical features of *Moraxella* corneal ulcers

Organism	Central	Peripheral	Deep	Superficial	Hypopyon	No Hypopyon	Total*
Moraxella lacunata		1		1	1		2
M. lacunata, subspecies *liquefaciens*	4	2	2	5	5	1	7
Moraxella nonliquefaciens	3	5	3	3	5	2	8
Mima *Moraxella*	2	1			2		3

*Data on certain cases were incomplete. Thus, the total number of cases caused by an organism might be larger than the sum of cases exhibiting a particular clinical feature (for example, central and peripheral ulcers).

TABLE 2

INCIDENCE OF BACTERIAL CORNEAL ULCERS AT BELLEVUE HOSPITAL, 1965-1974

Year	*Moraxella*	Others	Total	% *Moraxella*
1965	3	4	7	43
1966	7	7	14	50
1967	9	9	18	50
1968	5	3	8	63
1969	0	9	9	0
1970	2	1	3	67
1971	0	9	9	0
1972	2	8	10	20
1973	0	4	4	0
1974	0	6	6	0
Totals	28	60	88	32

Thus, it appears that the ability to liquefy Loeffler's medium does not imply that an organism will produce a more severe ulcer, although it may affect the organism's ability to overcome host resistance. *M. nonliquefaciens* belongs to the normal flora of the upper respiratory tract[11]; therefore, one might expect to see infection with *M. nonliquefaciens* more commonly than with *M. lacunata*, but this is not the case.[12] All species were sensitive to penicillin and most other antibiotics, confirming Henriksen's[11] findings for *M. nonliquefaciens*. It is not surprising that the clinical picture of a corneal ulcer is not pathognomonic for a particular organism. Nor is it surprising that a strain of low virulence elsewhere in the body can cause severe ocular disease.

When normal subjects are examined, *Moraxella* is found in 11.3% of nasal cultures.[11] Van Bijsterveld,[9] in a more recent study, found only a 0.3% incidence of *Moraxella* in the conjunctivae and eyelid margins of normal controls and a 12.9% incidence of *Moraxella* in nasal isolates. This compares to finding *Moraxella* in 22% of normal conjunctivae in 1921.[13] We found that 35% of Bowery alcoholics harbored *Moraxella* in their noses and we found the organism in the eyelids and conjunctivae in 5.5% of the eyes cultured. This colonization may be the source of corneal infection in hosts who have decreased resistance and who are subject to many episodes of minor trauma while on alcoholic binges. The greater frequency of corneal ulcers in the

TABLE 3

RESULTS OF CULTURES OF 100 ALCOHOLICS ON THE BOWERY, 1968

Organism	Eyelids		Conjunctivae		Nose
	Right	Left	Right	Left	
Moraxella	1	2	4	4	35
Staphylococcus	74	74	65	65	75
Hemophilus aegyptius	1	2	1	1	7
Streptococcus	0	0	0	0	3
Proteus	1	1	0	2	1
Other	3	3	2	2	3

left eye may be the result of violence in this derelict population since the left eye is opposite an adversary's right hand.

The decline in *Moraxella* infections of the eye in the latter half of the 20th century has been attributed to improved nutrition and, possibly, the absence of pyridoxine deficiency.[12] Van Bijsterveld found phagocytosis and overall bactericidal activity of polymorphonuclear leukocytes to be mildly but significantly reduced in pyridoxine-deficient guinea pigs.[14,15] Fourteen of our patients had poor nutritional status.

Alcohol intoxication may be another risk factor. Olsen and McCrae showed that alcoholics are more susceptible than the general population to pneumonia.[16] Alcohol increased susceptibility to experimental infections in animals,[17] and decreased the rate of leukocyte mobilization into traumatized skin of nutritionally normal people.[18] All but one of our patients admitted to significant daily alcohol use, and many noted frequent binges. The poor sanitary habits in 15 of our patients may have played a role in their susceptibility to infection.

These predisposing factors, combined with the large reservoir of *Moraxella* organisms found in our Bowery population, may explain the high incidence of *Moraxella* keratitis in our series. It is interesting that the incidence of *Moraxella* keratitis at Bellevue Hospital has decreased since 1969. The Manhattan Bowery Project was founded in late 1967. This program cleans, delouses, and detoxifies Bowery derelicts. Thus, although the incidence of alcoholism in the Bowery has not changed significantly over the years (R. Morgan, M.D., Medical Director of the Manhattan Bowery Project, personal communication, February 1979), perhaps the improvement in sanitation and nutrition resulting from this rehabilitation program was sufficient to decrease the reservoir and incidence of *Moraxella* infections in the local population.

Summary

Infection with diplobacilli of the *Moraxella* group was the most common cause of bacterial corneal ulcer in a population of derelict alcoholics living in the Bowery district in New York City from 1965 through 1968. No distinction could be made between *Moraxella* subspecies on the basis of severity of ulcer or antibiotic sensitivities. Of 100 Bowery inhabitants examined, 35 harbored *Moraxella* in their noses. After 1968 and coinciding with an improvement in nutrition and sanitation, but with no apparent change in the incidence of alcoholism in this population, we noted a marked decline in the incidence of corneal ulcers caused by *Moraxella* species.

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OPHTHALMIC MINIATURE

"My dear sir, there is no occasion for us to go into strictly professional details together," said he soothingly. "If, for example, I were to say that you have interstitial keratitis, how would you be the wiser?"

Sir Arthur Conan Doyle,
"A False Start." in *Round the Red Lamp*

ADENOVIRUS TYPE 10 KERATOCONJUNCTIVITIS WITH INCREASED INTRAOCULAR PRESSURE

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Physicians treating adenovirus keratoconjunctivitis are usually concerned about its infectiousness and only rarely about its effect on intraocular pressure.[1-5] There are few data documenting intraocular pressure in the acute state of adenovirus infection.

We report a case of bilateral keratoconjunctivitis with transient increased intraocular pressure probably caused by adenovirus type 10.

CASE REPORT

An 18-year-old man had had pharyngitis and a temperature of 40°C on Nov. 21, 1977. Discharge and hyperemia developed two days later in both eyes. On Nov. 28, he came to an ophthalmic clinic because of bilateral photophobia, hyperemia, and blurred vision. Ocular examination disclosed a corrected visual acuity of 6/6 (20/20) in each eye. He also had mild swelling of the eyelids, chemosis, episcleral vessel dilatation, and conjunctival hyperemia with slight follicles (Fig. 1). The corneas showed diffuse epithelial opacities, but could not be stained with fluorescein. The central corneal thickness was normal. We found some preauricular lymphadenopathy and anterior chamber reaction. We diagnosed epidemic keratoconjunctivitis, and prescribed eyedrops of erythromycin and dexamethasone, to be used three times each day.

On Nov. 29, we measured his intraocular pressure with Goldmann's applanation tonometer because we suspected increased intraocular pressure after a digital examination and after finding corneal epithelial opacities. The intraocular pressure was R.E.: 30 mm Hg and L.E.: 26 mm Hg. The pupils were equal in size and reacted normally to light. The optic disks were normal. On Nov. 30, applanation pressure was R.E.: 30 mm Hg and L.E.: 38 mm Hg. Schiøtz tonometer gave readings of R.E.: 34 mm Hg and L.E.: 37 mm Hg. Tonography revealed an outflow facility of 0.25 and 0.13 μl/min/mm Hg in the right and left eye, respectively. The angles appeared open by gonioscopy, with normal trabecular meshwork and no synechiae. We treated the patient with orally administered acetazolamide, 500 mg twice daily, and 0.1% betamethasone eyedrops, four times each day. However, the intraocular pressure stayed between 28 and 46 mm Hg in each eye for the next seven days. By Dec. 2, the swelling of the eyelids, chemosis, episcleral vessel dilatation, and conjunctival hyperemia had greatly diminished. Diffuse epithelial opacities of the corneas had almost disappeared. The anterior chamber had moderate cells and flare without synechia. The patient did not complain of blurred vision or photophobia.

On Dec. 3, fine punctate corneal opacities developed bilaterally (Fig. 2). These were located peripherally and stained minimally with fluorescein. The conjunctival hyperemia was minimal. Flare and cells in the anterior chamber lasted during the next week. On Dec. 12, both corneas had numerous punctate opacities, mainly located at the periphery and in the epithelium and superficial stroma. Some of these were stainable with fluorescein. The intraocular pressure was 18 mm Hg in each eye. The patient

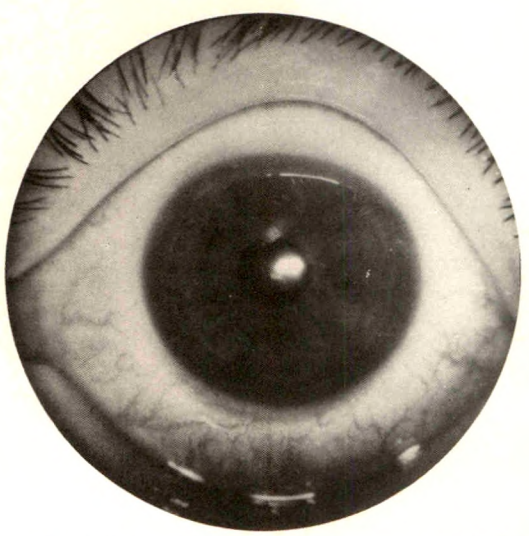


Fig. 1 (Hara and associates). Acute conjunctivitis with dilated episcleral vessels seven days after onset of pharyngitis.

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Fig. 2 (Hara and associates). Slit-lamp photograph showing fine punctate corneal opacities two weeks after onset of the disease.

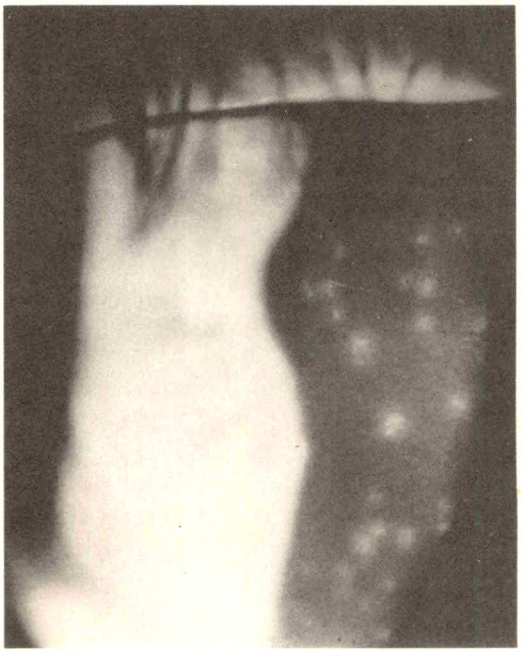


Fig. 3 (Hara and associates). Slit-lamp photograph showing adenovirus type 10 keratitis. About two weeks after the appearance of corneal opacities shown in Figure 2, coarse punctate epithelial and subepithelial opacities developed.

continued to take orally administered acetazolamide, 125 mg, and used 0.1% dexamethasone and 1.25% epinephrine eyedrops twice each day. On Dec. 22, punctate corneal opacities were scattered on the entire corneal surface (Fig. 3). The intraocular pressure was increased again. Applanation pressure readings were R.E.: 30 mm Hg and L.E.: 28 mm Hg. The anterior chambers were quiet. We discontinued dexamethasone eyedrops and added 2% pilocarpine HCl eyedrops in place of epinephrine.

On Jan. 4, 1978, the intraocular pressure of both eyes was normal. Outflow facilities were 0.43 and 0.45 μl/min/mm Hg in the right and left eye, respectively. Ophthalmic examinations disclosed no lesions except bilateral punctate corneal opacities. The patient stopped taking all medication. He has remained totally asymptomatic for ten months. Ocular examination on Sept. 10, 1978, showed normal visual acuity, Goldmann visual fields, and disks. We found no corneal opacities. The intraocular pressure was normal in both eyes.

We compared antibody titers of his acute- and convalescent-phase sera against adenoviruses by complement fixation, hemagglutination inhibition, and neutralizing tests (Table).

We used adenovirus type 4 as the antigen in the complement-fixation test since all types possess the group cross-reactive antigen. The rise in complement-fixation antibody against adenoviruses was evident. The adenoviruses used as antigen in the hemagglutination-inhibition test were types 3, 4, 7, 8, 10, and 11. Only titers of hemagglutination-inhibition antibody against type 10 increased. We also demonstrated a rise in neutralizing antibodies against type 10 adenovirus. We did not detect complement-fixation antibody against herpes simplex virus. Results of a bacteriologic examination of the conjunctival sac on Nov. 29 were normal.

Discussion

Adenovirus infections are commonly caused by adenovirus types 2, 3, 4, 5, 7, and 8.[5-7] Ophthalmically, type 8 is the most important and most frequent cause of epidemic keratoconjunctivitis.[2-4] Ocular diseases caused by type 10 are rare.[8-10] In 1977, Zografos[9] described a new form of viral keratoconjunctivitis caused by adenovirus type 10. The predominant symptoms, after disappearance of the benign conjunctivitis signs by the fifth day, are edema of the eyelids, chemosis, many corneal erosions, and folds in Descemet's membrane, reaching their peak by the 15th day. The course of the disease is about 30 days. In 1978, Stenson, Jaffe, and Newman[10] reported an outbreak of adenovirus type 10 disease characterized

TABLE

ANTIBODY TITERS TO ADENOVIRUS

Titers	Date			
	Nov. 30, 1977	Dec. 9, 1977	Dec. 22, 1977	Jan. 4, 1978
Complement-fixation	NE*	< 4	NE*	16
Hemagglutination-inhibition				
Type 3	<10	<10	<10	<10
Type 4	<10	<10	<10	<10
Type 7	<10	<10	<10	<10
Type 8	<10	<10	<10	<10
Type 10	<10	10	20	20
Type 11	<10	<10	<10	<10
Neutralizing	NE*	< 4	4	64

*NE, not evaluated.

by pseudomembranes and subconjunctival and eyelid hemorrhages. The corneal changes were consistent with those of epidemic keratoconjunctivitis. Systemic symptoms were rare, and the ocular disease was resolved in one to three weeks. In 1979, Tullo and Higgins[7] reported an outbreak of keratoconjunctivitis caused by an adenovirus serologically related to types 10 and 19. The epidemiologic and clinical features of this outbreak were similar to those found in classical adenovirus keratoconjunctivitis. However, none of these reports comment on intraocular pressure.

Our case of keratoconjunctivitis, serologically diagnosed as adenovirus type 10 infection, included transient increased intraocular pressure. Diffuse epithelial corneal opacities and digital examination of ocular tension led us to measure the intraocular pressure accurately. Increased intraocular pressure of about 40 mm Hg in both eyes was present for the next ten days despite medical therapy.

In this case, conjunctivitis appeared several days after the onset of pharyngitis, while punctate keratitis developed about two weeks later and disappeared within ten months. Our observation indicates that adenovirus type 10 may produce corneal lesions similar to those described in epidemic keratoconjunctivitis caused by adenovirus type 8.

We considered several possible causes of the increased intraocular pressure: (1) We excluded corticosteroid glaucoma because corticosteroid eyedrops were used for only one day before the initial increase in intraocular pressure. (2) Swelling of the eyelids and episcleral vein dilation suggest extraocular glaucoma caused by obstructed venous return in the retrobulbar tissues. However, high intraocular pressure continued after disappearance of episcleral vein dilation. Therefore, we ruled out extraocular glaucoma. (3) It is also unlikely that the increase in intraocular pressure occurred as happens in primary open-angle glaucoma. No glaucomatous hereditary tendency was found in the medical history of the patient and his family. (4) On the basis of the above and the evidence of anterior chamber reaction, we concluded that keratoconjunctivitis caused by adenovirus type 10 probably caused the increased intraocular pressure.

SUMMARY

An 18-year-old man, with serologically diagnosed adenovirus type 10 infection, had keratoconjunctivouveitis of both eyes after pharyngitis with transient increased

intraocular pressure. Increased intraocular pressure, ranging from 46 to 28 mm Hg during medical therapy, lasted for about ten days in the early stage of the disease. Many punctate corneal opacities developed bilaterally two weeks later and disappeared within ten months. Analysis of the clinical findings demonstrated that the increased intraocular pressure may have been secondary glaucoma due to keratoconjunctivitis caused by adenovirus infection.

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TUBEROUS SCLEROSIS ASSOCIATED WITH A RETINAL ANGIOMA

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Tuberous sclerosis, or Bourneville's disease, is one of a group of hereditary diseases characterized by disseminated harmartomas involving the eye, skin, and central nervous system. In 1923 Van de Hoeve[1] introduced the term "phakomatoses" to describe this group, which now includes six disease entities: tuberous sclerosis, von Recklinghausen's neurofibromatosis, von Hippel-Lindau disease, meningocutaneous angiomatosis (Sturge-Weber syndrome), ataxia-telangiectasia (Louis-Bar syndrome), and arteriovenous communication of retina and brain (Wyburn-Mason syndrome).[2] Investigators have described associations among and overlapping manifestations of these diseases,[3-6] but there has been only one reported case of retinal angiomas in a patient with tuberous sclerosis.[7] To our knowledge, our case represents the first angiographic documentation of this association in a patient with the typical stigmata of tuberous sclerosis and a peripapillary exophytic retinal angioma. This latter lesion was properly diagnosed only after retinal angiography.

CASE REPORT

A 62-year-old woman known to have tuberous sclerosis came to us because of a peripapillary retinal elevation in her left eye. She denied having ocular symptoms. As a teen-ager she had suffered from both petit mal and grand mal seizures and had tuberous sclerosis. There was no known family history of tuberous sclerosis or other phakomatoses. She used diphenylhydantoin to control the seizures. Adenoma sebaceum (Pringle's disease) was first diagnosed in 1940. In 1977, the patient had most of the skin lesions removed by dermabrasion. In 1978 she underwent neurological and radiological examinations. The neurological findings were unremarkable. Skull X-rays showed ill-defined radiolucencies associated with calcific deposits (Fig. 1). Computed axial tomography disclosed many calcified masses scattered around the margins of the lateral ventricles and protruding into the ventricular space. There was also generalized brain atrophy with dilated sulci, sylvian fissures, and ventricular system (Fig. 2).

In February 1980, her best corrected visual acuity was R.E.: 6/6.75 (20/25) and L.E.: 6/6 (20/20). Aside from an early posterior subcapsular cataract in the right eye, there were no anterior segment abnormalities. Retinal examination of the right eye disclosed a normal optic disk with a myopic crescent and normal retinal vessels. Approximately 6 mm nasal to the disk, we noted a 1.5-mm whitish-yellow, elevated irregular lesion on the retinal surface (Fig. 3). Fluorescein angiography showed only transmitted hyperfluorescence, indicating attenuation of the surrounding and underlying retinal pigment epithelium (Fig. 4). There were no other lesions in this eye. We detected an epipapillary whitish lesion in the center of the optic disk surrounded by sheathed retinal vessels in the left eye. The nasal peripapillary retina was elevated and thickened (Fig. 5). Fluorescein angiography showed diffuse disk leakage, staining of

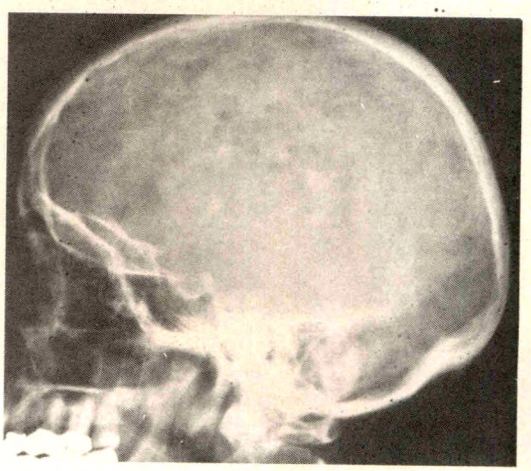


Fig. 1 (Schwartz, Beards, and Maris). Calcified lesions in periventricular location.

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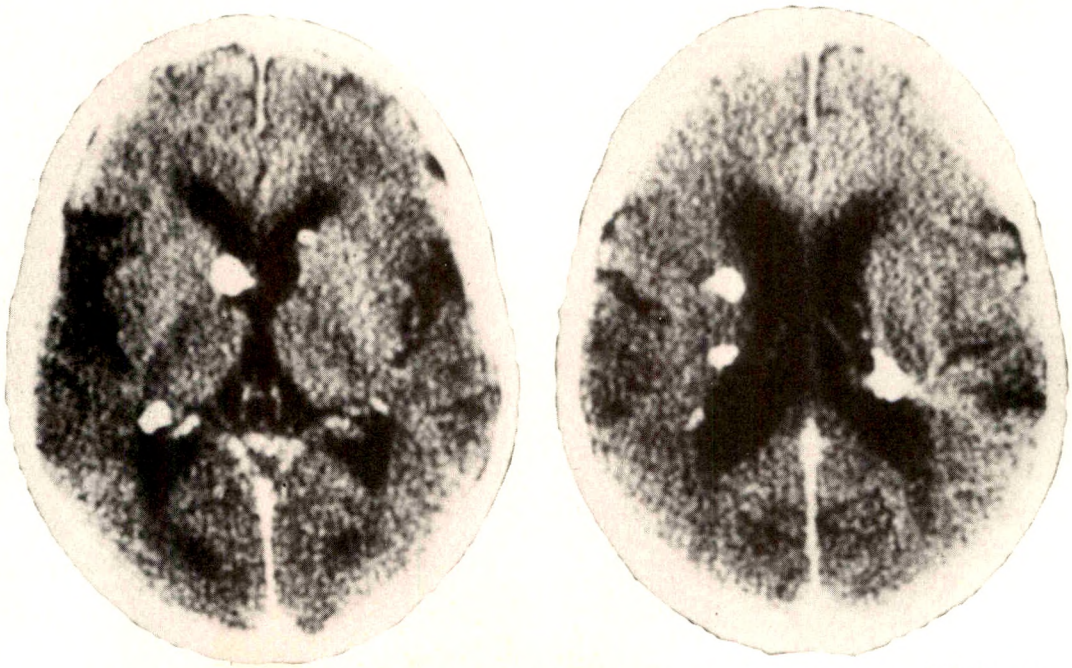


Fig. 2 (Schwartz, Beards, and Maris). Computed axial tomography disclosed many calcified masses surrounding the lateral ventricles.

the epipapillary lesion, and peripapillary intraretinal leakage (Fig. 6). We interpreted the epipapillary disk lesion in the left eye and retinal surface lesion in the right eye as astrocytic hamartomas. We interpreted the peripapillary lesion in the left eye as an exophytic retinal capillary angioma. We did not recommend ocular therapy.

DISCUSSION

Tuberous sclerosis, inherited as a sporadic dominant condition with low penetrance, is characterized by astrocytic glio-

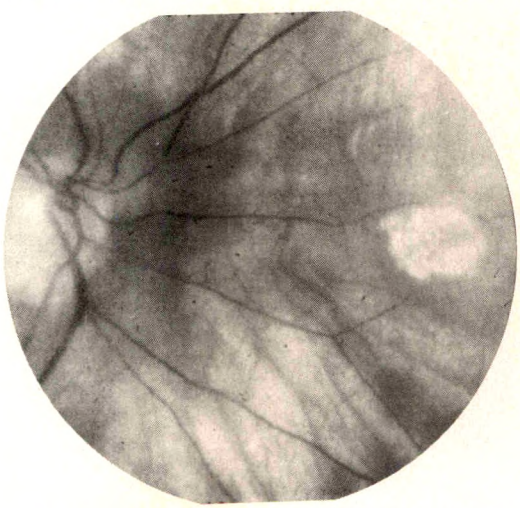


Fig. 3 (Schwartz, Beards, and Maris). Right eye, Astrocytoma on the retinal surface approximately 6 mm nasal to the optic disk.

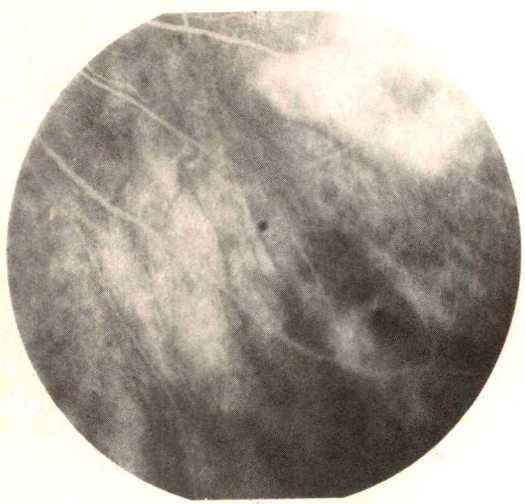


Fig. 4 (Schwartz, Beards, and Maris). Right eye. Mid-venous phase of fluorescein angiogram shows transmitted hyperfluorescence surrounding astrocytoma.

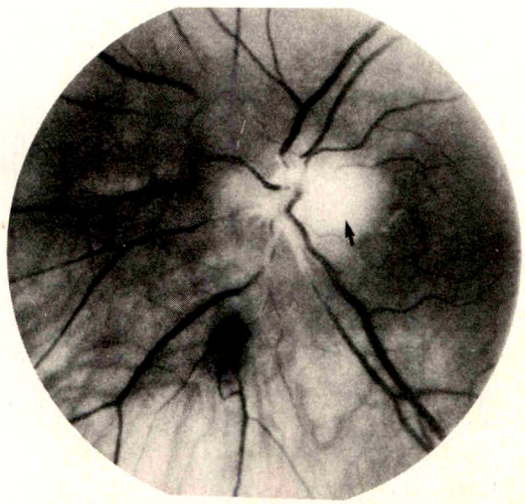


Fig. 5 (Schwartz, Beards, and Maris). Left eye. Epipapillary astrocytoma (arrow), sheathed disk vessel, and elevated thickened papillary retina with superficial retinal hemorrhage.

mas of the optic disk, retina, and central nervous system and hamartomatous lesions of the skin (angiofibromas), heart, lung, and kidney. Retinal capillary angiomas have generally been considered features of angiomatosis retinae (von Hippel's disease). They are usually endophytic, growing above the surface of the retina, are orange-red in color, and are supplied and drained by retinal arteries and veins. Exophytic hemangiomas may be relatively flat and inconspicuous. They are often suspected only after intraretinal and subretinal exudation[8] and confirmed by fluorescein angiography. Lindau was the first to associate capillary angiomas of the cerebellum with angiomatous tumors of the retina.[2]

Mortality is high in patients with tuberous sclerosis; 75% do not live beyond the age of 20 years.[2] One cause of death is obstruction of cerebrospinal fluid by periventricular and intraventricular gliomas. Papilledema may be the first sign, indicating a need for neurosurgical intervention. Our patient was able to avoid extensive neurologic testing because retinal angiography established the correct diagnosis.

Summary

A 62-year-old woman known to have tuberous sclerosis had an unusual peripa-

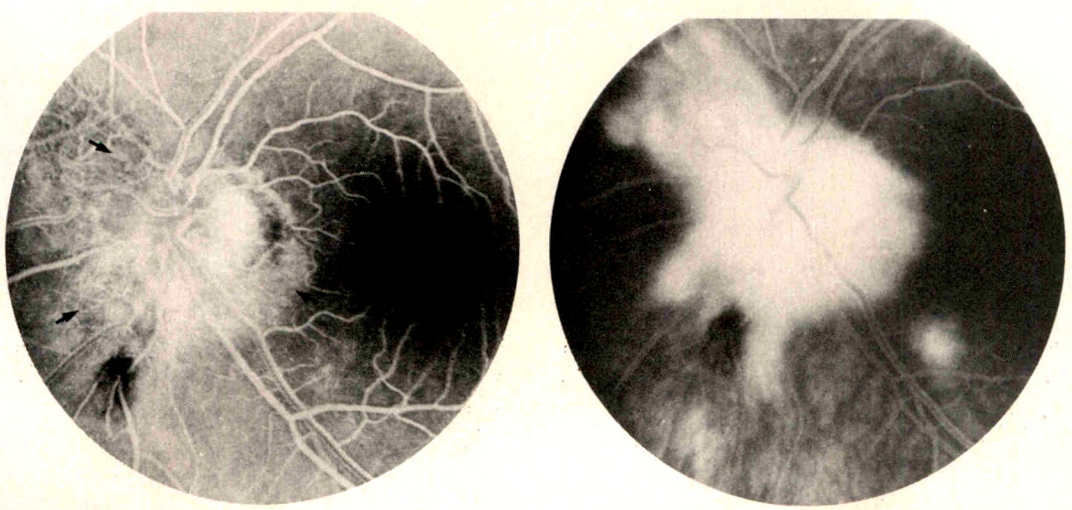


Fig. 6 (Schwartz, Beards, and Maris). Left eye. Left, Early venous phase of fluorescein angiogram shows papillary intraretinal angioma (arrows). Right, Late phase shows diffuse peripapillary and disk leakage.

pillary retinal elevation in her left eye. The patient had no specific ocular symptoms and we considered the diagnosis of papilledema secondary to increased intracranial pressure. Retinal angiography established that she had a peripapillary exophytic retinal angioma associated with an epipapillary astrocytic hamartoma in this eye.

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FILAMENTARY KERATITIS AND STROMAL NEOVASCULARIZATION ASSOCIATED WITH BRAIN-STEM INJURY

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Filamentary keratitis is characterized by the presence of fine filaments on the cornea. These filaments, which are composed of mucus threads and separating epithelial cells, may produce foreign body sensation and pain.[1,2]

We studied three cases of filamentary keratitis, two with stromal neovascularization, associated with brain-stem injury.

CASE REPORTS

Case 1—A 28-year-old man had had redness in his left eye for ten days. It had not responded to treatment with a mixture of 0.1% dexamethasone and neomycin. Approximately two years previously he had sustained a brain-stem injury which resulted in a permanent coma.

Ocular examination in February 1974 showed decreased but present corneal sensation in the left eye and perilimbal injection with several filaments on the cornea. Both seventh cranial nerves were intact. Artificial tears, patching, and debridement did not improve his filamentary keratitis. Two months later the right eye had developed several filaments. The filaments on the right eye resolved within two months. The left cornea developed a stromal vascular ingrowth medially, approximately four months after the filaments developed. Over the next 16 months the vessels increased to include the entire stroma. The filaments then resolved, followed by regression of the vessels.

Case 2—In July 1978, a 19-year-old woman who had sustained a brain-stem injury in an automobile accident in November 1977 came to us because of photophobia, pain, and severe blepharospasm of both eyes.

Ocular examination revealed severe filamentary keratitis with stromal neovascularization in both eyes (Fig. 1). There was corneal sensation. She also had a partial right-sided third nerve paralysis. The right pupil was in mid-position and poorly reactive. Both seventh cranial nerves were intact, shown by bilateral blepharospasm secondary to pain from the filamentary keratitis.

We treated the patient with soft bandage lenses (base curve, 8.1 mm; diameter, 14 mm) (Fig. 2). The filamentary keratitis and neovascularization resolved in both eyes (Fig. 3). She lost the left lens several weeks after insertion, but the keratitis did not recur.

Case 3—In September 1975, we examined a 60-year-old man who had a two-month history of pain in the right eye. He had sustained a brain-stem injury in an automobile accident in October 1974.

Ocular examination showed filamentary keratitis of the right eye with decreased but present corneal

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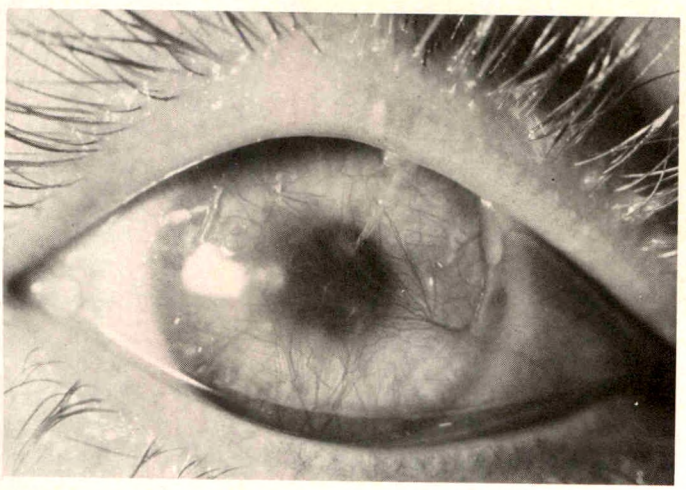


Fig. 1 (Davis, Drewry, and Wood). Case 2. Right eye with filaments and neovascularization before insertion of soft contact lens.

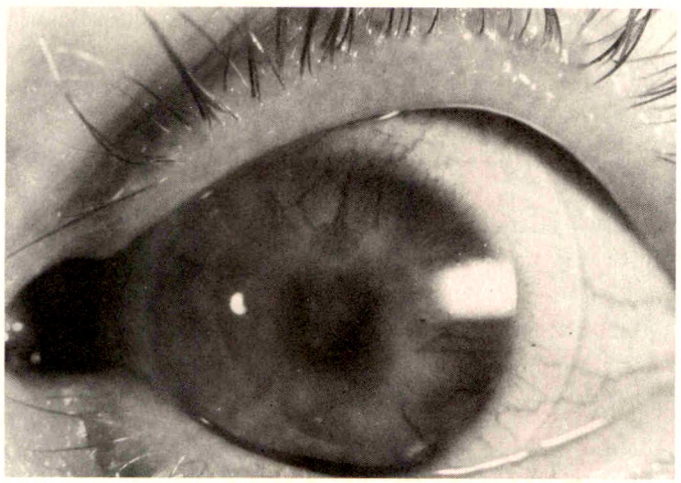


Fig. 2 (Davis, Drewry, and Wood). Case 2. Left eye several days after insertion of soft contact lens.

sensation. Both seventh cranial nerves were intact. The patient had vertical nystagmus and a visual acuity of 6/120 (20/400) in each eye. There were no other unusual findings.

We treated the eye with a soft bandage lens (base curve, 8.1 mm; diameter, 14.00 mm). The filamentary keratitis resolved within 48 hours and did not recur.

Discussion

The cause of filamentary keratitis remains uncertain. It is associated with keratitis sicca,[3] recurrent erosion, herpetic keratitis, atopic dermatitis, neurotrophic and neuroparalytic keratitis secondary to fifth and seventh cranial nerve paralysis, prolonged occlusion, retained foreign body beneath the upper eyelid, superficial punctate keratitis, keratoconus, psoriasis,[4] cataract extraction,[5] Osler-Weber disease,[6] beta radiation, diabetes mellitis, ectodermal dysplasia and aerosol keratitis,[7,8] and traumatic subdural hematoma.[9]

Two of our patients, in whom filamentary keratitis had been present for an extended period, showed severe stromal neovascularization. The third patient,

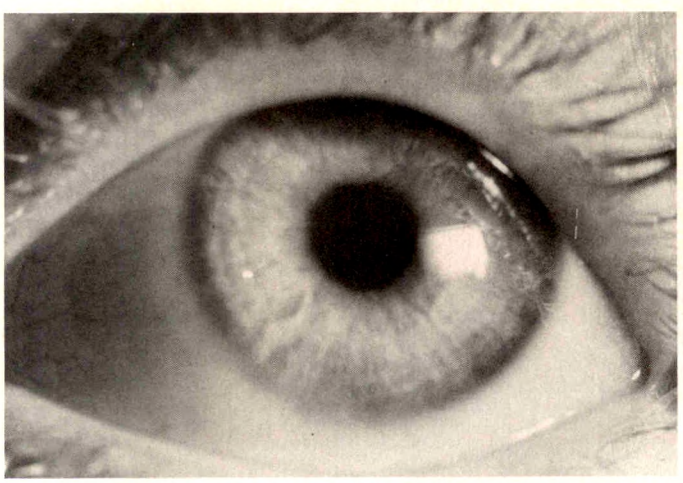


Fig. 3 (Davis, Drewry, and Wood). Case 2. Left eye after resolution of neovascularization and filaments; contact lens has been removed.

who received earlier treatment with a soft contact lens, did not develop stromal neovascularization. The common findings in these three cases are the occurrence of filamentary keratitis in brain-stem injuries with preserved corneal sensation and the absence of other causes of filamentary keratitis.

The two patients treated with soft contact lenses responded dramatically with resolution of the filaments within 48 hours. In Case 2 the neovascularization and filamentary keratitis did not recur even when one lens was lost.

Summary

Three cases of filamentary keratitis were associated with brain-stem injuries. Severe stromal neovascularization developed in two cases. There was corneal sensation in the involved eyes. The first case resolved spontaneously over a 20-month period after stromal neovascularization. Two patients responded rapidly to treatment with soft contact lenses. The vascularization resolved in both cases, but much sooner in the eyes treated with the soft contact lenses.

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AN ULTRASTRUCTURAL STUDY OF THE CORPORA AMYLACEA OF THE OPTIC NERVE HEAD AND RETINA

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Corpora amylacea are microscopic structures found in histologic preparations of the optic nerve and the nerve fiber layer of the retina.[1] In the central nervous system they are found in the brain and the spinal cord. In histologic sections, they usually appear as homogeneous oval bodies approximately 15 μ in diameter, but occasionally they are laminated with dense centers. They stain positively with basophilic stains, iodine, methyl violet, PAS, Hale's colloidal iron, and Best's stain for glycogen.[2] Stam and Roukema[3] showed that the corpora amylacea do not stain with Congo red, and disputed the thesis of von Recklinghausen that corpora amylacea are deposits of amyloid.

In an ultrastructural study of the brain tissue in a single human case, Ramsey[4] interpreted corpora amylacea as being glial in nature. Blackwood and Corsellis,[2] Yanoff and Fine,[5] and Avendano and associates[6] concurred that corpora amylacea are astrocytic. Anzil and colleagues[7] examined the brains from aged patients and claimed to see corpora amylacea inside astrocytic processes and also observed them within neuronal processes on rare occasions. Takahashi, Iwata, and Nakamura[8] noted that corpora amylacea are "intra-axonal" in the central nervous system. Using silver carbamate staining, Wolter and Liss[9,10] followed the development of corpora amylacea, which they termed hyaline bodies, from nerve fibers of the optic nerve and retina. Hogan and Zimmerman[11] described them as being "acellular" in ocular tissues.

The pathologic significance of corpora amylacea has not been determined. Yanoff and Fine[1] believed that they have no clinical significance and are a phenomenon of aging. Yagashita and Itoh,[12] however, found them in axons of peripheral nerve in patients with familial spastic paralysis, whereas Takahashi and colleagues[8,13] noted corpora amylacea in the spinal cords and central nervous systems of patients with degenerative nervous diseases. In a case of syphilitic optic atrophy, Wolter and Liss[10] observed an abundance of corpora amylacea in both optic nerves.

We examined the optic nerve heads of four human eyes and observed definite evidence that corpora amylacea in the retina and the optic nerve head are axonal in nature.

MATERIAL AND METHODS

We enucleated four eyes from four patients. Three cases had been diagnosed as malignant melanoma of the choroid and the fourth as recurrent fibrous histiocytoma of the orbit. Subsequent pathologic examination confirmed the clinical diagnosis in each case. These patients had each received an injection of sickle-cell blood into their anterior chambers before enucleation for studies of hyphema and glaucoma.[14]

After 12 to 116 hours of increased intraocular pressure, ranging from 20 to

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60 mm Hg, the eyes were enucleated, opened, and immediately immersed in McDowell and Trump's[15] fixative. The tissues were postfixed in Dalton's chrome osmium, dehydrated, and embedded in an araldite-based agent (Durcupan). We cut sections of optic nerve head and retina with an ultramicrotome and stained them with uranyl acetate and lead citrate. We stained thick sections with Mallory's azure II and methylene blue.

Results

Light microscopic observations—We found corpora amylacea in the sections of the retina and optic nerve of all four patients. We noted mild vacuolations of nerve fibers and pathologic changes caused by acute increase in intraocular tension of the optic nerve head and peripapillary retina in each patient. We have described these elsewhere.[16] The corpora amylacea were in the lamina scleralis, choroidalis, and retinalis of the optic nerve head, as well as in the nerve fiber layer and inner plexiform layer of the peripapillary retina. We observed them most often where vacuolation and degenerative changes of axons were prominent.

In histologic sections, most of the corpora amylacea appeared as isolated oval bodies with a discrete outline (Fig. 1). They had diameters of about 5 to 25 μ. Some appeared to be enclosed by a plasma membrane. Occasionally the surrounding axons were displaced (Fig. 1, top left). Under higher magnification, we saw dense (Fig. 1, top left) or pale (Fig. 1, top right) central cores with concentric lamellae. Some corpora amylacea had scattered granules at the periphery (Fig. 1, top right and bottom right). With Mallory's stain on plastic-embedded sections, the corpora amylacea appeared mildly metachromatic.

Electron microscopic observations—Each of the corpora amylacea consisted of a filamentous tangle with a whorl-like pattern that corresponded to the concentric laminations noted by light microscopy (Fig. 2). The tangle had a discrete nonmembranous border. The filaments were 8 to 12 nm in diameter and were arranged in small cartwheel groupings in a dense matrix. Within the filamentous tangle, some discrete areas exhibited a loose ground substance which contained scattered tubules (Fig. 3). In other areas, the filaments aggregated together to form small bundles 25 to 50 nm in diameter (Fig. 4). Occasionally, we observed vacuolated organelles and dense lamellar bodies suggestive of degenerated mitochondria enmeshed with the filaments (Fig. 4). In still other corpora amylacea, we saw dense granular particles resembling glycogen within the filamentous tangle (Fig. 5).

A thin rim of axoplasm and a plasmalemma surrounded most of the corpora amylacea. The axoplasm sometimes consisted of mitochondria, neurotubules, and normal cytoplasmic ground substance (Fig. 3). The axoplasm around other corpora amylacea appeared lucent and watery (Fig. 2). In still others, numerous dense lamellated bodies, multivesicular bodies, and degenerated mitochondria accumulated in the axoplasm (Fig. 6). The filamentous tangle of the corpora amylacea was surrounded by plasmalemma without intervening axoplasm (Fig. 5) in rare instances.

We found individual corpora amylacea in myelinated as well as unmyelinated nerve fibers (Fig. 7). The segments of axons that contained the structures were greatly increased in diameter. The axons proximal or distal to the swelling containing the corpora amylacea appeared to have a normal diameter (Fig. 3). Many of the surrounding axons were normal in size. The glial processes surrounding these axons appeared normal and did not contain corpora amylacea (Fig. 2).

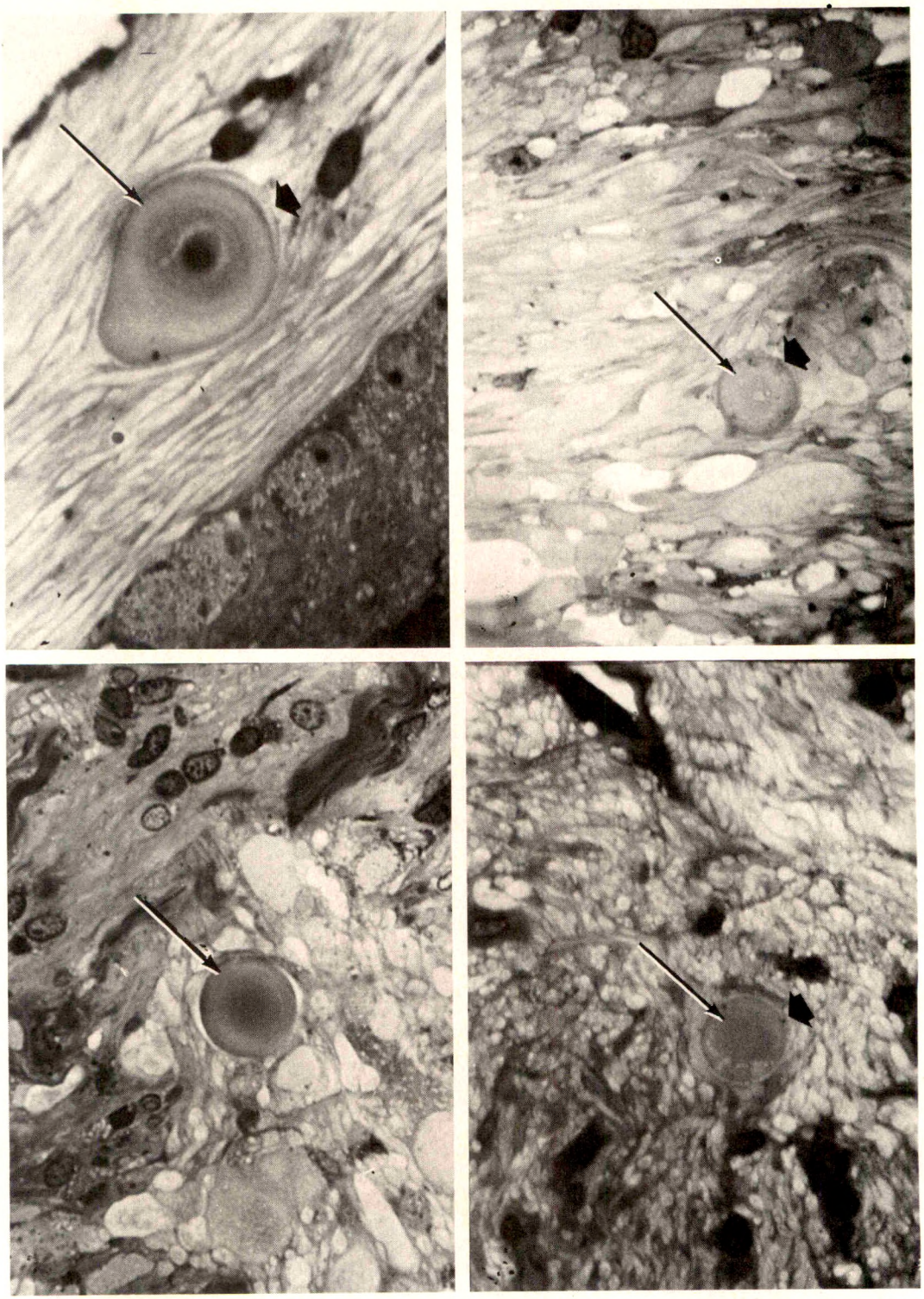


Fig. 1 (Woodford and Tso). Top left, A corpus amylaceum (thin arrow) in the nerve fiber layer of the peripapillary retina is enclosed by a plasma membrane (thick arrow) and exhibits a dense core with concentric laminations. The surrounding axons are displaced (× 800). Top right, A corpus amylaceum (thin arrow) in optic nerve head consists of a pale core with concentric laminations and small peripheral granules (thick arrow) (× 800). Bottom left, A corpus amylaceum (thin arrow) in optic nerve head appears relatively homogeneous with a dark center (× 800). Bottom right, A corpus amylaceum (thin arrow) in optic nerve head appears homogeneous with peripheral granules (thick arrow) (methylene blue and azure II × 800).

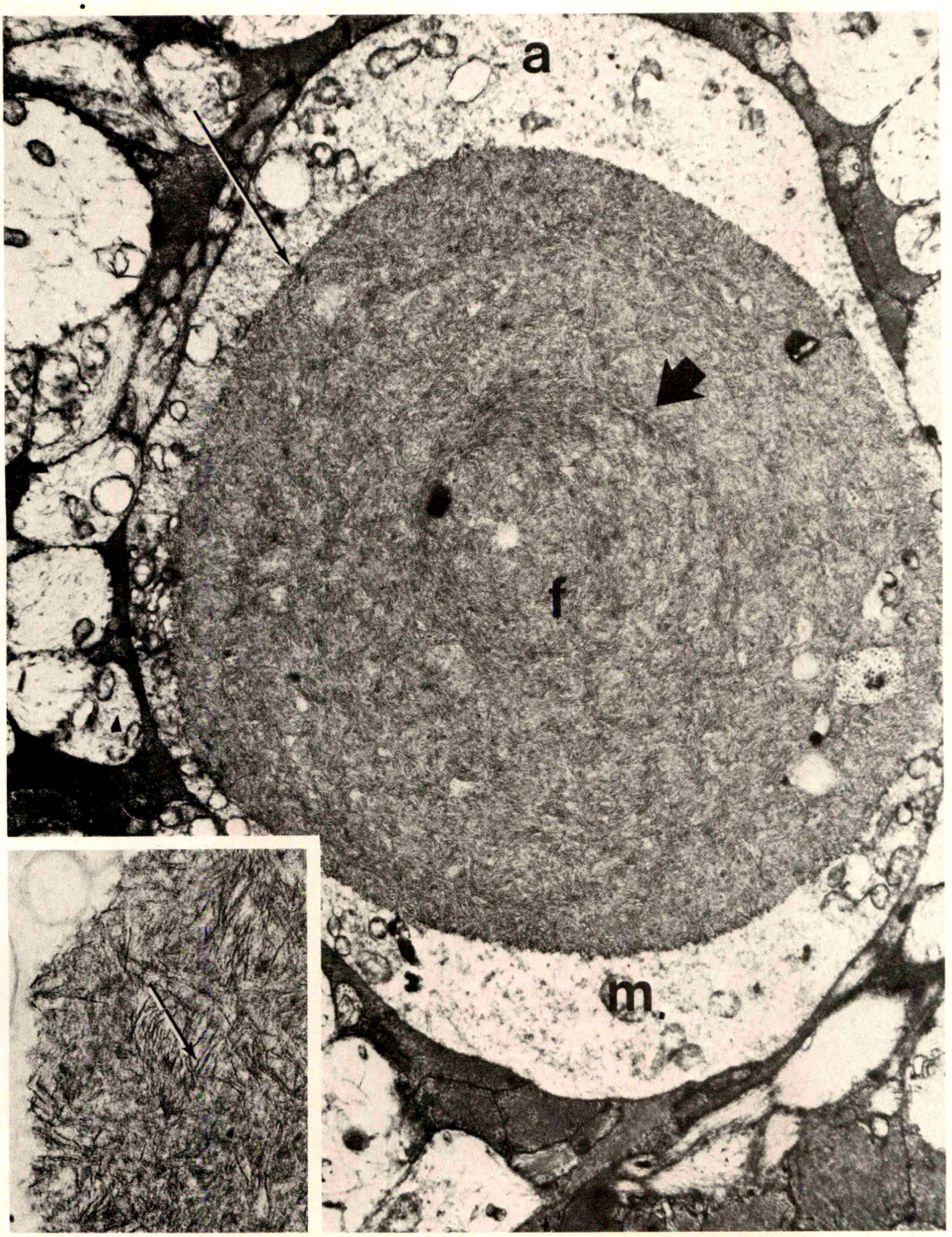


Fig. 2 (Woodford and Tso). A corpus amylaceum surrounded by lucent cytoplasm (a), containing vacuolated mitochondria (m). The central filamentous tangle (f) is arranged in whorls (thick arrow) and delineated by a discrete nonmembranous border (thin arrow) (× 8,400). Inset, Cartwheel arrangement of filaments is evident under higher power (arrow) (× 44,000).

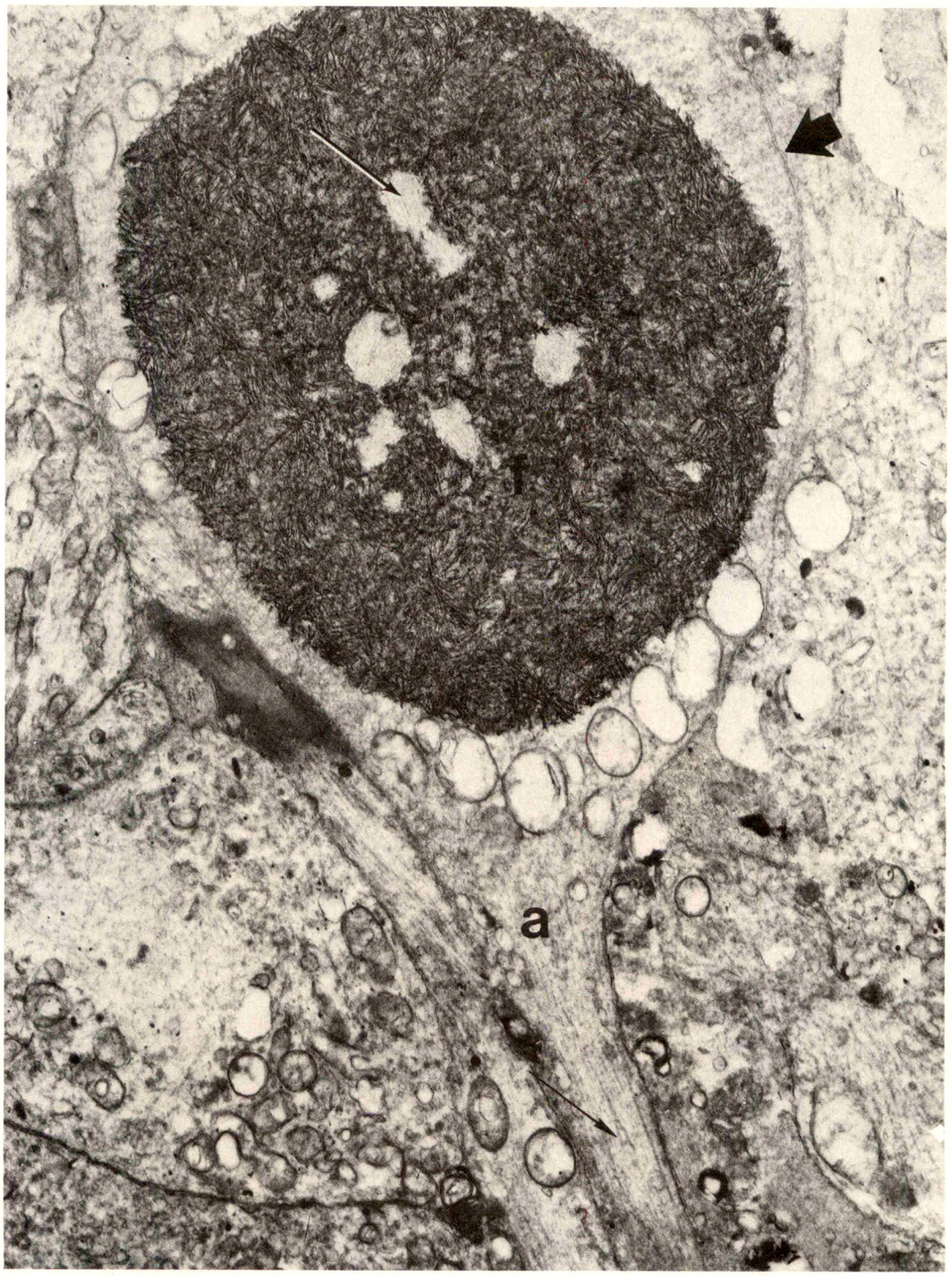


Fig. 3 (Woodford and Tso). Swollen segment of an axon (thick arrow) contains a corpus amylaceum and merges into a narrow axonal segment (a). Normal neurotubules (thin black arrow) are present in the normal axonal segment. Central areas of loose ground substance within the filamentous tangle (f) also contain similar tubules (black-and-white arrow) (× 17,600).

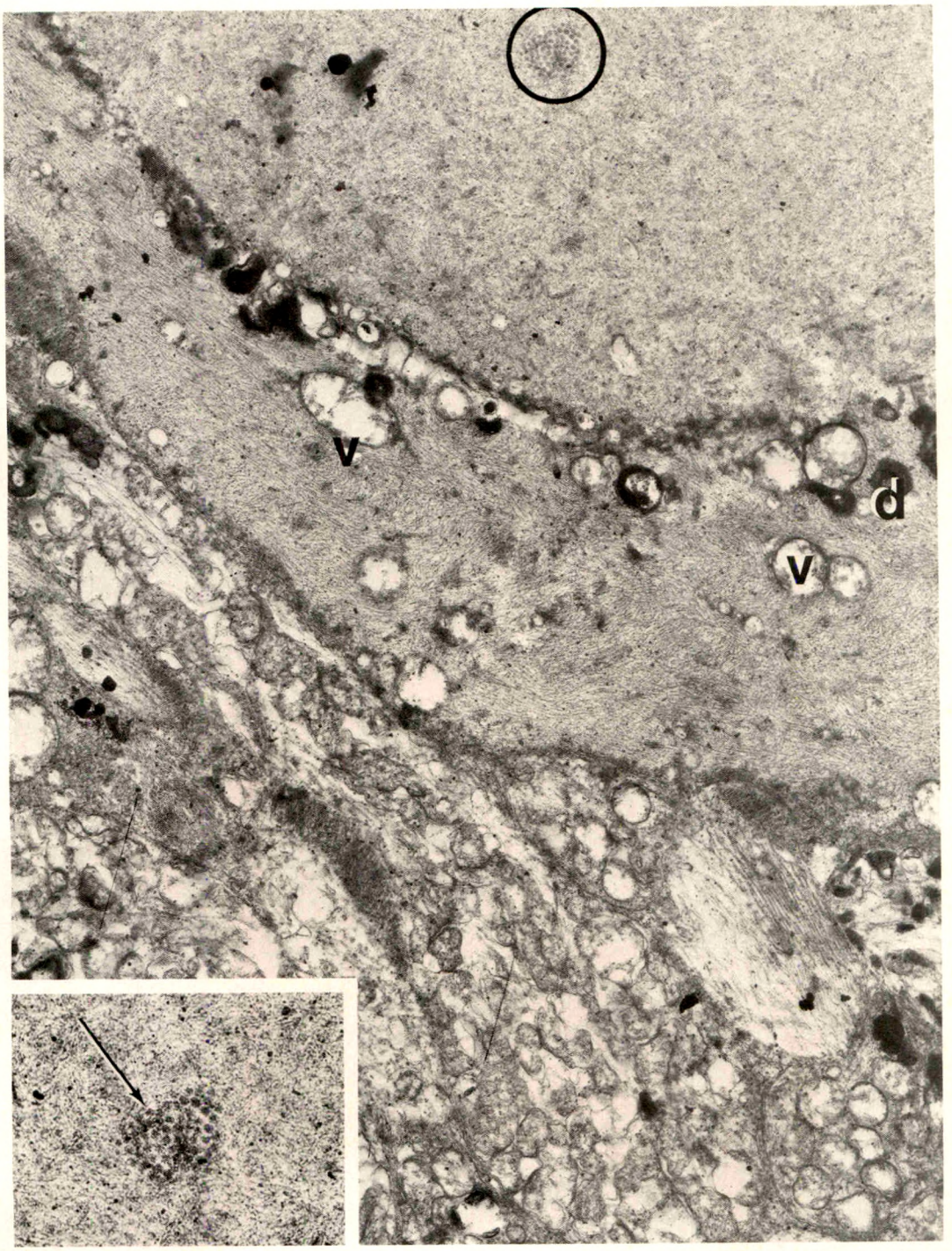


Fig. 4 (Woodford and Tso). Portion of a corpus amylaceum containing vacuolated organelles (v), dense bodies (d), and bundles of filaments (circle) (× 15,000). Inset, Fine filaments arranged in small bundles (arrow) (× 26,600).



Fig. 5 (Woodford and Tso). Corpus amylaceum showing glycogen particles (arrows) within the filamentous tangle (f) (× 8,500).

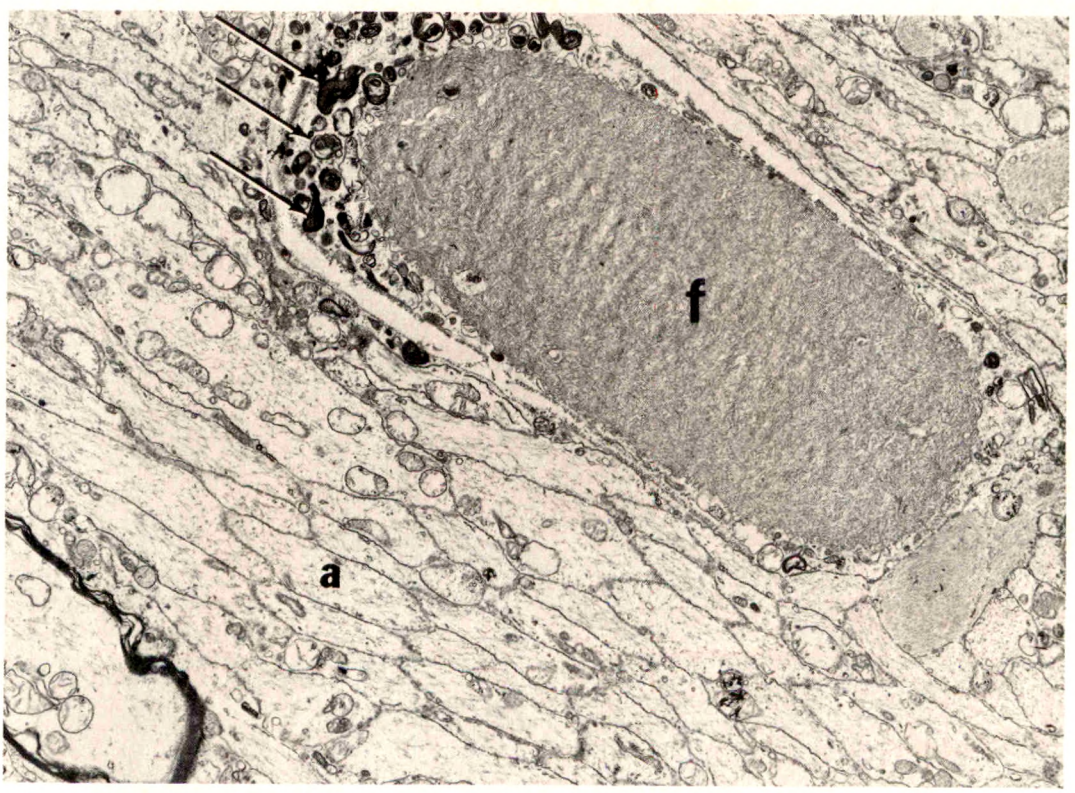


Fig. 6 (Woodford and Tso). Corpus amylaceum (f) surrounded by dense lamellated bodies (arrows) in the axoplasm. The surrounding axons (a) appear relatively normal (× 6,500).

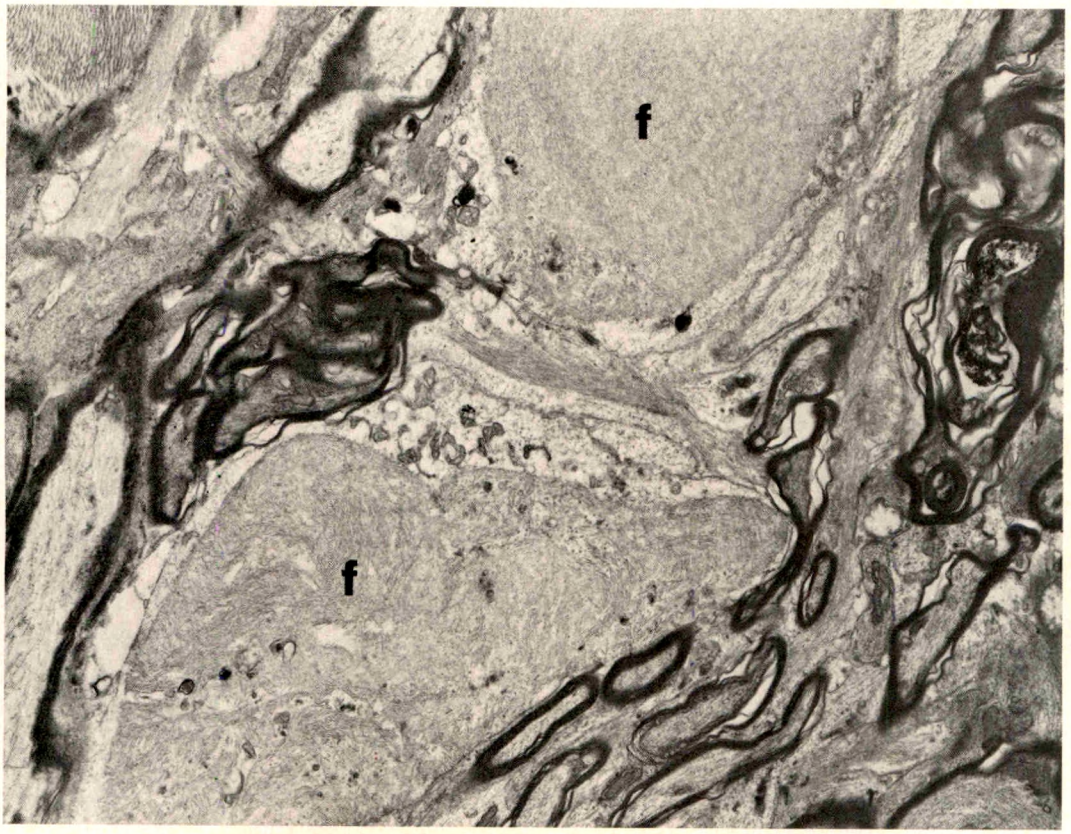


Fig. 7 (Woodford and Tso). The filamentous tangles (f) of corpora amylacea in myelinated optic nerve (× 5,800).

We saw some swollen axons, which appeared to be precursors of the corpora amylacea, in the optic nerve head (Fig. 8). These axons consisted of a central filamentous core that merged imperceptibly with the surrounding axoplasmic components. The diameter of the filaments was 8 to 12 nm, the same as that of the filaments in the corpora amylacea. These filaments appeared more orderly than those seen in the filamentous tangle of the corpora amylacea (Fig. 8, inset). Mitochondria and dense bodies surrounded this central filamentous core.

Discussion

Our electron microscopic study showed that the corpora amylacea in the optic nerve head and the retina are intracellular organelles found in axonal swellings. They are not glial in origin and they are not found in astrocytes as claimed by Yanoff and Fine[5] and Avendeno and associates.[6]

In our morphologic study, the corpora amylacea consisted of tangles of filaments 10 nm in diameter, comparable to the neurofilaments of normal axons.[17] We also saw occasional clusters of tubules (25 nm in diameter) within the tangle. These tubules resembled the microtubules of normal axons.

Also intermingled within the filamentous tangles of corpora amylacea were lamellated or dense bodies and mitochondria. This arrangement distinctly resembled cytoid bodies[18] found in the retina or optic nerve head with papilledema or

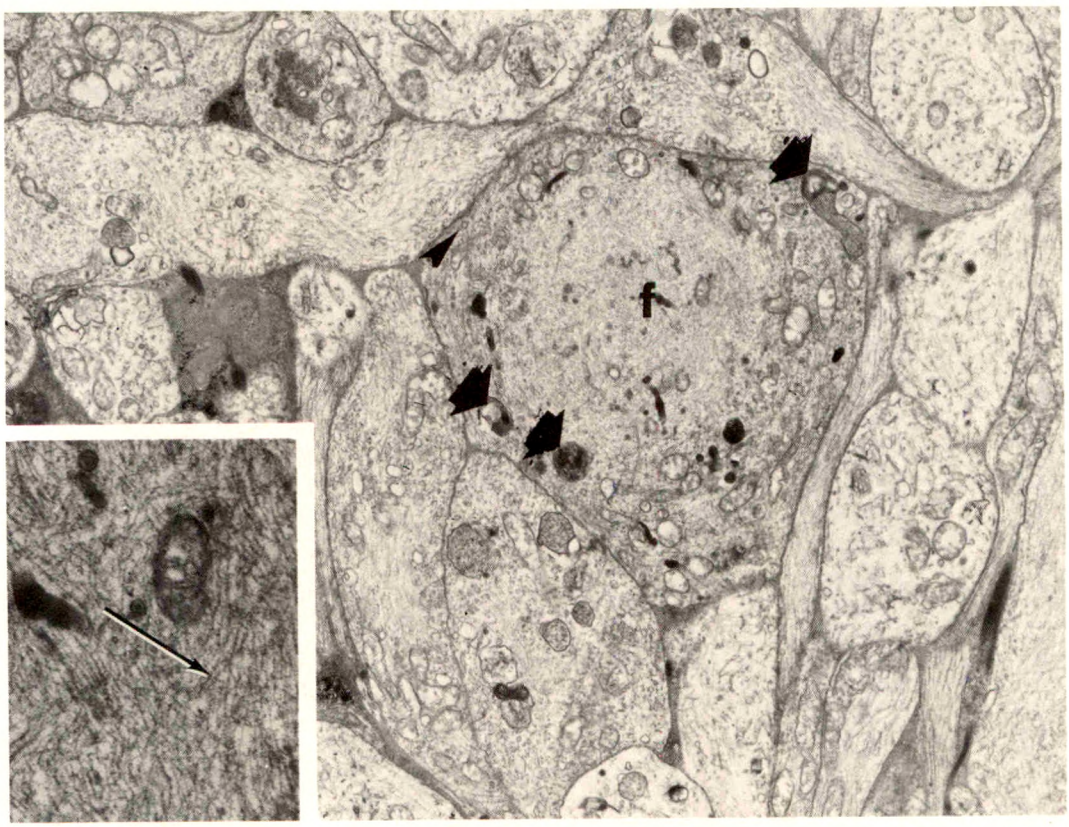


Fig. 8 (Woodford and Tso). A degenerated axon with a central filamentous core (f) surrounded by mitochondria and dense bodies (thick arrows) (× 7,500). Inset, Filaments (arrow) in the central core (× 22,500).

glaucoma. We saw a similar intermingling of accumulated neurofilaments and axoplasmic components in axons of peripheral nerve[19] and spinal cord[20] associated with disturbance of axoplasmic transport.

Some of the filamentous tangles were surrounded by a rim of normal-looking axoplasm containing mitochondria, neurotubules, and neurofilaments. Other axons containing corpora amylacea showed an accumulation of laminated or dense bodies, resembling axonal swellings immediately proximal to the site of obstruction of axoplasmic transport. Still other axonal swellings containing the corpora amylacea had watery cytoplasm and vacuolated mitochondria, resembling axonal swelling more proximal to the site of axoplasmic transport block. The varied appearance of corpora amylacea and the surrounding axoplasm may reflect different stages in their formation as well as the various degrees of severity of axonal degeneration.

Tangles of neurofilaments similar to those of corpora amylacea have been produced experimentally in neurons in response to toxic substances. Seil and Lampert[21] induced neurofilament tangles after exposing cultured rabbit spinal cord to vincristine and vinblastine sulfate. Selkoe and associates[22] produced neurofilament tangles in the spinal cord of rabbits with aluminum chloride; biochemical analysis of the tangles identified "triplet" proteins, which are considered

to be the components of normal neurofilaments. β-β′-iminodipropionotrile, a slow axoplasmic transport inhibitor, caused neurofilament tangles with accumulation of axoplasmic organelles in the spinal cords of rats.[23] Morphologic and biochemical studies showed that these tangles consisted of 10-nm filaments arranged in "whorled fascicles" associated with scattered microtubules and an accumulation of triplet proteins.[24] These studies suggest that the neurofilament tangles consist of triplet proteins and may be related to axoplasmic transport disturbance. Morphologically, the filament tangles of corpora amylacea resemble those produced experimentally.

Sakai and co-workers[25] and Stam and Roukema[3] observed that approximately 80% of the isolated corpora amylacea preparations consisted of carbohydrates. They believed that the filaments in corpora amylacea are fibrils of glucose polymer. In our ultrastructural studies, we observed osmiophilic granules consistent with those of glycogen. It is possible that both glucose polymer and triplet protein of neurofilaments are involved in the formation of corpora amylacea.

The significance of corpora amylacea is still uncertain. Anzil and colleagues[7] found them in abundance in neurons of aged human brain and suggested that they may be a result of metabolic defects. Yagashita and Itoh[12] implicated generalized axonal degeneration in their study of corpora amylacea in peripheral nerves of patients with inherited spastic paralysis. Similarly, LaFora bodies, which are diagnostic for myoclonic epilepsy, have ultrastructural and biochemical features comparable to corpora amylacea.[7,12,25,26] These studies implied that corpora amylacea are manifestations of degenerative axonal disease.

In our study, the relative abundance of corpora amylacea in the optic nerve heads of four patients with increased intraocular pressure suggests that corpora amylacea may indeed be related to the disturbance of axoplasmic transport and axonal degeneration. However, the question of why certain selected individual axons are associated with corpora amylacea requires further investigation.

Summary

We studied corpora amylacea in the optic nerve heads and retinas of four human eyes with increased intraocular pressure by light and electron microscopy. Ultrastructurally they consisted of a mass of filamentous tangle discretely bordered by normal or abnormal axoplasm within an axonal swelling. We found neurotubules, mitochondria, and the accumulation of dense bodies in the axoplasm surrounding the filamentous tangle. We noted ultrastructural similarities between corpora amylacea and cytoid bodies, swollen axons with transport block, and experimentally induced neurofibrillary tangles. We concluded that corpora amylacea are intracellular organelles found in axonal swellings and are not glial in origin, as previously believed. There is a possible relationship between corpora amylacea and disturbance of axoplasmic transport.

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RHEGMATOGENOUS RETINAL DETACHMENT WITHOUT MACULAR INVOLVEMENT TREATED WITH SCLERAL BUCKLING

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Results of surgical repair of rhegmatogenous retinal detachment are evaluated by assessment of the anatomic reattachment rate and visual acuity. Although various authors have mentioned that macular status is important in visual outcome, many reviewers have not differentiated between cases in which the macula was included in the detachment and those in which the macula was spared.[1-7] In regard to visual outcome, eyes with rhegmatogenous retinal detachments and spared maculas represent a distinct group because they generally have good central visual acuity preoperatively. Although the functional outcome of successfully treated retinal detachments with spared maculas is expected to be excellent, postoperative visual acuity may occasionally be reduced because of unforeseen complications. To gain information that might be helpful in our preoperative discussions with patients, we reviewed the anatomic success and visual results in a consecutive series of treated eyes that had rhegmatogenous retinal detachments without macular involvement. We evaluated various preoperative factors, operative methods, and postoperative events that might be related to these results.

SUBJECTS AND METHODS

Between August 1969 and October 1977, one of us (D.M.R.) treated 682 consecutive primary rhegmatogeous retinal detachments with scleral buckling. We used information prospectively collected on special retinal code sheets for statistical analysis. Data from a minimal follow-up period of six months were available for 646 eyes. Of these 646 eyes, 173 (27%) had rhegmatogenous retinal detachments with spared maculas. We determined the ultimate anatomic success and visual outcome for this group, and analyzed the results in relation to various factors, including preoperative findings, surgical management, and postoperative events. We compared some of these factors with similar data derived from a separate analysis of 473 eyes that had rhegmatogenous retinal detachments with macular involvement.

RESULTS

Of the 173 eyes with rhegmatogenous retinal detachments and spared maculas, 153 (88%) had preoperative central visual acuities recorded as 6/15 (20/50) or better. In comparison to eyes with retinal detachments involving the macula, eyes with rhegmatogenous retinal detachments and spared maculas had lower incidences of aphakia and detachment of the nonpigmented epithelium but higher incidences of lattice degeneration and demarcation lines. The detachments generally had more anteriorly located tears, involved fewer quadrants, and tended to occur in younger patients (Table 1). Factors that were similar in each group included sex, history of trauma, incidence of vitreous hemorrhage, incidence of glaucoma, number of subretinal precipitates, and number of retinal breaks.

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TABLE 1

PREOPERATIVE FINDINGS IN 646 EYES

Findings	Macula Spared (No. = 173)		Macula Detached (No. = 473)	
	No.	%	No.	%
Average age of patients (yrs)	53	—	58	—
Preoperative visual acuity				
6/4.5 to 6/9 (20/15 to 20/30)	126	72.8	14	3
6/12 to 6/15 (20/40 to 20/50)	27	15.6	33	7
6/18 (20/60) or worse	19	11	426	90
No. of detached quadrants				
≤1	94	54	57	12
2	62	36	184	39
≥3	17	10	232	49
Aphakia	19	11	104*	22*
Duration of detachment <1 mo	99	57	312	66
Demarcation lines	47	27	85*	18*
Lattice degeneration	57	33	104*	22*
Detachment of pars plana epithelium	7	4	95†	20†
Tears posterior to equator	26	15	109‡	23‡

*$P < .01$ by χ^2 test.
†$P < .001$ by χ^2 test.
‡$P < .05$ by χ^2 test.

At surgery, we used an encircling buckle in only 55 (32%) of the eyes with spared maculas, compared to 241 (51%) in those with macular involvement (Table 2). We released subretinal fluid through a sclerotomy in 92 (53%) of the eyes with spared maculas, compared to 383 (81%) with macular involvement.

Postoperatively, 171 (99%) of the 173 eyes were anatomically reattached and 150 (87%) had visual acuities of 6/15 (20/50) or better (Table 2). The two anatomic failures were related to the development of massive periretinal proliferation.

Of the 173 eyes, 15 (9%) had improvement in visual acuity of more than two lines. Events responsible for improvement included dissipation of vitreous hemorrhage or debris, resolution of uveitis, and resolution of posttraumatic hyphema or retinal edema.

Of the 173 eyes, 18 (10%) had a decrease in visual acuity of more than two lines. Of these 18 eyes, we had treated ten (56%) with an encircling buckle and 12 (67%) with release of subretinal fluid. Seven eyes required more than one procedure; only five of the seven were ultimate successes. This reoperation rate of 39% contrasts with a rate of 10% in our total series of 646 eyes.

Decreased visual acuity in these eyes is related primarily to changes in the maculas after surgery. In two patients, pigment was found under the maculas after the operation, although the maculas had been considered uninvolved before the operation. In these two patients, the detachments possibly progressed to involve the maculas between the time of the evaluation and the surgical repair. In three patients, maculas became detached after the first procedures failed (Table 3).

DISCUSSION

Macular status is important in evaluating rhegmatogenous retinal detachments, for once the macula is detached, visual acuity seldom returns to normal. Nevertheless, many studies reviewing results of surgical repair for retinal detachment do

TABLE 2

POSTOPERATIVE FINDINGS IN 646 EYES

Findings	Macula Spared (No. = 173)		Macula Detached (No. = 473)	
	No.	%	No.	%
Encircling buckle	55	32	241*	51*
Release of subretinal fluid	92	53	383*	81*
Anatomic reattachment	171	99	426†	90†
Final visual acuity				
6/15 (20/50) or better	150	87	175*	37*
6/9 (20/30) or better	125	72	85*	18*

*$P < .0001$ by χ^2 test.
†$P < .001$ by χ^2 test.

not indicate whether the macula had been spared or detached.[1-7] However, in reviewing a small series of 31 eyes, Kaufman[8,9] noted that all seven eyes with spared maculas were successfully repaired and had postoperative visual acuities of 6/12 (20/40) or better. Grupposo[10] reviewed 251 eyes and observed that 72 had spared maculas, 88% of which had final visual acuities of 6/22 (20/70) or better. He noted that 15% had final postoperative visual acuities worse than the preoperative visual acuities. Kreissig,[11] describing 70 patients, stated that all recovered presurgical visual acuity if the macula was not involved. Norton[12] reported on 110 eyes with rhegmatogenous retinal detachments and spared maculas and observed that 84% of those successfully reattached had postoperative visual acuities of 6/15 (20/50) or better. Bernardczykowa[13] observed that the final visual acuity was between 6/6 (20/20) and 6/7.5 (20/25) for 12 patients with spared maculas. Marquez,[14] however, observed that visual recovery was only 75% to 95% if the macula was spared preoperatively.

TABLE 3

CAUSES OF POSTOPERATIVE VISUAL ACUITY DECREASED MORE THAN TWO LINES*

Cause	No. of Eyes
Preretinal membrane and pucker	6
Macular detachment recognized after first operation	3
Pigment migration under macula; macular detachment not recognized preoperatively	2
Increase in vitreous debris with possible cystoid macular edema	2
Progression of cataract	3
Ultimate failure	2

*In 173 patients with spared maculas.

In our group of patients with rhegmatogenous retinal detachments and spared maculas, we observed an anatomic success rate of 99%. Burton and Lambert[2] also noted a high anatomic success rate (98%) in their patients who had preoperative visual acuities of 6/15 (20/50) or better, regardless of whether the macula was included in the detachment. We had an identical anatomic success rate of 98% (49 of 50 patients) for our patients who had detachments with macular involvement, provided that their visual acuities were 6/15 (20/50) or better preoperatively (unpublished data).

Several factors may account for the high success rate for patients with retinal detachments and spared maculas. The detachments are generally less extensive,

and thus there is less likelihood of missing a hole at surgery. Detachments that have minimal or no macular involvement may be those with little tendency to develop proliferative changes in the vitreous and the periretinal tissue. In relation to retinal detachments that include the macula, detachments with spared maculas are more likely to have demarcation lines (27% vs 18%). Benson, Nantawan, and Morse[15] believed that retinal detachments with demarcation lines represented a "less malignant" form of retinal detachment, as they noted only one failure in 66 cases. Retinal detachment had existed longer than one month in 43% of eyes with spared maculas and in 34% of eyes with macular involvement. Eyes with macular attachment, in spite of a relatively long duration of peripheral detachment, may fall into a more benign group; that is, they are less likely to have dynamic proliferative factors leading to extension of detachment, macular pucker, or ultimate failure. Nevertheless, other factors must account for the high success rate among the 72% of eyes without demarcation lines and the 57% of eyes with detachments of relatively short duration. We noted a lower incidence of aphakia in eyes with rhegmatogenous retinal detachment and spared maculas, a finding also observed by Norton[12] and Ashrafzadeh and associates.[16] Although one of our failures was in an aphakic eye, the low incidence of aphakia cannot explain our favorable results, since aphakia has not generally been associated with a reduced anatomic success rate or reduced visual acuity.[6,12,17] Patients with spared maculas tended to be younger than patients with macular detachment. This difference suggests that extension of a retinal detachment to involve the macular retina is more likely to occur quickly in older patients.

Our analysis of surgical technique indicates that retinal detachment with spared macula was less often managed by an encircling procedure and release of subretinal fluid than was detachment with macular involvement. We believe, however, that the variations of surgical technique were dictated by the character of the retinal detachments. Hence, we cannot relate the high rates of anatomic reattachment to the use of a lesser amount of surgery.

Because most patients with rhegmatogenous retinal detachment and spared maculas have excellent visual acuities preoperatively, the preservation of this visual acuity is important. The overall functional results in this series were favorable, as 150 eyes (87%) had visual acuities of 6/15 (20/50) or better postoperatively compared with 153 (88%) preoperatively. However, 18 eyes (10%) had a decrease in central visual acuity of more than two lines after the operation. This decrease was partially offset by the 15 eyes (9%) that had postoperative improvement of more than two lines in visual acuity. Of the eyes that had a significant decrease in visual acuity postoperatively, two were anatomic failures because of massive periretinal proliferation, three developed macular detachment before a second operation reattached the retina, six developed significant preretinal membrane in the macula, and two had pigment clumping under the macula. The latter two eyes may have had macular involvement that was not recognized initially, or they may have developed macular detachment between the time of the initial evaluation and surgery, for it is unlikely that pigment would migrate under an attached retina.[18] Hilton[19] concluded that migration of pigment under the macula was not detrimental to visual recovery. Our results do not contradict that conclusion.

Macular changes causing functional visual loss may be related to surgical trauma.[11,20] Hagler and Aturaliya[21] com-

mented that the development of postoperative pucker might be related to the amount of cryotherapy or diathermy used or to the number of operations performed. Davies and Gundry[22] indicated that eight of 104 eyes had less than average recovery in visual acuity because of excessive cryotherapy. Although Robertson and Priluck[23] related cryotherapy to preretinal membrane formation if the inner limiting lamina of the attached retina was disrupted by many heavy cryoapplications, an analysis of eyes receiving more than 50 cryoapplications in this study revealed that 14 of 16 had postoperative visual acuities of 6/15 (20/50) or better. The two eyes that received more than 50 cryoapplications and had final visual acuities worse than 6/15 (20/50) both had preoperative visual acuities poorer than 6/15 (20/50). Of the 16 eyes receiving large numbers of cryoapplications, only one had a decrease in visual acuity of more than two lines after the operation. In this one eye, visual acuity decreased from 6/6 (20/20) to 6/12 (20/40) as a result of vitreous debris. We saw neither preretinal membrane nor cystoid edema, although we did not specifically study the macula with fluorescein angiography. These observations indicate that the judicious use of large numbers of cryoapplications for repair of rhegmatogenous retinal detachment is unlikely to be associated with macular complications.

Summary

Among 173 patients who had rhegmatogenous retinal detachment with spared maculas, a high percentage achieved anatomic and functional success. We reattached 171 (99%) of the retinas; 150 (87%) of the eyes had final visual acuities of 6/15 (20/50) or better. Eighteen eyes (10%) had final postoperative visual acuities reduced more than two lines from the preoperative level. This reduced visual acuity was primarily related to postoperative changes occurring in the macula. The prognosis both for anatomic reattachment and visual acuity after scleral buckling for rhegmatogenous retinal detachment is significantly better when the macula is spared than when it is not. Large numbers of cryoapplications were not associated with macular complications.

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Ophthalmic miniature

Eye ailments

Put a few drops of castor oil in eye.

A sty can be removed by running the tip of a black cat's tail over it.

Edited by Eliot Wigginton, *The Foxfire Book*
New York, Anchor Press, 1972

PERIPHERAL PROLIFERATIVE RETINOPATHY WITHOUT OXYGEN THERAPY IN A FULL-TERM INFANT

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In a newborn infant, peripheral retinal neovascularization is most often seen with oxygen-induced retrolental fibroplasia. However, investigators have noted similar fundus changes,[1] apparently unrelated to increased arterial oxygen levels from supplemental oxygen therapy, in patients with anencephaly,[2-4] congenital anomalies,[2,5] cyanotic heart disease,[2,6,7] incontinentia pigmenti,[8] and familial exudative vitreoretinopathy.[9-15] We recently examined a healthy 4-month-old child who had fundus changes consistent with retrolental fibroplasia. However, the infant had been full-term and had received no supplemental oxygen therapy.

CASE REPORT

A 2,948-g boy was born Feb. 28, 1979, by spontaneous vaginal delivery at 40 weeks' gestation. The pregnancy had been uncomplicated. The mother was a 19-year-old primigravida. The only medications taken during pregnancy were vitamins with iron.

Results of a physical examination of the child after birth were normal. Laboratory test results, including a complete blood cell count, were also unremarkable. The Apgar score was 8 at one minute and 9 at five minutes. On the fifth day after birth the infant went home.

On March 17, 1979, the infant was admitted to a local hospital. He had a fever and was irritable. Except for a temperature of 39.8°C on admission, results of the physical examination were normal, with no localized signs of sepsis. Blood cultures were taken and the infant was given ampicillin, 100 mg/kg/day intravenously, and gentamicin, 6 mg/kg/day intramuscularly. Results of a lumbar puncture were normal. On the following day, the fever was reduced, and antibiotics were discontinued when the blood, urine, and cerebrospinal fluid cultures were found to be negative. He was discharged on March 23, 1979.

The infant was first seen by an ophthalmologist at 4 months of age for evaluation of "jerky eye movements" his mother noted soon after his birth. Ocular examination demonstrated pendular nystagmus with unsteady central fixation bilaterally. After instillation of atropine, refraction was $-8.50 + 1.50 \times 180$ degrees in the right eye and -6.50 in the left eye. The fundus was not well visualized, but the physician noted areas of retinal neovascularization in the temporal periphery of each eye. He made a tentative diagnosis of retrolental fibroplasia.

On July 9, 1979, at age 4½ months, the patient was admitted here. Results of a physical examination were unremarkable, except for nystagmoid eye movements. Measurements of weight, length, and head diameter were at the 50th percentile. All developmental milestones were within normal limits. An examination the next day with the child under anesthesia disclosed intraocular pressures by Schiøtz tonometry of 17.3 mm Hg bilaterally. The anterior segments were normal.

Ophthalmoscopic examination of the right eye showed an atrophic macular scar with moderate temporal displacement of the macula. The retina was totally avascular in the far periphery (Fig. 1). There was a shelf of elevated neovascular tissue temporally at the margin between the posterior perfused vascular bed and the anterior avascular retina. A circumferential vitreous band was present posterior to the neovascularization (Fig. 2). Elsewhere the retinal vasculature terminated well posterior to the ora serrata. Examination of the left fundus disclosed fine granular pigmentation in the posterior pole and wrinkling of the internal limiting membrane (Fig. 3). The retinal vessels terminated abruptly at a ridge of neovascular tissue in the temporal periphery. Fluorescein angioscopy disclosed leakage of dye from neovascular tissue in the temporal periphery of each eye.

Complement-fixation antibodies for herpes simplex virus and cytomegalovirus were negative. The indirect fluorescent antibody test for toxoplasmosis was negative. The rubella hemagglutination antibody reciprocal titer was 20 on June 19 and July 10; a skull X-ray series was normal.

The patient's parents were unmarried and he had no siblings. Results of an ocular examination, including careful ophthalmoscopic examination of the mother, were completely normal. The father and other family members were unavailable for examination.

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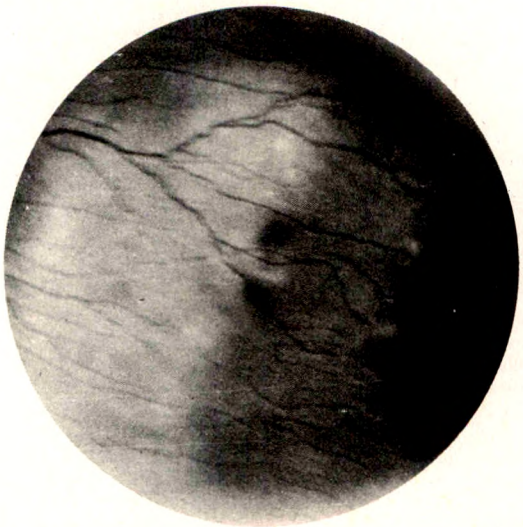


Fig. 1 (Schulman, Jampol, and Schwartz). Nasal periphery of right eye showing abrupt termination of the retinal vascularization (arrows).



Fig. 2 (Schulman, Jampol, and Schwartz). Temporal midperiphery of the right eye showing a circumferential vitreous band posterior to neovascular ridge.

Discussion

Retrolental fibroplasia, a disorder of prematurity associated with oxygen administration, was initially described by Terry[16] in 1942. He noted opaque retrolenticular membranes in a 6-month-old twin, born two months prematurely. Terry attributed the changes to a persistence of the tunica vasculosa lentis. Kinsey and Zacharias[17] were among the first to suggest the possible role of oxygen in the evolution of retrolental fibroplasia. The relationship between oxygen administration, prematurity, and retrolental fibroplasia was firmly established by a cooperative study in 1956.[18]

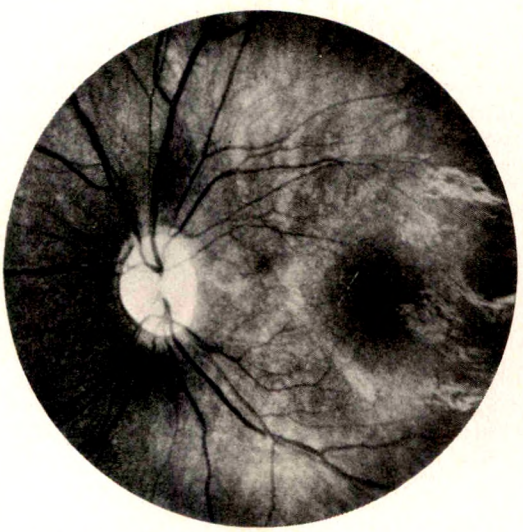


Fig. 3 (Schulman, Jampol, and Schwartz). Posterior pole of left eye shows distortion of internal limiting membrane reflex. Macula in this eye was not displaced.

Ashton, Ward, and Serpell[19] developed an animal model of retrolental fibroplasia in the newborn kitten in 1953. The retina of the newborn kitten is incompletely vascularized and comparable in development to that of the premature human infant. Exposure to a high concentration of oxygen in the newborn kitten resulted in vasoconstriction of retinal arterioles and obliteration of developing retinal vessels. Hypoxia followed withdrawal of this supplemental oxygen, leading to vascular proliferative changes identical to retrolental fibroplasia in the human. In contrast, exposure to oxygen in the fully vascularized retina of more mature kit-

tens resulted in only transient vasoconstriction.[20]

Cogan,[21] using a trypsin-digest technique on human eyes, demonstrated that vascularization of the nasal periphery of the retina is completed at eight months' gestation, but the temporal periphery is not vascularized until after birth. This developmental difference explains the greater susceptibility of the temporal retina to the development of retrolental fibroplasia. Since the temporal periphery is still immature at birth, oxygen administration might result in retinal damage. This factor may explain the occasional cases of retrolental fibroplasia reported in full-term infants.[22]

Owens and Owens[23] have suggested that vitamin E deficiency plays a role in the development of retrolental fibroplasia. Recently, Johnson, Schaffer, and Boggs[24] demonstrated a decrease in the incidence and severity of retrolental fibroplasia in a study of 173 low-birthweight infants who received vitamin E in addition to supplemental oxygen therapy. Phelps and Rosenbaum[25] also demonstrated that vitamin E has a protective effect against retrolental fibroplasia in kittens. Since our patient was not premature and did not have malabsorption syndrome, there is no reason to suspect vitamin E deficiency as a cause for his ophthalmic findings.

Evidence indicates that retrolental fibroplasia may occasionally occur in the absence of high levels of arterial oxygen tension. Foos[2] briefly described a premature infant with a patent ductus arteriosus. The child was given 30% to 60% oxygen for 22 days. Her left-to-right shunt maintained arterial oxygen levels near 50 mm Hg, with the highest recorded level at 91 mm Hg. After surgical correction of the patent ductus arteriosus, the patient was found to have retrolental fibroplasia. Foos proposed that the reversal of any condition that interferes with the oxygen-carrying capacity of blood or that causes tissue hypoxia may result in a "relative hyperoxia" capable of initiating the vascular changes characteristic of retrolental fibroplasia. Other similar cases have been documented.[6,7]

Bruckner[26] has also reported the development of retrolental fibroplasia without oxygen administration in a premature cyanotic infant whose mother was anemic. Patz,[27,28] studying this case and other occurrences of retrolental fibroplasia in the absence of supplemental oxygen, emphasized that in utero the ductus arteriosus and foramen ovale are patent, resulting in a mixing of arterial and venous blood. Spontaneous closure of the ductus arteriosus and foramen ovale at birth increases arterial oxygen tension. He suggested that this increase in oxygen tension may be sufficient in some infants to cause retinal vasoconstriction, vaso-obliteration, and, subsequently, retrolental fibroplasia. He also speculated that other reported cases of retrolental fibroplasia in the absence of supplemental oxygen therapy may be the result of this normal rise in arterial oxygen tension. At birth, the oxygen pressure of the newborn infant receiving ambient oxygen increases from 50 to 90 mm Hg. Perhaps this rise is responsible for some cases of retrolental fibroplasia.

Retrolental fibroplasia has also been reported in a full-term infant who had no exposure to oxygen but received a double-volume exchange transfusion for hyperbilirubinemia.[29] Because fetal hemoglobin has a greater affinity for oxygen than adult hemoglobin, an exchange transfusion using adult blood causes the oxygen dissociation curve to shift to the right. This results in greater delivery of oxygen to all tissues, including the retina. This factor may have contributed to the development of retrolental fibroplasia in this child.

Addison, Font, and Manschot[3] suggested a similar mechanism to explain the reported changes similar to those of retrolental fibroplasia found in infants with anencephaly.[2-4] They noted that the retina in an anencephalic infant is incompletely developed and nonfunctional. They suggested that this decreased metabolic activity may result in a state of relative hyperoxia sufficient to initiate vasoconstriction of the still immature retinal vessels.

Retrolental fibroplasia has rarely been documented in healthy full-term infants in the absence of supplemental oxygen. Stefani and Ehalt[30] described 15 patients with non-oxygen-induced retinal proliferation, but many of these patients clearly had other conditions (including Norrie's disease, Coats' disease, and trauma). Brockhurst and Chishti[31] described six adult patients with retrolental fibroplasia who had not received oxygen in infancy. Three of these patients had been full-term infants with no associated birth defects.

Our patient represents a well-documented case of apparent retrolental fibroplasia occurring in a healthy infant in the absence of supplemental oxygen. He was born at 40 weeks' gestation and had a birthweight of 2,948 g. His Apgar scores were normal. He had no associated congenital anomalies or birth defects, and did not receive an exchange transfusion. Two hereditary conditions resemble retrolental fibroplasia, however, and we considered them when making our diagnosis: incontinentia pigmenti and familial exudative vitreoretinopathy.

Incontinentia pigmenti[8] is an X-chromosome-linked dominant skin condition, usually fatal in males. Patients with this condition show cutaneous, dental, and ocular abnormalities. Patients may have peripheral capillary nonperfusion and proliferative retinopathy with preretinal fibrotic tissues. It is unclear from published reports at what age these changes can first be detected. Two children, aged 2 years, had temporal peripheral vascular disease,[8] and similar changes have also been reported in a 4-week-old child.[32] If these changes are present in a newborn infant, they could certainly be confused with retrolental fibroplasia. The absence of skin disease, however, rules out this condition in our patient.

Criswick and Schepens[9] in 1969 described a condition called familial exudative vitreoretinopathy, which clinically may resemble retrolental fibroplasia, in six members of two families. Gitter and associates[10] later described nine members of a single family with similar findings. Several recent reports[11-15] have described similar kindreds. This disorder, unlike retrolental fibroplasia, is dominantly inherited. The expression of this disorder appears to be variable, and the penetrance of the condition is not as yet certain.

Clinical features of familial exudative vitreoretinopathy may include heterotopia of the macula, posterior vitreous detachment, organized vitreous membranes in the periphery, snowflake-like vitreous opacities, retinal traction, retinal breaks, retinal folds, peripheral retinal neovascularization, and peripheral capillary nonperfusion. These changes may be indistinguishable from those of retrolental fibroplasia. Ober and associates[15] noted that careful examination of the peripheral fundus may be necessary to recognize mild cases of this disorder. Patients with familial exudative vitreoretinopathy are, like our patient, usually full-term infants who have had no exposure to oxygen. Patients as young as 1½ years of age have shown ocular changes,[9] and one patient was described as "blind from birth."[11] (We have seen an infant with familial exudative vitreoretinopathy and the changes were very similar to those of the present case.) The absence of

any family history of ocular disorders makes the diagnosis of familial exudative vitreoretinopathy in our patient less likely. Although the patient's mother had a normal ocular examination, our inability to examine the father and other family members makes it possible that our patient has a sporadic type of familial exudative vitreoretinopathy.

Oxygen toxicity alone cannot explain all cases of peripheral proliferative retinopathies in newborn infants. The occurrence of retrolental fibroplasia in low-birthweight cyanotic infants with congenital heart defects[2,6,7] is good evidence that arterial oxygen concentration does not have to be increased. Furthermore, many infants (including low-birthweight infants) exposed to high oxygen concentrations do not develop retrolental fibroplasia.[33] Additional work is needed to identify and define further all the factors involved in the pathogenesis of the changes in retrolental fibroplasia. Additionally, we should remember that previous reports describing cases of retrolental fibroplasia in healthy full-term infants, without supplemental oxygen, could also represent sporadic cases of familial exudative vitreoretinopathy. Careful family studies should be done on all such infants.

Summary

A 2,948-g full-term male infant, the product of an uncomplicated pregnancy, developed fundus changes consistent with retrolental fibroplasia in the absence of supplemental oxygen therapy. He had no associated illnesses or congenital anomalies, and had not received exchange transfusions. There was no family history of ocular disease.

Changes similar to those of retrolental fibroplasia do occasionally occur in full-term infants who have had no supplemental oxygen therapy. A relative hyperoxia caused by the increase in arterial oxygen saturation occurring at birth is one possible explanation for these events. This rise, plus susceptibility factors apart from prematurity, may account for these unusual cases. Additionally, these cases may represent sporadic examples of familial exudative vitreoretinopathy.

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PHOTOCOAGULATION TREATMENT OF PROLIFERATIVE RETINOPATHY SECONDARY TO A CAROTID-CAVERNOUS FISTULA

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Fistulas between the carotid artery and cavernous sinus, whether traumatic or spontaneous in origin, usually come to the attention of the ophthalmologist because of ocular complications such as proptosis, conjunctival chemosis, tearing, and diplopia.[1-3] Kalina and Kelly[4] recently described the development of optic disk neovascularization in two patients with carotid-cavernous fistulas who subsequently developed vitreous hemorrhage. We have also examined a patient with a carotid-cavernous fistula who developed disk neovascularization. Treatment with scatter-type argon laser photocoagulation produced virtually complete regression of the neovascularization.

CASE REPORT

A 48-year-old woman suffered mild head trauma in 1965. She was subsequently well until 1967 when she noted a roaring noise in her head associated with proptosis and tearing of the left eye. Cerebral angiography performed at another hospital showed a left carotid-cavernous fistula fed through the external and internal circulations.

In June 1969, artificial embolization of the fistula through the external carotid artery was performed and the external carotid was ligated at its origin. Postoperatively, the patient noticed a decreased intensity of the bruit and decreased tearing but there was continued arterialization of the conjunctival vessels and persistent exophthalmos. The fistula was presumed to be still patent but no further neurosurgical procedure was recommended. The patient was subsequently followed up at regular intervals by her ophthalmologist.

In May 1978, abnormal vessels on the surface of the left optic disk were first noted and the patient was referred here. Visual acuity was R.E.: 6/6 (20/20)-1 and L.E.: 6/7.5 (20/25)-2. Visual fields were full bilaterally. Hertel measurements were 11 mm on the right and 16 mm on the left with a base of 100 mm. Extraocular movements were full. There was marked dilation and arterialization of the conjunctival vessels of the left eye. Slit-lamp examination showed a normal anterior chamber on the right. On the left, the cornea and anterior chamber were normal but the iris vessels were dilated and leaked fluorescein (Fig. 1). Ophthalmoscopy in the right eye showed a normal disk, vessels, macula, and periphery. The left disk, however, was hyperemic with neovascularization extending into the vitreous (Fig. 2). Some retinal vessels were sausage-shaped and most were markedly dilated. The fluorescein angiogram showed that venous filling did not begin until 26 seconds after dye injection (Fig. 3, left). The late-phase angiogram showed leakage from the disk neovascularization (Fig. 3, right).

To reduce the likelihood of visual loss caused by vitreous hemorrhage from the neovascularization, scatter-type argon laser photocoagulation was instituted. Approximately 1,000 applications of energy were placed throughout the left fundus in two treatment sessions, one week apart. Spot size was 500 μ; intensity, 1,000 mW.; and duration of exposure, 0.1 seconds. The area adjacent to the disk and

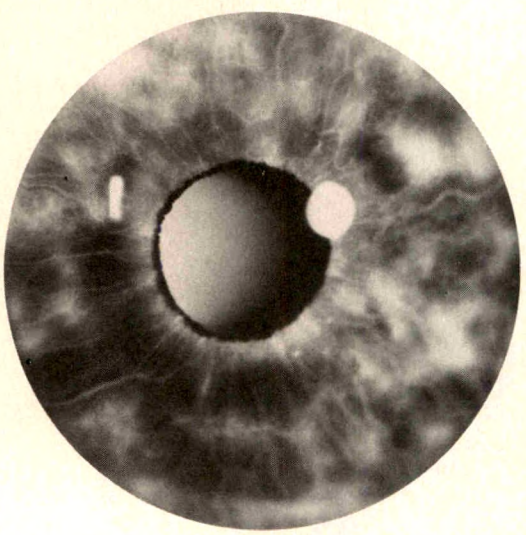


Fig. 1 (Harris, Fine, and Miller). Pretreatment iris angiogram shows late leakage from dilated tortuous surface vessels.

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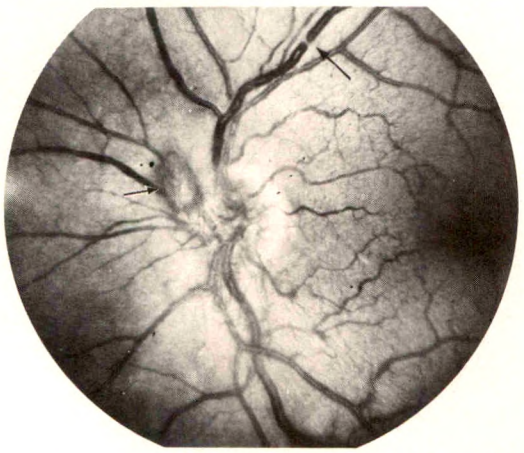


Fig. 2 (Harris, Fine, and Miller). Note disk neovascularization (small arrow) extending into the vitreous and tortuous beaded retinal veins (large arrow).

macula was left untreated. Within two months after treatment, the neovascularization began to regress (Fig. 4). Subsequent evaluation 17 months after treatment showed less disk hyperemia and essentially complete regression of the new vessels with only fibrous remnants remaining (Fig. 5). Fluorescein leakage from the iris vessels was also dramatically reduced (Fig. 6). Results of the patient's examination were otherwise unchanged; the subjective bruit was still present and the conjunctival vessels remained dilated and arterialized. Ophthalmodynomometric measurements were consistent with a patent left carotid-cavernous fistula. Fluorescein angiography showed persistent late leakage from the disk, a common finding even when disk vessels have disappeared on clinical ophthalmoscopic examination.

Discussion

Carotid occlusive disease has been associated with a stasis retinopathy consisting of microaneurysms, retinal hemorrhages, dilated tortuous veins, and disk neovascularization.[5-7] In one patient with carotid stenosis, panretinal photocoagulation produced regression of disk neovascularization and rubeosis iridis.[7] Regression of the retinopathy has also been reported after carotid endarterectomy.[6] A similar stasis retinopathy with neovascularization may occur after retinal venous obstructive disease. A common mechanism may underlie the development of new vessels in these conditions as well as in cases of carotid-cavernous fistulas. Blood flow through the retinal capillary bed is dependent upon the retinal arteriovenous pressure gradient. A decreased gradient, caused by either decreased arterial pressure or increased venous pressure, embarrasses the retinal circulation and may result in ocular

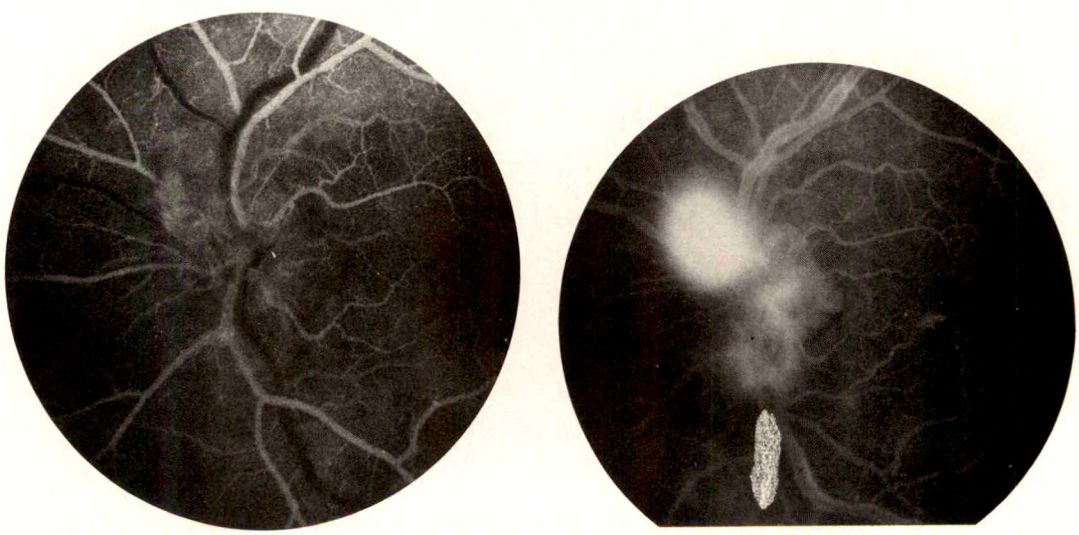


Fig. 3 (Harris, Fine, and Miller). Left, The early phase of venous filling began 26 seconds after dye injection. Right, Later in the angiogram. Note leakage from disk neovascularization.

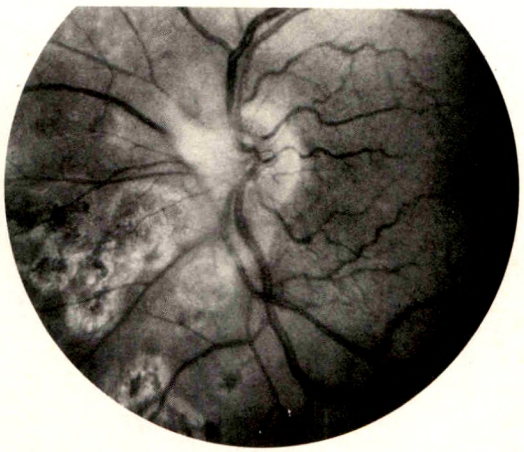


Fig. 4 (Harris, Fine, and Miller). Two months after treatment. Note regression of neovascularization and reduction of disk hyperemia.

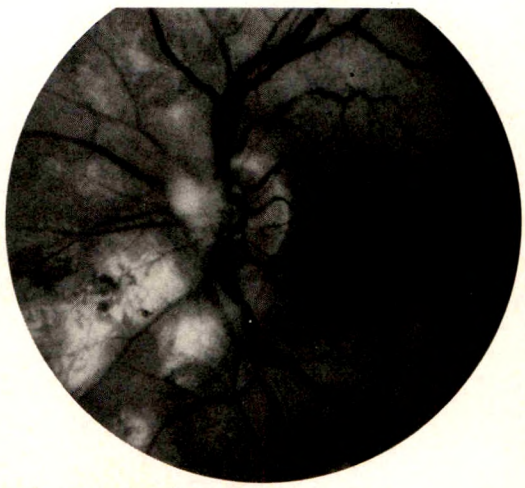


Fig. 5 (Harris, Fine, and Miller). Seventeen months after treatment. Note further regression and fibrosis of disk neovascularization.

ischemia. Hypoxic retinal tissue then may liberate a vasoproliferative substance that stimulates the growth of new vessels.

In patients with carotid-cavernous fistulas, the retinal arteriovenous pressure gradient is decreased by a combination of reduced arterial pressure and increased venous pressure in the orbital venous bed.[1] Even though the fluorescein angiogram shows no capillary closure, the fistula may reduce retinal capillary perfusion and cause retinal ischemia. The ocular circulatory embarrassment may be increased by carotid ligations designed to diminish the flow through the fistula. [1,2,8] Kalina and Kelly[4] described two patients in whom disk neovascularization developed after carotid ligations for carotid-cavernous fistulas. They attributed the proliferative retinopathy to ocular ischemia produced by the surgery. In our patient, no manipulations of the internal carotid arteries were performed, and it is unlikely that the embolization procedure through the external carotid could further embarrass retinal perfusion; in fact, it may have partially closed the fistula, thereby increasing carotid and ophthalmic artery perfusion pressure. Thus, unlike the patient reported by Kalina and Kelly,[4] in our patient the disk neovascularization probably resulted from retinal ischemia produced by the carotid-cavernous fistula itself.

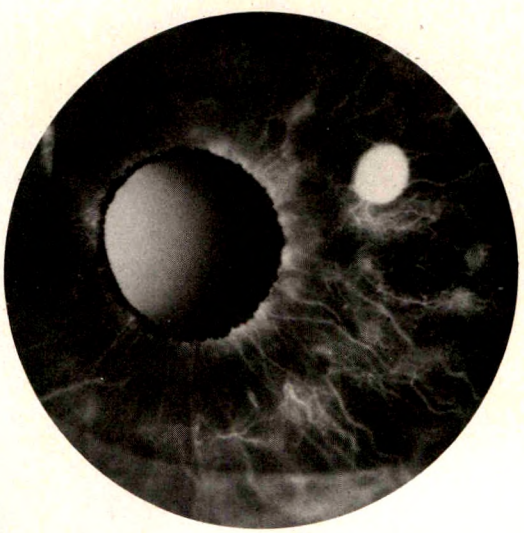


Fig. 6 (Harris, Fine, and Miller). Iris angiogram 17 months after treatment shows reduced leakage.

Scatter-type argon laser photocoagulation was successful in promoting regression of the disk neovascularization and rubeosis iridis. To our knowledge, the

rapid involution and fibrosis of the new vessels represents the first reported case of successful treatment of neovascularization associated with a carotid-cavernous fistula. We believe that neurosurgical procedures to close the fistula are not indicated if the sole indication is the development of stasis retinopathy and neovascularization. The new vessels on the disk probably reflect retinal ischemia and, therefore, should respond to the scatter-type photocoagulation used in the treatment of diabetic retinopathy.

Summary

Neovascularization of the optic disk occurred in a patient with a carotid-cavernous fistula. Treatment with scatter-type argon laser photocoagulation successfully promoted regression of the new vessels.

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PREGNANCY AND DIABETIC RETINOPATHY

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Diabetic retinopathy is one of the long-term complications of juvenile-onset diabetes that is more likely to occur as the duration of diabetes increases. This problem usually makes its appearance about 15 years after the onset of diabetes, and thus can be expected in young women 20 to 30 years of age who developed diabetes when they were 5 to 15 years old. Since the years between ages 20 and 30 are also the peak child-bearing years the onset or progression of diabetic retinopathy during a pregnancy may be coincidental or it may be related to the dramatic endocrinologic and metabolic changes associated with pregnancy.

Pregnancy and diabetes certainly complicate each other in many ways. The severity of the complications increases with the duration of the diabetes and the lack of consistent control.[1] In recent years, however, advances in the treatment of juvenile-onset diabetes and obstetric care have allowed many more young diabetic women to reach their child-bearing years with reasonably good health and the capability of becoming pregnant.[2] There are still risks of fetal mortality and of morbidity for both mother and child, but they are decreasing.[3] Beetham,[4] in 1950, described the results of 18 pregnancies in 12 women with proliferative diabetic retinopathy, and reported that none of these patients had a successful pregnancy while maintaining useful visual acuity. Since that time, photocoagulation and other procedures for the treatment of diabetic retinopathy have become available and have altered this dismal prognosis.

A prospective study to prove a causal relationship between pregnancy and deterioration of diabetic retinopathy would be practically impossible to design. We have, however, collected data on the behavior of diabetic retinopathy during pregnancy and the effect of photocoagulation treatment in 30 patients. We selected records of 30 women with juvenile-onset diabetes who had never been pregnant and attempted to match each woman in the pregnant group as closely as possible with another patient for age and duration of diabetes. These retrospective data, covering a 20-year period, provide valuable information about the visual prognosis for these prospective young mothers with diabetic retinopathy, and they provide a basis for evaluating the traditional obstetric recommendation of therapeutic abortion for patients with diabetic retinopathy.

SUBJECTS AND METHODS

Thirty patients with juvenile-onset diabetes had repeated complete ophthalmoscopic evaluations, including fundus photography, during and after a total of 35 pregnancies. In some cases the initial ocular studies were done before pregnancy, and in others at various times during pregnancy. These patients were not selected from the diabetic population at random; they were referred here because of a tentative diagnosis of diabetic retinopathy. They make up a small percentage (0.5%) of the total population of patients with diabetic retinopathy seen here between 1961 and 1980. Only pa-

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tients observed to the end of pregnancy and for at least six months after (mean follow-up was five years) were included in this study. Their ages ranged from 20 to 35 years at the time of conceiving, with a mean of 25 years; the duration of diabetes ranged from 7 to 34 years, with a mean of 17 years. Remarkably, seven patients were 30 years or older during pregnancy, and four had had diabetes for more than 20 years.

For the purpose of comparing the course of diabetic retinopathy and results of treatment without pregnancy, 30 patients were selected who had never been pregnant and who matched the pregnant group on the basis of age and duration of diabetes. Their ages ranged from 18 to 35 years, with a mean of 25 years. The duration of diabetes ranged from 11 to 31 years, with a mean of 17 years (Table 1). The nonpregnant women had retinopathy similar to that found in the pregnant group (Table 2), suggesting that the duration of diabetes is more important in determining the severity of ocular problems than the presence or absence of pregnancy.

In addition to similar ocular findings, the two groups were parallel in other ways, including the fact that seven patients in each group died of renal failure or myocardial infarction. The deaths in the nonpregnant group occurred one to seven years after their first retina consultations, with a mean of four years; in the pregnant group deaths occurred two to nine years after pregnancy, with a mean of five years.

TABLE 1

COMPARATIVE DATA FOR THE TWO GROUPS

Data	Pregnant Patients	Nonpregnant Patients
Age (yrs)		
Range	20-35	18-35
Mean	25	25
Duration of diabetes (yrs)		
Range	7-34	11-31
Mean	17	17
Follow-up (yrs)		
Range	1-13	1-15
Mean	5	5

TABLE 2

CLASSIFICATIONS FOR 60 PATIENTS (120 EYES)

Classification	Pregnant Patients*	Nonpregnant Patients
B	21	19
I	5	5
II	16	20
III	10	12
IV	7	4
D	1	0

*Patients with two pregnancies were classified at the time of the second pregnancy.

Classification of diabetic retinopathy—Each eye was classified at each examination by the Okun-Cibis system.[5] This classification system quickly and concisely conveys the essential information.

"B" indicates background or simple retinopathy with no preretinal proliferation. The degree of fibrovascular proliferation present is designated as follows:

I, three or fewer areas of surface neovascularization or 3 disk diameters of proliferation;

II, four or more such areas, or the presence of flat disk neovascularization;

III, elevated proliferation into the vitreous cavity less than 4 disk diameters in size; and

IV, proliferation into the vitreous more than 4 disk diameters in extent.

"D" indicates dense hemorrhage totally obscuring the fundus.

Treatment—Xenon photocoagulation was performed under retrobulbar anesthesia in most cases. Argon laser photocoagulation was also performed as supplementary therapy in a few patients. The exact techniques have been described elsewhere.[6] No attempt was made to

standardize treatment for the patients in this study, and treatment was administered as frequently and extensively as the condition of the eye warranted. There were no adverse effects on the pregnancies in those patients who were treated with photocoagulation. Two patients in each group underwent pituitary ablation as their initial therapy.

RESULTS

Pregnant group—Thirteen of the pregnancies were interrupted before the third trimester, nine by spontaneous abortion and four by therapeutic abortion. Twenty-one patients gave birth to live infants (20 by cesarean section); one stillborn infant was delivered by cesarean section.

Many changes occurred in the retinopathy during pregnancy. Most eyes not treated with photocoagulation before pregnancy deteriorated in both visual acuity and grade of classification; these eyes were therefore treated. Some of the eyes treated before pregnancy also deteriorated, but generally to a lesser degree. In considering the changes in the eyes during pregnancy, it is important to differentiate between those with background retinopathy and those with proliferative retinopathy. One must also distinguish patients treated before they became pregnant (whose classification may have been converted from proliferative to background retinopathy by treatment) from those not treated before conceiving.

BACKGROUND RETINOPATHY IN BOTH EYES—Eight patients had background retinopathy in both eyes before pregnancy. One of these patients had had proliferative retinopathy (Class II) in both eyes two years before conceiving. The proliferative retinopathy had been successfully treated and converted to background retinopathy. Her pregnancy was terminated by spontaneous abortion at eight weeks of gestation, and no deterioration in her retinal condition occurred. Another patient had had background retinopathy which had not changed with her first pregnancy, but which deteriorated to proliferative retinopathy two years later before a second pregnancy. The other six women all experienced deterioration of their retinopathy which changed from background to proliferative stages during pregnancy.

BACKGROUND RETINOPATHY IN ONE EYE—Five patients had background retinopathy in one eye and proliferative retinopathy in the other at the time of conceiving. One patient was unusual in that she had had transphenoidal pituitary ablative surgery four years before conceiving in an effort to ameliorate florid proliferative retinopathy. The pituitary ablation was obviously incomplete and one eye was treated with extensive photocoagulation, which eliminated the proliferative tissue and converted the proliferative to background retinopathy. The untreated eye, which had remained stable after the pituitary surgery, deteriorated and developed vitreous hemorrhage in the last trimester of pregnancy, while the treated eye maintained its "B" classification. Two other eyes in this group had also been converted from the proliferative stage to background retinopathy before pregnancy by photocoagulation, and no deterioration occurred. The remaining two eyes (untreated) both deteriorated from background to proliferative retinopathy during pregnancy.

Fifteen of the 21 eyes in the B classification progressed to proliferative retinopathy during pregnancy; of the six that did not, five had been previously treated with photocoagulation.

These data indicate an extremely poor prognosis for background retinopathy during pregnancy. However, these cases cannot be used to assess the frequency of conversion from background to proliferative retinopathy during pregnancy since they were all selected cases in which the

referring physician had noted retinopathy severe enough to warrant referral for possible treatment.

Twenty-seven patients (49 eyes) had proliferative retinopathy at the time of conceiving. Of these, 14 had already been treated with photocoagulation in at least one eye before pregnancy. A total of 24 eyes had undergone photocoagulation before pregnancy; seven of these deteriorated during pregnancy (seven of 24 or 29%). Of the 30 not treated before pregnancy, 26 (87%) deteriorated. In one patient whose eyes had not deteriorated during pregnancy, the proliferative retinopathy worsened nine months after delivery while she was undergoing dialysis for renal failure. The other three patients all had avascular inactive fibrotic retinitis proliferans, which had not changed since their pregnancies.

Of the patients who had visual acuities of 6/12 (20/40) or better at the last examination, ten had interrupted (ten of 13 or 77%) and 15 uninterrupted pregnancies (15 of 22 or 68%).

Of the 49 eyes with proliferative retinopathy, 33 (67%) deteriorated during pregnancy; 26 were treated with photocoagulation but seven were considered too far advanced for treatment. Of 26 treated eyes, 22 (85%) were considered successes on the basis of elimination of neovascular tissue and stabilization of vision.

The total group of 27 patients with proliferative retinopathy included only three patients who became blind during or immediately after their pregnancies. The vast majority have useful visual acuities, 22 of the 27 patients (81%) with proliferative retinopathy have visual acuities of 6/12 (20/40) or better in at least one eye (Tables 3 and 4).

The final visual outcome was essentially the same in the women who had interrupted pregnancies as in those who carried babies to full term (Table 5). Four of the five patients who had two pregnancies maintained a visual acuity of 6/12 (20/40) in at least one eye.

TABLE 3

VISUAL ACUITIES OF 60 PATIENTS

Visual Acuity*	Pregnant Patients	Nonpregnant Patients
6/12 (20/40) or better		
Initial	24	27
Latest	23	24
6/15 to 6/30 (20/50 to 20/100)		
Initial	6	3
Latest	2	4
6/90 to 6/120 (20/200 to 20/400)		
Initial	0	0
Latest	2	0
Counting fingers		
Initial	0	0
Latest	3	2

*In at least one eye.

Nonpregnant group—Eight patients had background retinopathy in both eyes when first examined; four did not deteriorate to proliferative retinopathy during the follow-up period. All eight eyes in the remaining four patients developed proliferative retinopathy within three years and required photocoagulation.

Three patients had background retinopathy in one eye and proliferative

TABLE 4

VISUAL ACUITIES OF 6/12 (20/40) OR BETTER AT LATEST EXAMINATION

Classification at Time of Treatment	No. of Eyes	
	Pregnant Patients	Nonpregnant Patients
B (not treated)	1	5
I	2	2
II	13	11
III	5	6
IV	2	0
Total	23	24

TABLE 5

VISUAL ACUITY RESULTS FOR PREGNANT PATIENTS

Pregnancies*	No. of Eyes	Visual Acuities of 6/12 (20/40) or Better†
Uninterrupted	22	
Initial		16
Latest		17
Interrupted	13	
Initial		12
Latest		10

*There were 35 pregnancies in 30 women.
†In at least one eye.

retinopathy in the other. All three of the eyes classified as B deteriorated to proliferative retinopathy within 18 months after the first examination.

Twenty-five patients (47 eyes) had proliferative retinopathy when first examined. All but two were treated with photocoagulation. The two exceptions had pituitary ablation surgery (four eyes). Two patients lost all useful vision in both eyes; 24 patients (80%) retained visual acuities of 6/12 (20/40) or better at the last follow-up visit.

DISCUSSION

Many questions can be raised about the advisability of pregnancy for a woman with juvenile-onset diabetes and retinopathy. There are medical, ethical, religious, and socioeconomic issues involved. White asked; "Why permit or encourage young women with diabetes to bear children?" She answered her own question: "To many, to nearly all, life lacks meaning without successful childbearing." After 45 years of experience with juvenile-onset diabetes, White recognized only two indications for termination of pregnancy in diabetes: proliferative retinopathy and renal failure.[7]

Cassar and associates[8] reviewed published reports on diabetic retinopathy and pregnancy and described 15 patients, four of whom had proliferative retinopathy. Two of these patients underwent photocoagulaion during pregnancy and maintained normal vision. Cassar and associates concluded that although diabetic retinopathy may progress during pregnancy, it is not an absolute contraindication, and the use of photocoagulation further improves the prognosis.

Hercules and associates[9] studied the course of proliferative diabetic retinopathy with disk neovascularization in 11 patients during and after 13 pregnancies. Even with minimal argon laser therapy, only 9% became blind; and none had visual acuities of less than 6/60 (20/200). They concluded that therapeutic abortion and sterilization cannot be justified for ophthalmic reasons.

Our experience with diabetic retinopathy in pregnancy is perhaps biased by our referral practice. The prognosis however, even in cases of proliferative diabetic retinopathy, is surprisingly good with the use of photocoagulation. This is particularly true for patients treated before conceiving, but applies even to those first treated during pregnancy. The comparison between pregnant and nonpregnant women with juvenile-onset diabetes shows that long-term visual efficiency is almost equal for the two groups.

A young woman with juvenile-onset

diabetes who wishes to have a child should plan to become pregnant as early as possible. A careful examination for diabetic retinopathy is necessary; if present, it should be treated before pregnancy. Patients with background retinopathy should be carefully observed during pregnancy; if proliferative retinopathy develops, it should be treated with photocoagulation.

Proliferative diabetic retinopathy is neither an absolute contraindication to pregnancy nor an absolute indication for therapeutic abortion. It is, however, still a serious threat to the mother and a probable cause of morbidity to both mother and child. Proliferative retinopathy, along with other long-term complications of diabetes, must be assessed in each individual patient. If the prospective mother does not have an intense desire for a child, proliferative diabetic retinopathy can be considered strong grounds for a therapeutic abortion.

Summary

Diabetic retinopathy can be expected to progress during the term of pregnancy. A retrospective study indicated that photocoagulation was successful in preserving vision in 70 to 80% of 30 pregnant diabetic patients as well as in a similar group of nonpregnant patients with juvenile-onset diabetes. Proliferative diabetic retinopathy, in and of itself, is not an absolute indication for therapeutic abortion.

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MASSIVE VITREOUS HEMORRHAGE AND SENILE MACULAR CHOROIDAL DEGENERATION

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Hemorrhagic detachment of the retinal pigment epithelium is a common occurrence in senile macular choroidal degeneration.[1-4] It can be difficult to distinguish this condition from malignant melanoma of the choroid associated with vitreous hemorrhage. We reviewed 13 cases of massive vitreous hemorrhage secondary to senile macular choroidal degeneration in order to determine the best means of differentiating between the two conditions.

SUBJECTS AND METHODS

This study included 13 patients, seven men and six women, who had massive vitreous hemorrhages dense enough to obscure details of the fundus during ophthalmoscopic examination. All the patients were treated between 1973 and 1978. None were younger than 60 years, and the average age was 74 years. We excluded all patients with vitreous hemorrhages secondary to causes other than senile macular choroidal degeneration (such as diabetic retinopathy, posterior vitreous detachment with or without retinal tears, and venous occlusion diseases).

We have selected one representative case (Table, Case 1) for detailed discussion. The Table contains clinical summaries for the other 12 patients.

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CASE REPORT

Case 1—An 83-year-old man had a six-month history of visual loss in the left eye. Visual acuity was 6/12 (20/40) in the right eye and light perception with inaccurate light projection in the left eye. There was a left afferent pupillary defect and a 15-diopter exotropia. Numerous drusen were present in the right posterior pole. A massive vitreous hemorrhage obscured all fundus detail in the left eye. Ultrasonography showed a posterior vitreous detachment, dense vitreous opacities, a retinal detachment with numerous opacities beneath it, and a large, broad-based, relatively flat mass in the posterior pole (Fig. 1, top left). A radioactive phosphorus (^{32}P) uptake test was negative.

Six months later, the eye had lost all light perception. Although ultrasonography showed fewer vitreous opacities, the lesion in the posterior pole was unchanged (Fig. 1, top right).

Three years later, the patient returned because of a sudden loss of visual acuity to the level of finger counting in his right eye, caused by a large hemorrhagic disciform macular detachment (Fig. 1, bottom left). Ultrasonography of the right eye showed a broad-based, relatively flat posterior pole lesion (Fig. 1, bottom right) similar to that previously demonstrated in the left eye. The left eye, which had developed a mature cataract with anterior synechiae and hemosiderosis of the anterior segment, now showed a funnel-shaped total retinal detachment by ultrasonography.

RESULTS

Ten of the 13 eyes with massive vitreous hemorrhage were left eyes. All 13 fellow eyes exhibited some degree of senile macular choroidal degeneration. Initially, nine showed drusen or pigmentary changes; two of these progressed to a hemorrhagic disciform detachment and one developed a serous detachment of the retinal pigment epithelium. Two had hemorrhagic disciform detachments, and two had early disciform scars when first seen.

Three of the patients had medically controlled hypertension (one had been

TABLE

SUMMARY OF PATIENT DATA

Case No., Age (yrs.), Sex	Vitreous Hemorrhage	Initial Visual Acuity	Medical History	Ultrasonography
1, 83, M	Left eye	R.E.: 6/12 (20/40) L.E.: LP	Noncontributory	Vitreous hemorrhage
2, 73, F	Left eye	R.E.: 6/9 (20/30) L.E.: H.M.	Noncontributory	Vitreous hemorrhage; vitreous adherence to posterior pole lesion
3, 79, F	Left eye	R.E.: 6/60 (20/200) L.E.: H.M.	Noncontributory	Vitreous hemorrhage; retinal detachment; posterior pole mass
4, 60, F	Left eye	R.E.: 6/60 (20/200) L.E.: 6/600 (2/200)	Noncontributory	Vitreous hemorrhage; posterior pole lesion
5, 71, F	Left eye	R.E.: 6/6 (20/20) L.E.: H.M.	Hypertension	Vitreous hemorrhage; posterior pole lesion
6, 84, M	Left eye	R.E.: H.M. L.E.: LP	Glaucoma; blastomycosis	Vitreous hemorrhage; posterior pole lesion
7, 72, F	Left eye	R.E.: 6/6 (20/20) L.E.: H.M.	Hypertension	Vitreous hemorrhage; posterior pole lesion
8, 78, M	Right eye	R.E.: NLP L.E.: 6/7.5 (20/25)	Noncontributory	Vitreous hemorrhage; retinal detachment; posterior pole lesion
9, 64, M	Right eye	R.E.: CF L.E.: 6/6 (20/20)	Noncontributory	Vitreous hemorrhage; posterior pole lesion
10, 75, M	Left eye	R.E.: 6/6 (20/20) L.E.: LP	Myocardial infarct 1970; anticoagulants; absolute glaucoma	Vitreous hemorrhage; total hemorrhagic retinal detachment; posterior pole lesion
11, 71, M	Left eye	R.E.: 6/9 (20/30) L.E.: CF	Laryngeal carcinoma	Not done
12, 83, F	Left eye	R.E.: 6/30 (20/100) L.E.: CF	Hypertension	Vitreous hemorrhage; posterior pole lesion
13, 68, M	Right eye	R.E: LP L.E.: 6/120 (20/400)	Noncontributory	Vitreous hemorrhage; posterior pole lesion

Fellow Eye Initially	^{32}P Test	Length of Follow-up	Final Visual Acuity	Follow-Up Findings	
				Initally Involved Eye	Fellow Eye
Drusen	25-56%	2 yrs	R.E.: CF L.E.: NLP	Dense cataract; total retinal detachment; choroidal mass lesion unchanged; vitreous not cleared	Hemorrhagic disciform detachment
Drusen	Not done	6 mos	R.E.: 6/12 (20/40) L.E.: CF	Vitreous cleared; disciform scar	Unchanged
Hemorrhagic disciform detachment	Not done	17 mos	R.E.: 6/120 (20/400) L.E.: HM	Vitreous cleared; disciform scar	Disciform scar; exudative retinal detachment
Disciform scar	Not done	6 mos	R.E.: 6/60 (20/200) L.E.: 6/60 (20/200)	Vitreous cleared; disciform scar	Unchanged
Drusen	Not done	1 yr	R.E.: 6/6 (20/20) L.E.: CF	Vitreous cleared; disciform scar	Retinal pigment epithelial detachment
Total glaucomatous cup; hemorrhagic disciform detachment	Not done	3 mos	R.E.: LP L.E.: LP	Vitreous cleared; disciform scar	Unchanged
Drusen	12%	1 yr	R.E.: 6/6 (20/20) L.E.: NLP	Total retinal detachment; disciform scar; synchisis syntillans; vitreous cleared	Unchanged
Drusen	Not done	7 mos	R.E.: NLP L.E.: 6/7.5 (20/25)	Cataract, vitreous hemorrhage; posterior pole lesion; retinal detachment	Unchanged
Drusen	Not done	3 yrs	R.E.: 6/21 (20/70) L.E.: 6/6 (20/20)	Eccentric disciform scar; vitreous cleared	Choroidal neovascular membrane; successfully photocoagulated
Drusen	Not done	Left eye enucleated	R.E.: 6/6 (20/20)	L.E.: Vitreous hemorrhage; total hemorrhagic retinal detachment	Unchanged
Drusen	25-34%	9 mos	R.E.: 6/9 (20/30) L.E.: NLP	Vitreous cleared; retinal detachment; disciform scar	Unchanged
Drusen	Not done	6 wks	R.E.: 6/30 (20/100) L.E.: CF	Resolving vitreous hemorrhage originating from disciform scar	Unchanged
Disciform scar	3%	2 wks	R.E.: CF L.E.: 6/120 (20/400)	Vitreous cleared; disciform scar	Unchanged

taking anticoagulants), two had cancer, one had blastomycosis, and one had glaucoma. The remaining six patients were otherwise healthy.

Sudden and profound visual loss was a common symptom in all patients. Twelve of the 13 eyes initially had visual acuities of finger counting or worse. One had visual acuity of 6/600 (2/200). One eye had no light perception when first examined and four lost light perception because of hemorrhagic retinal detachments three to 24 months later. The final visual acuities were finger counting to light perception in six eyes, 6/60 (20/200) in one eye, and 6/22 (20/70) in one eye that had an eccentric disciform scar.

Ultrasonographic and ophthalmoscopic findings—Ultrasonography disclosed posterior choroidal lesions in 12 eyes. We did not examine one patient by ultrasonography initially but later found a disciform scar by indirect opthalmoscopy after the vitreous hemorrhage had cleared. The lesions were shallowly elevated, broad-based, and ultrasonographically homogeneous with varying degrees of vitreous opacification overlying them (Fig. 1, top left). We noted a prominent posterior detachment of the opacified vitreous in most cases. In some cases we found a focal adherence of the detached vitreous to the surface of the posterior pole lesion (Fig. 2, top left), probably at the point where the hemorrhagic disciform detachment had broken through the retina into the vitreous cavity. In four cases, there was a retinal detachment overlaying the posterior pole lesion (Fig. 1, top left) at the initial examination; two other eyes developed retinal detachments during the follow-up period. On follow-up ultrasonographic examination, none of the lesions showed recognizable expansion in elevation or lateral extension (Figs. 1, top left, and 2, top right), nor did these lesions demonstrate the ultrasonographic pattern associated with malignant melanomas of the choroid (Fig. 3, left). We saw no choroidal excavation in any of these lesions.

In ten of the 13 eyes (77%), the vitreous hemorrhage cleared so that we could confirm the ultrasonographic diagnosis of a disciform macular scar ophthalmoscopically. In three of the six eyes with retinal detachments, the vitreous eventually cleared enough to allow ophthalmoscopic confirmation of the retinal detachments. We believed that all the retinal detachments were nonrhegmatogenous, secondary to the posterior pole lesion.

Radioactive phosphorus (^{32}P) uptake test—We performed ^{32}P uptake tests over the posterior pole lesion in four eyes. In all four, the ^{32}P uptake increased less than 60% compared to the control site and was thus negative by our testing methods.

Discussion

In the elderly patient with a massive vitreous hemorrhage and an ultrasonographically demonstrable posterior pole mass lesion, a hemorrhagic disciform detachment is a likely cause of the hemorrhage. Degenerative changes in the posterior pole of the fellow eye make such a diagnosis even more likely. We observed at least some degree of senile macular choroidal degeneration in the fellow eyes of all 13 patients. Most of these fellow eyes had retained good visual function. Visual function, however, was severely reduced in all eyes with massive vitreous hemorrhage. Although most patients reported some decrease in visual acuity before the vitreous hemorrhage, some noted visual loss only when the vitreous hemorrhage occurred. One patient had no light perception in one eye because of a total hemorrhagic retinal detachment. During the follow-up period, four more eyes lost all light perception after they developed total hemorrhagic retinal de-

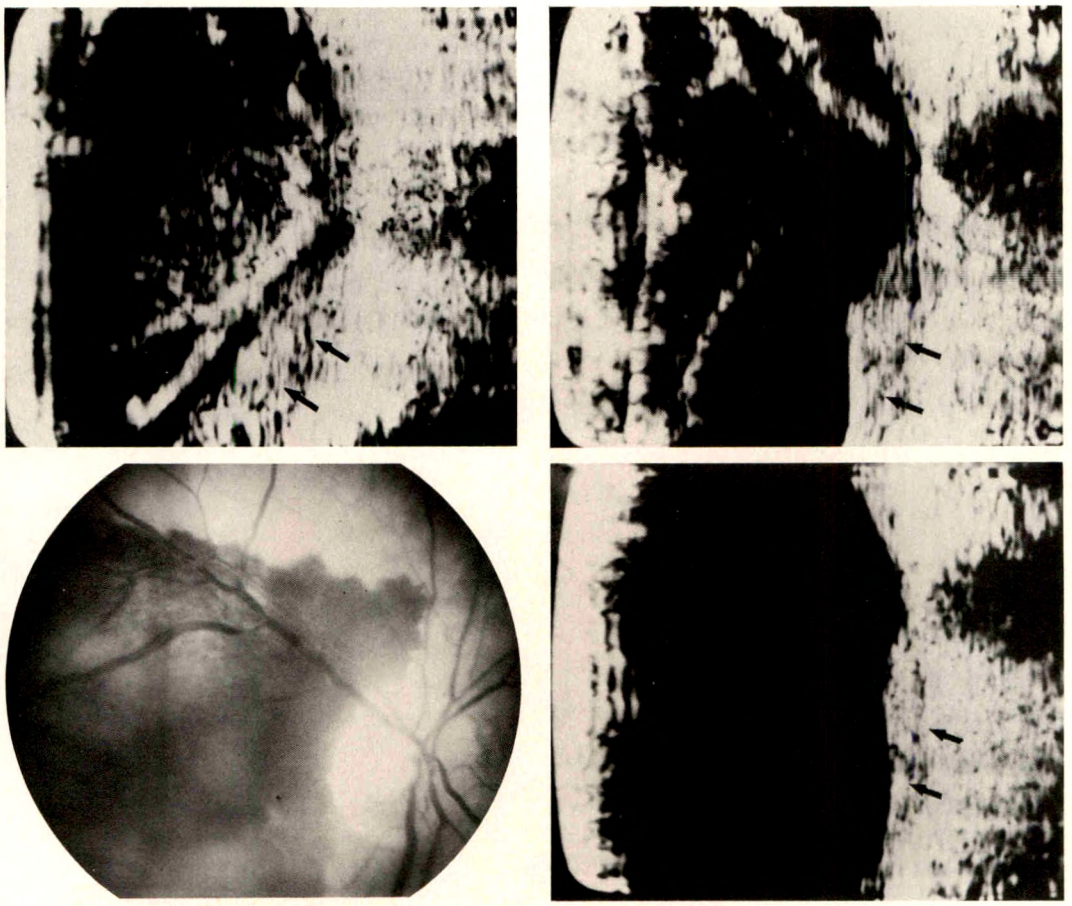


Fig. 1 (Tani, Buettner, and Robertson). Case 1. Top left, Initial ultrasonography demonstrates a broad-based homogeneous choroidal lesion in the posterior pole (arrows) with an overlying retinal detachment in the left eye. The vitreous is opacified. Top right, Six months later, the vitreous is ultrasonographically clearer. There is a total retinal detachment. The posterior polar lesion of the choroid is unchanged. Bottom left, Three years later, the right eye has developed a large hemorrhagic disciform detachment extending to the vascular arcades. Bottom right, Ultrasonographically this hemorrhagic disciform detachment resembles the lesion observed in left eye three years earlier.

tachments. Some investigators have observed complete loss of visual function because of total hemorrhagic retinal detachments.[5,6]

Others have considered hypertension,[2,5] blood dyscrasias,[2] and the use of anticoagulants[2,5-7] responsible for massive vitreous hemorrhages in some patients with senile macular choroidal degeneration. Such a relationship is difficult to demonstrate in our series, since only three patients had hypertensive cardiovascular disease and only one patient had been taking anticoagulants when the hemorrhage occurred. Silva and Brockhurst[8] also failed to show a clear correlation between the use of anticoagulants and the occurrence of a massive vitreous hemorrhage among six cases of eccentric hemorrhagic disciform detachment, three of which were associated with a vitreous hemorrhage.

Vitreoretinal vasoproliferative disease and posterior vitreous detachments, with

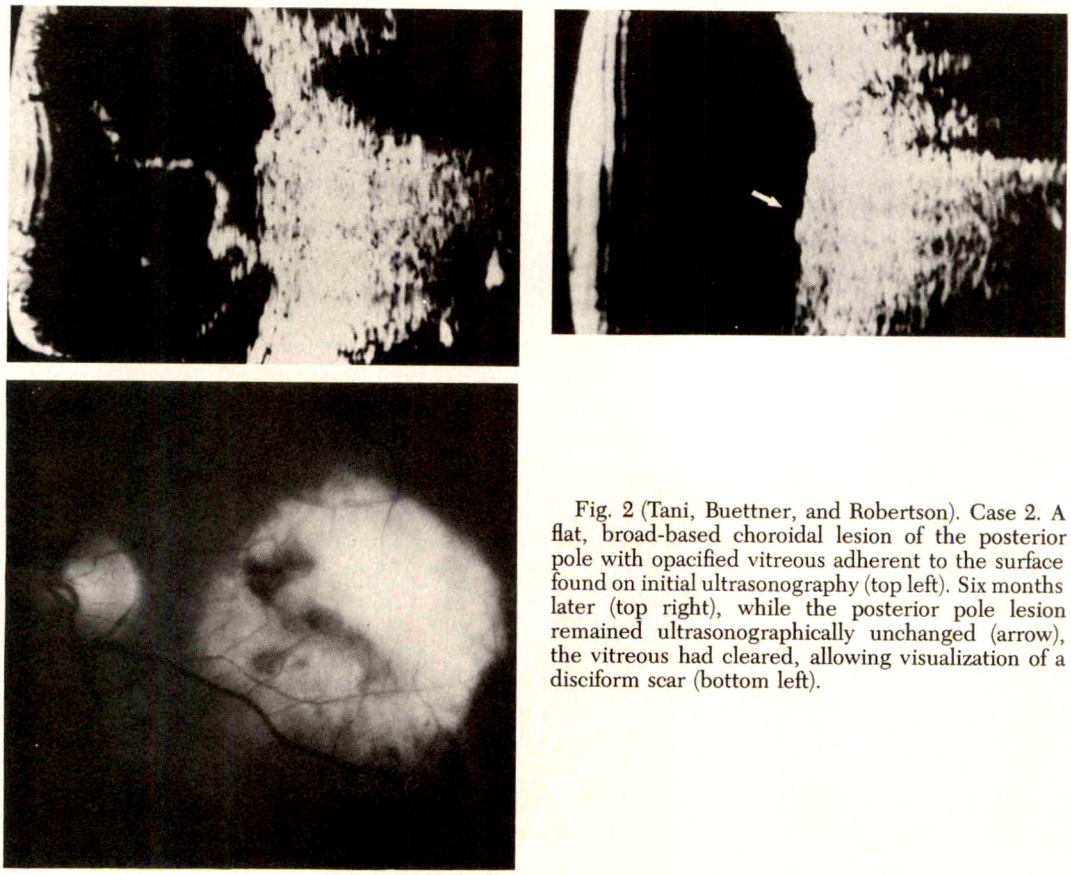


Fig. 2 (Tani, Buettner, and Robertson). Case 2. A flat, broad-based choroidal lesion of the posterior pole with opacified vitreous adherent to the surface found on initial ultrasonography (top left). Six months later (top right), while the posterior pole lesion remained ultrasonographically unchanged (arrow), the vitreous had cleared, allowing visualization of a disciform scar (bottom left).

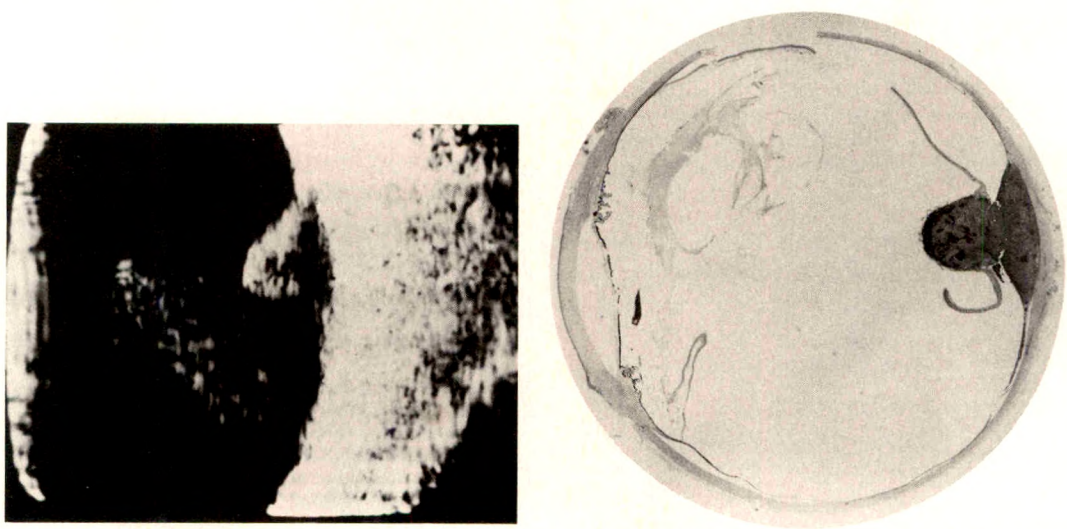


Fig. 3 (Tani, Buettner, and Robertson). Ultrasonogram (left) and corresponding photomicrograph (right) of an eye with a massive vitreous hemorrhage caused by a malignant melanoma of the choroid. Unlike a hemorrhagic disciform detachment, this lesion has a relatively narrow base, is highly elevated, and exhibits choroidal excavation (left).

or without retinal tears, can usually be ruled out as the cause of massive hemorrhage by the absence of a posterior pole mass lesion.

Massive vitreous hemorrhage originating from a malignant melanoma of the choroid is the one condition, we believe, that most urgently needs to be distinguished from a hemorrhage caused by senile macular choroidal degeneration. Intraocular hemorrhage does, indeed, occur in about 3% of eyes harboring a malignant choroidal melanoma.[9] Treatment of malignant melanomas of the choroid has recently become much less aggressive and many opthalmologists now observe rather than enucleate eyes with small or mid-sized choroidal melanomas.[10] In all of our cases, however, massive vitreous hemorrhage prevented us from observing the posterior pole lesion ophthalmoscopically. Furthermore, small choroidal melanomas are less likely to bleed massively into the vitreous than larger tumors. The posterior pole location of a choroidal mass lesion, although suggestive of a hemorrhagic disciform detachment under the circumstances discussed here, in no way excludes the possibility of a malignant melanoma of the choroid arising in the macular area. Conversely, eccentric hemorrhagic disciform detachments are not uncommon.[8] Therefore, the location of a mass lesion does not determine its nature.

Ultrasonography is of particular value in differentiating between massive vitreous hemorrhages caused by senile macular choroidal degeneration and by malignant melanoma of the choroid.[11]

Hemorrhagic disciform detachment of the macula caused by senile macular choroidal degeneration generally appears as a bright line of front surface echoes separated from the posterior wall of the globe by a sonolucent zone.[11] Although this appearance may be characteristic, the ultrasonographic findings in senile macular choroidal degeneration can nevertheless resemble those of malignant melanoma of the choroid, particularly if a hemorrhagic disciform detachment has begun to organize into a disciform scar. Ultrasonography may then demonstrate an acoustically opaque lesion indistinguishable from a small flat choroidal melanoma.[11] In such cases, follow-up examination becomes extremely important. Larger malignant melanomas of the choroid that have broken through Bruch's membrane and hemorrhaged into the vitreous body often exhibit not only a dome-shaped elevation ("collar-button" configuration) but also a choroidal excavation, an acoustic vacuole, or acoustic shadowing in the orbit (Fig. 3, left), findings that are uncharacteristic of hemorrhagic disciform detachment or fibrotic scar.[11] Since hemorrhagic disciform detachments usually do not increase, and sometimes decrease, in size or elevation, serial ultrasonography which establishes growth, especially progressive elevation, is suggestive of a tumor. Choroidal tumors other than malignant melanomas, such as hemangiomas and metastatic tumors, cannot always be differentiated by ultrasonography, but they almost never give rise to massive vitreous hemorrhage.[12]

When the ultrasonographic pattern of a posterior pole lesion suggests malignant melanoma of the choroid or when a posterior pole lesion enlarges during follow-up examinations, a ^{32}P uptake test[13] can be of great diagnostic value. It helps to differentiate between a neoplastic posterior pole lesion and a hemorrhagic disciform detachment as the source of the massive vitreous hemorrhage. Although false-positive ^{32}P uptake tests[13] have been reported, we believe this test is indicated under the circumstances dis-

cussed here. A positive ^{32}P test does not necessarily indicate a malignant melanoma of the choroid. Any lesion of a neoplastic nature can give rise to a positive ^{32}P test. However, massive vitreous hemorrhage is almost never observed with neoplastic lesions of the choroid other than malignant melanoma.[12] Unless ultrasonography and ^{32}P testing strongly suggest the possiblity of a choroidal malignant melanoma, it is probably best to observe the elderly patient with a massive vitreous hemorrhage and a posterior pole lesion, watching for any change that might allow a more definitive diagnosis.

Careful observation will also allow ophthalmoscopic evaluation of the fundus in a significant number of cases. In about three fourths of our patients with massive vitreous hemorrhage secondary to senile macular choroidal degeneration, the vitreous eventually cleared so that ophthalmoscopy could confirm the ultrasonographic diagnosis of a disciform scar and, in some cases, an associated total retinal detachment. These detachments are usually secondary to the disciform process and nonrhegmatogenous. Meticulous indirect ophthalmoscopy in our cases did not demonstrate retinal breaks and there were no significant vitreous proliferations or strands, suggesting the possibility of a traction retinal detachment. Even if the rhegmatogenous nature of such a detachment cannot be ruled out, scleral buckling procedures are rarely indicated, since most of the eyes will have no light perception, as observed in this series.

Summary

In the elderly patient with a massive vitreous hemorrhage in one eye and senile macular choroidal degenerative changes in the fellow eye, a hemorrhagic disciform detachment causing this massive vitreous hemorrhage must be considered in the differential diagnosis. Ultrasonography selectively combined with radioactive phosphorus (^{32}P) testing differentiates between a hemorrhagic disciform detachment or disciform scar and a malignant melanoma of the choroid. The ultrasonographic findings of a hemorrhagic disciform detachment may vary, but usually the posterior pole lesion is relatively flat and broad-based with a fairly homogeneous pattern without choroidal excavation. Posterior polar lesions secondary to senile macular choroidal degeneration do not grow. The vitreous hemorrhage clears in about three fourths of the patients, eventually allowing visualization of the posterior pole and ophthalmoscopic confirmation of a disciform scar. Almost one half of the eyes may be expected to lose all light perception because of the development of an initially hemorrhagic retinal detachment.

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RETINAL EMBOLI IN PATIENTS WITH MITRAL VALVE PROLAPSE

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Mitral valve prolapse is a common cardiological condition usually considered benign. There have been reports, however, describing the development of sudden, severe mitral regurgitation, chest pain, infective endocarditis, and fatal ventricular arrhythmias.[1,2] One of the recently reported complications of mitral valve prolapse is thromboemboli resulting in cerebral ischemic events and visual complications.[3-9] We believe that emboli secondary to mitral valve prolapse may cause retinal occlusive disease with a wide range of ophthalmic manifestations. Such emboli may explain many episodes of amaurosis fugax occurring in young patients, some cases of branch and central retinal artery occlusion, and perhaps some cases of retinal neovascularization secondary to recurrent small embolic arteriolar occlusions (atypical Eales' disease). We describe six patients with mitral valve prolapse who had retinal occlusive disease.

SUBJECTS AND METHODS

All patients were examined here between August 1979 and January 1980. They underwent complete ophthalmologic examination, including visual field testing on the Goldmann perimeter and stereoscopic color fundus photography. We performed fluorescein angiography on all patients except Patient 3. A qualified cardiologist examined all patients and found either a nonejection systolic click, a late systolic murmur, or both. Additionally, every patient had mitral valve prolapse demonstrated by echocardiography.

The first five cases are examples of suspected isolated retinal and choroidal emboli associated with mitral valve prolapse. The sixth's patient's clinical course suggested that small recurrent emboli from the mitral valve may give rise to a pattern of chronic retinal ischemia with neovascular proliferans (atypical Eales' disease).[10]

CASE REPORTS

Case 1—A 40-year-old woman had a paracentral scotoma of the left eye of six weeks' duration. At ocular examination her best correctable visual acuity was 6/9 (20/30) in both eyes. Results of an examination of the anterior segment, pupillary responses, and extraocular muscle function were normal. Visual field testing showed a superior nasal scotoma of the left eye. Posterior pole examination showed findings consistent with embolic occlusion of retinal and, possibly, small choroidal vessels. The left fundus showed an occluded inferior temporal artery just below the macula with infarcted retina distal to the occlusion. There was also a small white opacity about 1 disk diameter above the left optic disk which on slit lamp and biomicroscopic examination appeared deep to the retina. The right eye had two similar deep white lesions in the posterior pole. Fluorescein angiography was consistent with embolic occlusion of the inferior temporal artery in the left eye and multiple infarcts of either the choroid or deep retina in both eyes (Fig. 1).

The patient had no medical problems except a history of mild Raynaud's syndrome. She used no medications nor did she have a history of drug abuse. She had not used oral contraceptives within the preceding two years. Extensive physical and systemic examination showed no evidence of hypertension, infective endocarditis, carotid bruits, or migraine. An extensive rheumatologic evaluation, including erythrocyte sedimentation rate, antinuclear antibody titer, lupus erythematosis preparation, rheumatoid antigen, serum protein electrophoresis, and cryoglobulins, disclosed no unusual findings. Abnormal find-

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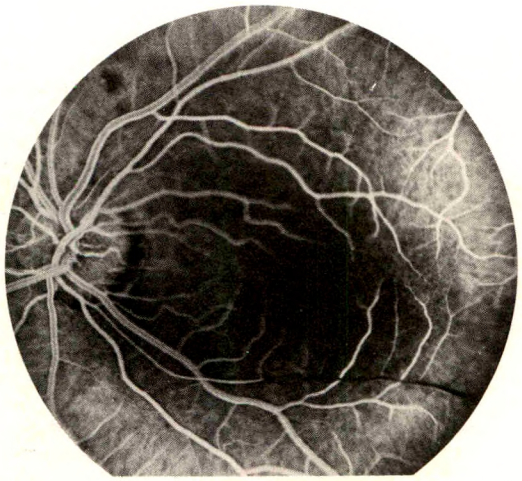


Fig. 1 (Caltrider and associates). Case 1. Early venous phase of fluorescein angiogram of the left eye showing occlusion of the inferior temporal artery and a hypofluorescent choroidal infarct with enhancing rim.

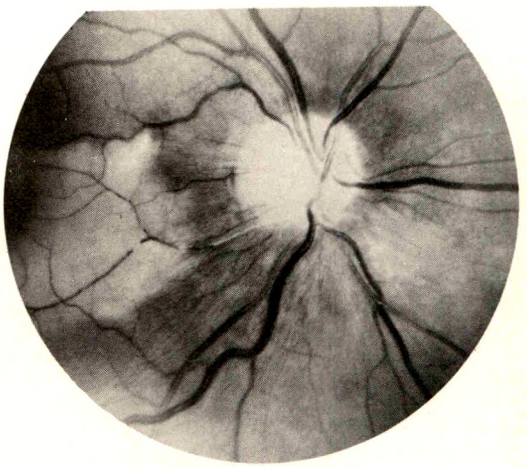


Fig. 2 (Caltrider and associates). Case 2. Branch retinal artery occlusion of the right eye.

ings included a grade 2/6 systolic murmur without clicks and a marked pectus excavatum. An echocardiogram showed pansystolic posterior mitral leaflet bowing. An electrocardiogram revealed a minor right-sided ventricular conduction delay.

Case 2—A 41-year-old woman complained of the sudden onset of a paracentral scotoma in the right eye. Visual acuity was correctable to 6/9 (20/30) in the right eye and 6/6 (20/20) in the left eye. Visual field testing disclosed a large centrocecal scotoma in the right eye and a scotoma below the blind spot in the left eye. Ophthalmoscopic examination showed an occlusion of a branch retinal artery supplying the nasal and inferior portion of the macula in the right eye (Fig. 2) and a small branch retinal arterial occlusion superior to the disk in the left eye. Extensive medical evaluation including complete blood cell count, VDRL, erythrocyte sedimentation rate, antinuclear antibody titer, rheumatoid factor, serum protein and immunoglobulin electrophoresis, platelet count, chest roentgenogram, and carotid and aortic arch angiography gave normal findings. She took no medications. Abnormal findings included a grade 2/6 systolic murmur; an M mode echocardiogram showed flattening of the mitral valve closure line. A two-dimensional echocardiogram showed mitral valve prolapse.

Case 3—A 48-year-old man had a superior field defect in the right eye that he had first noted six weeks previously. His best corrected visual acuity was 6/6 (20/20) in both eyes. The anterior segment, pupillary responses, and color vision were normal. Goldmann visual fields showed a dense superior nasal field defect of the right eye with a configuration consistent with a retinal lesion (Fig. 3). Ophthalmoscopic examination of the right eye showed a markedly narrowed arterial branch off the inferior temporal artery with a corresponding arcuate nerve fiber layer defect. The left eye was normal.

For approximately five years, he had had hypertension which was controlled with propranolol and methyldopa. Systemic evaluation showed his blood pressure was within the normal range and there was no evidence of carotid bruits or vasculitis. The only abnormal findings were a midsystolic click and a late systolic murmur; there was also a moderate pectus excavatum. An echocardiogram showed mitral valve prolapse.

Case 4—A 76-year-old woman had had a sudden, painless loss of vision in the right eye two months previously. Her ophthalmologist had diagnosed cen-

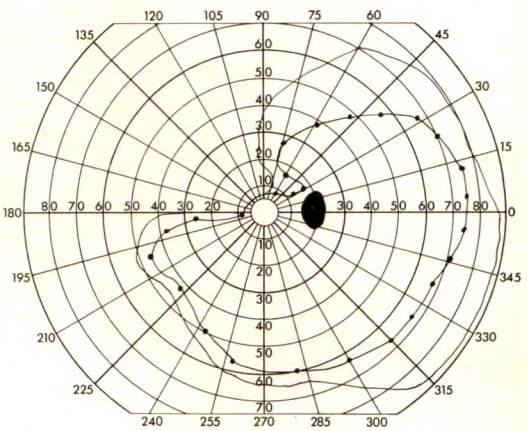


Fig. 3 (Caltrider and associates). Case 3. Goldmann visual field of the right eye showing a dense superior nasal field defect with configuration of a retinal lesion.

tral retinal artery occlusion. Ocular examination showed best corrected visual acuities of hand motions in the right eye and 6/6 (20/20) in the left eye. Pupillary examination showed a 4+ afferent pupillary defect in the right eye. Visual field testing showed a diffusely depressed visual field in the right eye; the left eye was normal. The anterior segments were normal. The fundus of the right eye showed narrow arteries with segmentation of the blood column; along the superior temporal artery there were aneurysmal dilations at branching sites (Fig. 4). These aneurysmal dilations suggested that the central retinal artery occlusion was secondary to emboli that had transiently lodged at these points, causing endothelial damage and subsequent wall weakening. We saw no embolic material in any of the retinal arteries. Fluorescein angiography showed that these aneurysms contained clotted blood and had flow only through their centers; there was retrograde filling of the inferior temporal artery from the perifoveal capillary net. The left fundus was normal.

There were no clinical manifestations of hypertension, arrhythmias, myocardial infarct, carotid bruits, or vasculitis. Her medical history included degenerative joint disease and hypothyroidism. She used aspirin and thyroid extract. Abnormal physical findings included a nonejection midsystolic cardiac click and a late regurgitant systolic murmur. The echocardiogram disclosed prolapse of both the anterior and posterior leaflets of the mitral valve.

Case 5—A 50-year-old woman had an episode of amaurosis fugax of the left eye. The episode was characterized as a shadow appearing in her upper temporal field and advancing inferiorly to total blindness. The recovery of vision was reversed, with vision regained in the lower field first; the total episode lasted more than five minutes. There were no associated neurologic symptoms. An ocular examination five days after the episode showed a best corrected visual acuity of 6/6 (20/20) in both eyes. Pupillary reflexes and extraocular muscle function were normal; the anterior segment was unremarkable. The posterior pole of both eyes showed areas of retinal pigment epithelial atrophy clustered around the macula. The retinal vessels showed mild arteriolar narrowing, but no evidence of embolic material. The fluorescein angiogram showed window defects in the areas of pigment epithelial atrophy.

The patient had been observed for atypical chest pain and palpitations; echocardiography had shown mitral valve prolapse. There was no evidence of myocardial infarction, carotid bruits, hypertension, coagulation, or hematologic abnormalities and no evidence of collagen vascular diseases. She took no medications at the time of her visual problem.

Case 6—A 14-year-old girl had experienced a painless decrease of visual acuity in both eyes in 1970. An ocular examination showed a best corrected visual acuity of 6/9 (20/30) in the right eye and 6/60 (20/200) in the left eye. There was hypotropia of 4 prism diopters in the left eye and the pupillary responses were sluggish in both eyes, but there was no afferent defect. Results of the anterior segment examinations were normal; there was no evidence of uveitis. Ocular tension by applanation tonometer was 16 mm Hg in both eyes. Posterior pole examination showed markedly ischemic fundi with thready arteries and massive retinitis proliferans involving the disks of both eyes. Ghost vessels could be seen in the periphery. Fluorescein angiography showed marked telangiectasia of the perifoveal vascular net and vascular collaterals across the macula in the left eye (Fig. 5).

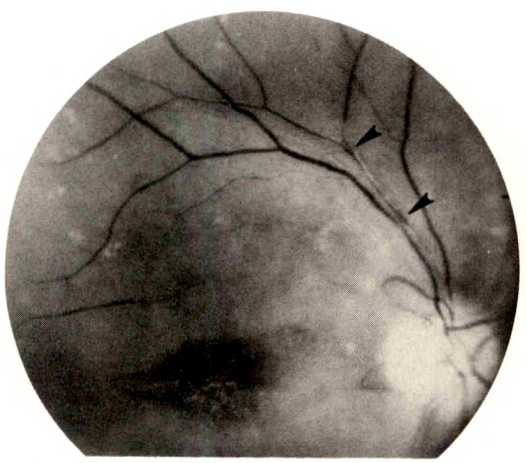


Fig. 4 (Caltrider and associates). Case 4. Aneurysmal dilations along the superior temporal artery (arrow) and a cherry-red spot in the macula after a central retinal artery occlusion.

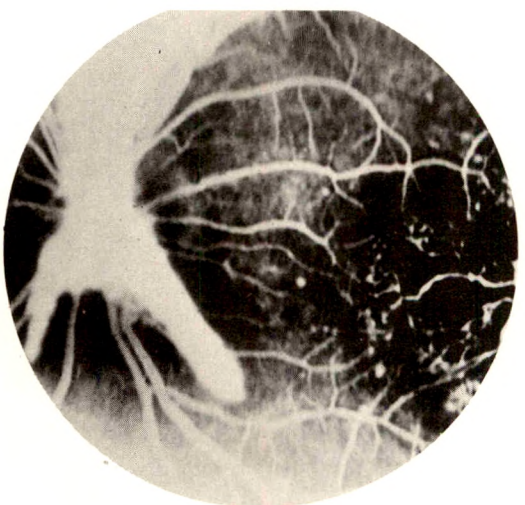


Fig. 5 (Caltrider and associates). Case 5. Late venous phase of fluorescein angiogram of the left eye showing telangiectasia of the perifoveal capillary net, vascular shunts across the macula, and leaking retinitis proliferans of the disk.

The patient's medical history was unremarkable. There was no family history of eye disease, diabetes, or collagen vascular disease. She took no medications. She was admitted to the hospital and a full diagnostic examination was done. The studies included a four-hour glucose tolerance test, a muscle biopsy for basement membrane thickening, a complete blood cell count, collagen vascular studies, serum protein and hemoglobin electrophoresis, coagulation studies, cryoglobulins, spinal tap, VDRL, blood chemistries, and a skin biopsy of a small telangiectasis. All gave normal results. Cardiac examination showed a grade 2/6 systolic murmur. An electrocardiogram recorded an occasional premature ventricular contraction.

The patient continued to have deteriorating vision over the following years. She underwent bilateral vitrectomies, lensectomies, and panretinal photocoagulation for rubeosis irides following the vitrectomies. The diagnosis was atypical Eales' disease. In 1974, at the age of 18, she developed grand mal seizures. A complete systemic evaluation, including carotid and cerebral arteriography, computed axial tomography of the brain, and lumbar puncture disclosed no unusual findings. Her present visual acuity is 6/60 (20/200) in the right eye and counting fingers at 3 feet in the left eye. A cardiogram in September 1979 again showed a systolic murmur and an M mode echocardiogram showed mitral valve prolapse.

Discussion

Atypical forms of Eales' disease primarily involving the retinal arterioles[10,11] differ from classic Eales' disease, which is characterized by inflammation of the peripheral retinal venules.[12] In these atypical cases there is arteriolar occlusive disease and the process extends posteriorly with extensive neovascularization of the posterior pole. The prognosis for useful vision is poor. To our knowledge, this is the first report suggesting that this clinical picture may be secondary to recurrent small emboli from prolapsing mitral valves. These emboli may occlude small arterioles, leading to areas of ischemia and neovascularization similar to that reported with recurrent talc and cornstarch emboli.[13]

The midsystolic click and late systolic murmur syndrome (also known as Barlow's syndrome, the click-murmur syndrome, and the billowing posterior leaflet syndrome) was first described in the late 19th century and has been the subject of renewed interest since the 1960s. Cuffer originally described the cardiac clicks in 1887. He thought they were secondary to pericardial adhesions and other extracardiac causes. In 1961, however, J. V. O. Reid suggested that the mitral valve was the source of the clicks, which other investigators later confirmed.[1] Mitral valve prolapse appears to have many causes and a wide range of clinical manifestations.

Estimates of the prevalence of mitral valve prolapse vary from 5% to 20%, depending on the diagnostic techniques used.[14] Patients with mitral valve prolapse often have associated thoracic skeletal abnormalities such as a narrowed anteroposterior diameter, pectus excavatum, straight thoracic spine, and scoliosis.[15]

Although mitral valve prolapse that is not secondary to coronary disease usually has a benign course, a number of complications have been reported.[12] One of the most recently recognized complications is thromboembolism. Barnett and associates[3] described 12 patients with angiographically proven mitral valve prolapse who had transient ischemic attacks and partial, nonprogressive strokes. None of the patients had atherosclerotic carotid or cerebral vascular disease, hypertension, or coagulation defects; none had used oral contraceptives. Arteriography showed that all 12 were free of atherosclerotic disease in the neck and intracranial arteries. Four patients had an intracranial artery occluded in an area appropriate for their symptoms. Malcolm and associates[6] studied 85 people with mitral valve prolapse and found that 11 had neurologic complaints, nine had transient ischemic attacks, and one had stroke. Hirsowitz and Saffer[4] described four young patients, ranging in age from 16 to 37 years, with mitral valve prolapse who suffered nonprogressive strokes. Three of the four

underwent carotid and cerebral artery angiography, and two showed occlusion of an appropriate cranial vessel. There was no evidence of atherosclerotic disease, hypertension, vasculitis, coagulation defects, or use of oral contraceptives in any of the four.

Few studies have reported retinal emboli in patients with mitral valve prolapse. Woldoff and associates[8] described two young women with retinal vascular thrombosis and mitral valve prolapse; Kimball and Hedges[9] reported a case of recurrent bilateral amaurosis fugax in a 28-year-old man with no abnormal findings except for mitral valve prolapse. Wilson and associates[7] described ten people with mitral valve prolapse who had visual disturbances consistent with embolization in the ophthalmic or posterior cerebral circulation.

Pathologic examination of these mitral valves frequently discloses redundant and thickened leaflets with a gelatinous consistency. Microscopic examination of the affected leaflets shows fibrosal degeneration with replacement by a myxomatous tissue containing acid mucopolysaccharides.[16] The cause of the degeneration is unknown, but some investigators believe it is related to a hereditary connective tissue disorder similar to Marfan's syndrome; others believe it is a nonspecific reaction to many kinds of injuries and hereditary degenerative processes.[16]

The source of the emboli is still a subject of controversy. Pomerance[17] reviewed 35 autopsy cases of myxomatous mitral valves and found adherent fibrin and red blood cells in ten. This fibrinous endocarditis may initiate platelet aggregation with microthrombus formation; these microemboli may be dislodged during sinus rhythm and, perhaps more frequently, during arrhythmias. Turbulent flow in the area around the valve may aid in the mechanism of platelet and fibrin aggregation. Recent studies[18] have suggested abnormal platelet survival time in patients with mitral valve prolapse, but the significance of this finding or its possible role in thromboembolic phenomena is poorly understood.

The differential diagnosis of retinal occlusion in the young patient includes emboli from infective endocarditis or secondary to rheumatic valvular heart disease with atrial fibrillation.[19] Other rare sources of emboli include fragments of atrial myxomas,[20] amniotic fluid emboli, fat emboli from skeletal fractures, and foreign products from intravenous drug abuse. Hypercoagulable states, such as those caused by the use of oral contraceptives, and blood dyscrasias, such as sickle cell anemia, macroglobulinemia, and cryoglobulinemia, should be ruled out. Arteritis associated with systemic lupus erythematosus, periarteritis nodosa, rheumatoid arthritis, and dermatomyositis may also be responsible for retinal occlusion.

In the older patient, carotid artery atherosclerotic disease is the most common source of retinal emboli. However, emboli can also result from mural thrombi after myocardial infarction, from calcified aortic valves, and from prosthetic heart valves.

All six of our patients with retinal occlusive disease underwent extensive noninvasive cardiac and systemic tests and were found to have prolapsed mitral valves. Admittedly, only two underwent carotid arteriography and none have come to autopsy. Thus, the evidence is inferential. We were impressed, however, by the high incidence of mitral valve prolapse found in our patients with otherwise unexplained retinal arterial occlusive disease. This theory is also supported by Barnett and associates[21] who recently found that the incidence of mitral prolapse was much higher in young patients with strokes and transient ischemic attacks than in the normal popula-

tion. Prolapse was detected in 40% of young patients with cerebral ischemic attacks, compared with 6.8% of controls.

Most patients with mitral valve prolapse are asymptomatic and need no medical therapy except prophylactic antibiotic therapy during dental or surgical procedures. Patients with mitral valve prolapse who have emboli are often treated with platelet inhibitors such as aspirin and dipyridamole. Therefore, all patients with ocular emboli without an obvious cause should be examined by a cardiologist and tested for mitral valve prolapse.

Summary

Small fibrin-platelet emboli caused by prolapsed mitral valves may cause retinal occlusive disease with a wide range of ophthalmic manifestations, including amaurosis fugax in young people, retinal or choroidal arteriolar occlusion, and retinal neovascularization (atypical Eales' disease). Six patients with retinal occusive disease underwent extensive noninvasive cardiac and systemic tests and were found to have prolapsed mitral valves. Patients with unexplained ocular emboli should be examined by a cardiologist for possible mitral valve prolapse.

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CORRELATION OF APHAKIC RETINAL DETACHMENT AND REFRACTIVE ERROR WITH GENDER

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Retinal detachment is more common in patients who are myopic[1-5] or aphakic.[3-9] Published reports on aphakic retinal detachment have implicated myopia as a risk factor.[3,4,10,11] The majority of patients have been men.[3,12] Previous studies, however, have not analyzed the aphakic refractive error distributions separately for male and female patients with aphakic retinal detachment. The purpose of the present study was to determine the importance of refractive error in the development of aphakic retinal detachment for each sex.

SUBJECTS AND METHODS

I reviewed 451 inpatient records from the McPherson Hospital and selected two groups of aphakic patients for study. One criterion for inclusion in this study was the absence of any evidence of previous ocular trauma or disease (excluding moderate hypertensive or arteriolosclerotic retinopathies). The aphakic refractive error was measured a minimum of three months after intracapsular extraction in order to minimize the variability associated with corneoscleral wound healing.[11,13] The aphakic refractive error was determined in each case by retinoscopy or manifest refraction or a combination of the two.

The patients studied ranged in age from 37 to 87 years at the time of intracapsular cataract surgery. These individuals had been observed at the McPherson Hospital for varying periods of time with first visits recorded in early childhood as well as at the time aphakic retinal detachment was diagnosed.

The group with aphakic retinal detachment consisted of 81 patients who had retinal detachments after intracapsular cataract extractions, I selected this group from 321 patients whose record numbers I had drawn at random from files of retinal detachments repaired between the years 1965 and 1975. Fifteen of the patients selected had bilateral aphakic retinal detachments.

I selected the control group by randomly choosing 130 patients from files of intracapsular cataract extractions performed between 1965 and 1975. Ninety-three of the selected patients met the criteria of the study. In patients with bilateral aphakia, a coin-flip determined the eye to be included in the control group.

I recorded the sex of and refractive error for each patient and the age at time of cataract extraction for 77 patients with aphakic retinal detachment and 91 controls. For patients with bilateral aphakic retinal detachment, I recorded data for each eye and determined the averages in order to treat each patient as a statistical unit. I considered a finding significant if the associated P value was less than .05 in a two-tailed test for difference between statistics.

RESULTS

The mean spherical equivalents for men did not differ significantly between the aphakic retinal detachment and con-

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TABLE 1

SPHERICAL EQUIVALENTS OF REFRACTIVE ERRORS

Group	Men	Women	Both Sexes
Control			
No.	41	52	93
Mean equivalent (diopters)	11.68	12.37	12.07
S.D.	± 1.23	± 1.66	± 1.52
Aphakic retinal detachments			
No.	48	33	81
Mean equivalent (diopters)	11.31	11.41	11.35
S.D.	± 1.22	± 1.36	± 1.27
P value	.156	.004*	.002*

*Significant at $P<.05$ (two-tailed test).

trol groups (P = .156) (Table 1). In contrast, the means of the women in the two groups showed a statistically significant difference of approximately 1.0 diopter (P = .004). For purposes of illustration, I plotted percentile distributions for each group according to spherical equivalent of refractive error. Comparison of the graphs for women (Fig. 1) and men (Fig. 2) shows the more apparent shift of the distribution of refractive errors for women with aphakic retinal detachments.

I determined the mean age at the time of cataract extraction within each group for each sex (Table 2). The women with aphakic retinal detachments were significantly younger than the female control group (7.0 years; P = .005). I also found a statistically significant difference in mean

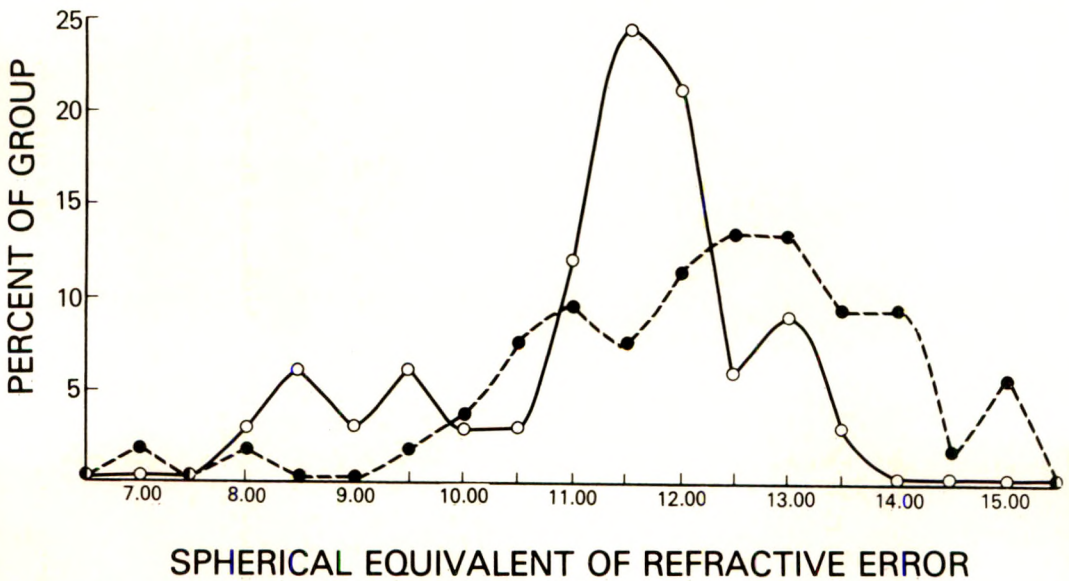


Fig. 1 (Bedrick). Percentile distributions for women. Open circles, Group with aphakic retinal detachments (No. = 33; mean spherical equivalent, 11.14 diopters; median, 11.75 diopters; range, 8.25 to 13.50 diopters). Closed circles, Control group (No. = 52, mean spherical equivalent, 12.37 diopters; median, 12.49 diopters; range, 7.25 to 15.37 diopters).

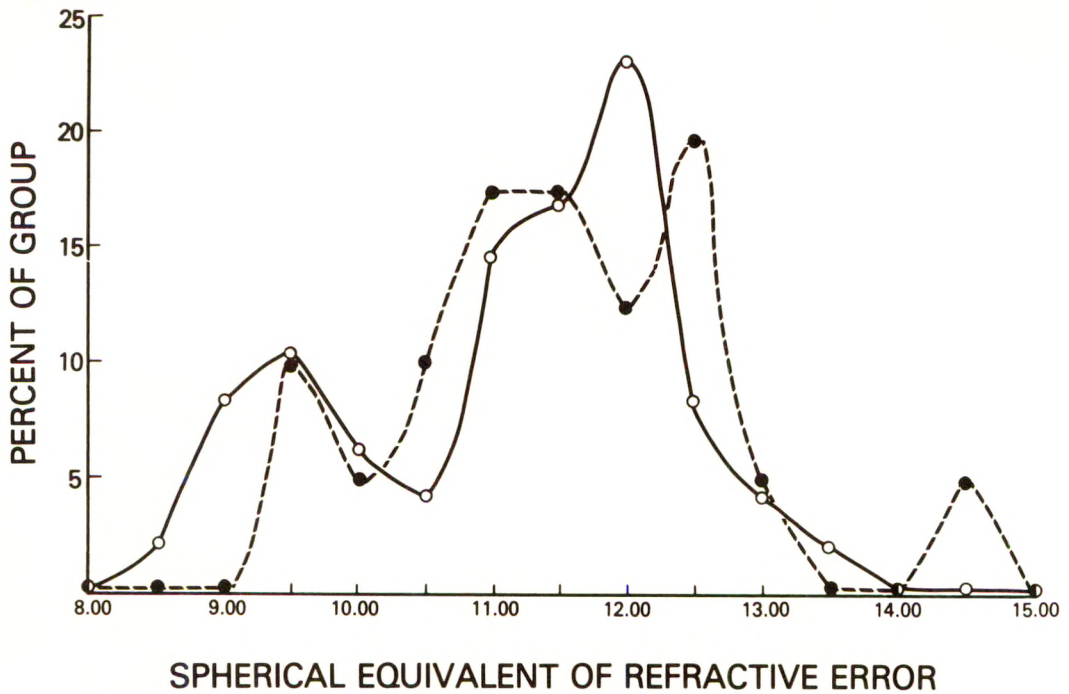


Fig. 2 (Bedrick). Percentile distributions for men. Open circles, Group with aphakic retinal detachments (No. = 48; mean spherical equivalent, 11.31 diopters; median, 11.70 diopters; range, 8.87 to 13.75 diopters). Closed circles, Control group (No. = 41; mean spherical equivalent, 11.68 diopters; median, 11.71 diopters; range, 9.50 to 14.87 diopters).

age between the groups of men (4.0 years; P = .039). In the aphakic retinal detachment population, the difference in age between men and women was not significant (P = .648). Similarly, I found no significant difference between the sexes in the control population (P = .374).

TABLE 2

AGE AT TIME OF CATARACT EXTRACTION

Group	Men	Women
Control		
No.	40	51
Mean age (yrs)	64.0	65.9
S.D.	±10.0	±10.3
Aphakic retinal detachment		
No.	45	32
Mean age (yrs)	60.0	58.9
S.D.	± 7.5	±12.1
P value	.039*	.005*

*Significant at P<.05 (two-tailed test).

I found a significantly higher percentage of men in the group with aphakic retinal detachments than in the general aphakic population (59% or 48 of 81 vs 44% or 41 of 93; P = .046). Comparison of patients with bilateral and patients with unilateral aphakic retinal detachments shows a statistically significant difference in the sex distribution: 87% (13 of 15) of the patients with bilateral and 53% (35 of 66) of the patients with unilateral aphakic retinal detachments were men (P = .017). In the aphakic control population studied, the men had a significantly lower mean spherical equivalent of refractive error than the women (P = .021), an observation consistent with the increased incidence of myopia in men with phakic eyes.[2]

Discussion

This study found that the mean aphakic refractive error of women who later developed aphakic retinal detachments was significantly more myopic than that of controls. This was not so in men. It appears that axial myopia is a risk factor for aphakic retinal detachment that is greater for women than for men.

I found no difference in age between men and women within either the aphakic retinal detachment group or the control group. Therefore, age differences are unlikely to account for the findings of this investigation.

Hyams, Bialik, and Neumann[10] found that the mean age at time of cataract extraction was lower for patients with aphakic retinal detachments than for the general aphakic population, a finding confirmed by this study. This age discrepancy may be caused in part by higher death rates in the older population that select against the occurrence of late surgical complications. Additionally, younger cataractous eyes may have a higher incidence of retinal disease that predisposes them to retinal detachment than eyes with cataracts of later onset.

The data in this study confirmed the previously documented finding that the majority of patients with aphakic retinal detachments,[3,12] particularly bilateral aphakic retinal detachments, are men.[14] We can, therefore, postulate the existence of at least one risk factor associated with men for nontraumatic aphakic retinal detachment. This risk factor or group of factors would overshadow the influence of refractive error in men and also predispose men to bilateral aphakic retinal detachments.

One of these risk factors may be cardiovascular disease, known to occur more often in men than in women.[15] Zinn, Field, and Schepens[16] found higher rates of coronary artery disease and systemic hypertension in a group of 100 consecutive patients with rhegmatogenous retinal detachment than in the general population of similar age. They postulated a mechanism by which generalized vascular disease results in relative tissue hypoxia in the choriocapillaris, leading to retinal tears. Therefore, cardiovascular disease may predispose men to aphakic retinal detachment more than women and, at the same time, diminish the importance of other risk factors, including axial myopia, for men.

Summary

I analyzed the aphakic refractive errors of men and women separately in a statistical study of patients with aphakic retinal detachments. I compared the distribution of aphakic refractive errors in a population of 81 adult patients with nontraumatic aphakic retinal detachment with that of a randomly selected control population of 93 adult patients with aphakia. As a group, women who developed aphakic retinal detachments were significantly more myopic than female controls (11.41 vs 12.37 diopters of spherical equivalent; $P = .004$). In contrast, the refractive error in men did not differ between the aphakic retinal detachment and control groups (11.31 vs 11.68 diopters; $P = .156$). A significantly higher percentage of patients with bilateral (87%) rather than unilateral (53%) aphakic retinal detachments were men ($P = .017$). In men other risk factors for aphakic retinal detachment, including cardiovascular disease, may overshadow the influence of axial myopia.

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RESOLUTION OF OPTIC DISK NEOVASCULARIZATION ASSOCIATED WITH INTRAOCULAR INFLAMMATION

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Optic disk neovascularization is a recognized complication of intraocular inflammation,[1,2] but, to our knowledge, previous case reports have not examined the ultimate course of the neovascularization, with or without therapeutic intervention. We describe three cases of optic disk neovascularization associated with intraocular inflammation that regressed when the inflammation subsided. In the first two cases we used intensive therapy, whereas in the third case we carefully observed the patient and used minimal therapy. These cases demonstrate regression of the neovascularization with resolution of the inflammation, suggesting a causal relationship.

CASE REPORTS

Case 1—A 21-year-old woman was referred from the Rheumatology Clinic for assessment of cloudiness in her left eye of one week's duration. She had gone to the clinic two days previously with a two-week history of a painful right knee. There was no history of a rash or oral or genital lesions. Her medical history disclosed no previous joint complaints, diabetes mellitus, gout, tuberculosis, or venereal disease. Results of the physical examination were normal except for a swollen tender right knee.

An arthrocentesis of the right knee was performed and demonstrated findings compatible with an acute inflammatory effusion. Complement levels of the effusion were normal. All cultures of the effusion as well as appropriate cultures of the oral, vaginal, and rectal areas for venereal disease were negative. Her complete blood cell count and uric acid levels were normal. A second-strength purified protein derivative test was negative. An antinuclear antibody titer was positive at 1:254 with a finely speckled diffuse pattern. All other serologic tests, including rheumatoid factor, anti-RNA, rapid plasma reagent, and HLA-B27 antigen, were negative. A Westergren sedimentation rate was 32 mm/hr. Roentgenograms of her chest and sacroiliac joints and an upper gastrointestinal study were normal. On the basis of the evaluation, no cause for her arthritis was apparent.

On initial ophthalmologic examination, March 16, 1979, her best corrected visual acuity was R.E.: 6/6 (20/20-1) and L.E.: 6/7.5 (20/25). The anterior segment of her right eye was normal. There were mutton-fat keratic precipitates on the endothelial surface of the left eye with 2+ cells and flare in the anterior chamber. Biomicroscopic examination of the vitreous gave normal results in the right eye but disclosed 1+ cells in the anterior vitreous of the left eye.

She began using topically applied prednisolone acetate 1%, five times each day, and homatropine 2%, three times each day, in the left eye. During the next two months her anterior chamber reaction decreased to trace flare in the left eye but on May 24, 1979, she complained of floaters in the left eye. We noted a subhyaloid hemorrhage with neovascularization of her optic disk (Fig. 1). Results of an ophthalmoscopic examination of the right eye were normal. Detailed biomicroscopy of the peripheral retina in the left eye, using scleral depression and a Goldmann three-mirror lens, showed normal retinal blood vessels. Fluorangioscopy did not disclose any areas of nonperfusion in the peripheral retina. Goldmann visual fields were full and normal to the II_4e and 0_4e test objects in both eyes.

We injected 4 mg of dexamethasone subtenonally in the left eye and prescribed 120 mg/day of orally administered prednisone for four days. The dosage was rapidly reduced to 100 mg every other day for one week and then 50 mg every other day for one week. A repeat fluorescein angiogram demonstrated decreased leakage from the neovascular network on the optic disk of the left eye (Fig. 2).

We then reduced the dosage of corticosteroids further to 10 mg every other day over a three-week period. A repeat fluorescein angiogram, at that time, six weeks after institution of therapy, showed marked regression of the neovascular network with some resolution of the subhyaloid hemorrhage. She was free of any joint complaints and her anterior segment inflammation had totally subsided. She stopped taking corticosteroids and, three months after beginning therapy, she remained asymptomatic with no evidence of leakage from the optic disk in her left eye (Fig. 3).

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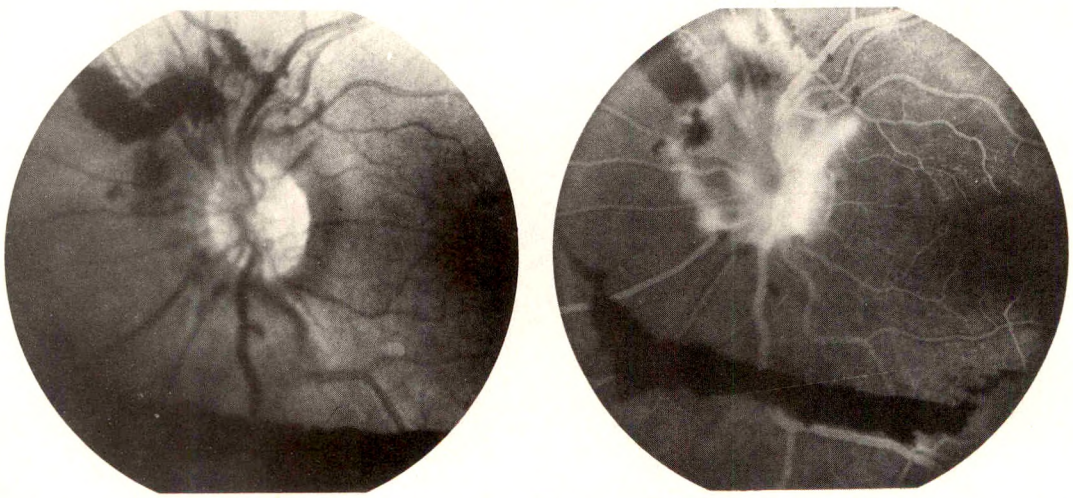


Fig. 1 (Kelly and Weiter). Case 1. Left eye two months after onset of iridocyclitis. Left, Initial appearance of neovascularization of the optic disk and subhyaloid hemorrhage. Right, Fluorescein angiogram shows neovascular frond with late-phase leakage.

Case 2—We had been observing a 48-year-old woman since 1976 because of recurring episodes of granulomatous anterior uveitis in her right eye, believed to be secondary to sarcoidosis. In October 1979, she had mutton-fat keratic precipitates on the corneal endothelial surface and 3+ cells and flare in her right eye. Her visual acuity was R.E.: 6/15 (20/50) and L.E.: 6/6 (20/20). We noted 3+ cells in her vitreous with inferior vitreous opacities when she was examined with her eyes dilated. There was sheathing of the retinal vessels and inferior retinal infiltrates in the right eye. The left eye seemed normal. The patient started using topically applied prednisolone acetate 1%, four times a day, in the right eye and homatropine 2%, three times a day, for her anterior segment inflammation.

During the next two months, we noted that the anterior chamber reaction decreased to 1+ cells and flare but 3+ cells persisted in the vitreous, as did the retinal vasculitis. The dosage of the medications was gradually reduced at this point.

In February 1980, four months after her initial visit, the patient had a markedly increased amount of inflammation in both the anterior chamber and vitreous, preventing an adequate examination of the fundus. Her visual acuity at this time was R.E.: 6/60

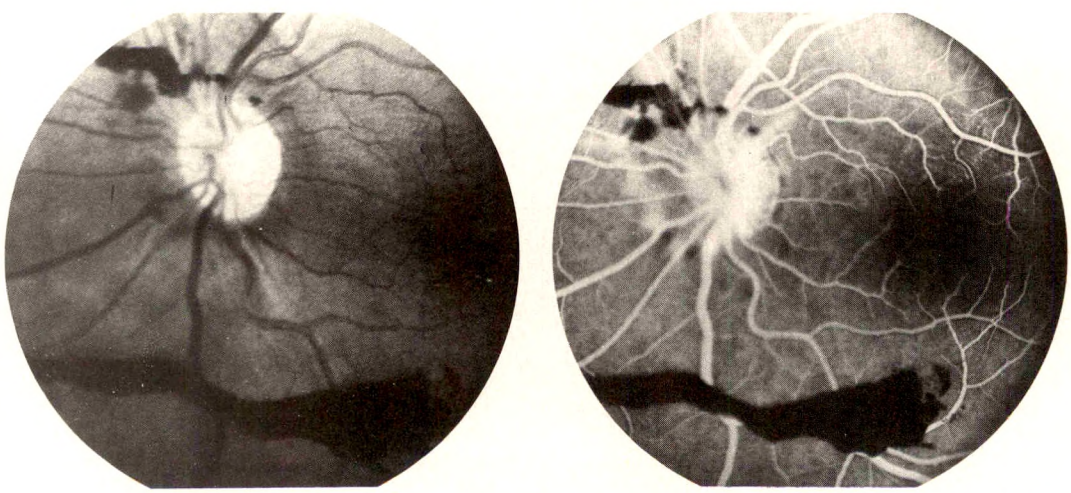


Fig. 2 (Kelly and Weiter). Case 1. Left eye after two weeks of therapy. Left, Regression of neovascularization. Right, Fluorescein angiogram shows decreased leakage from neovascular frond.

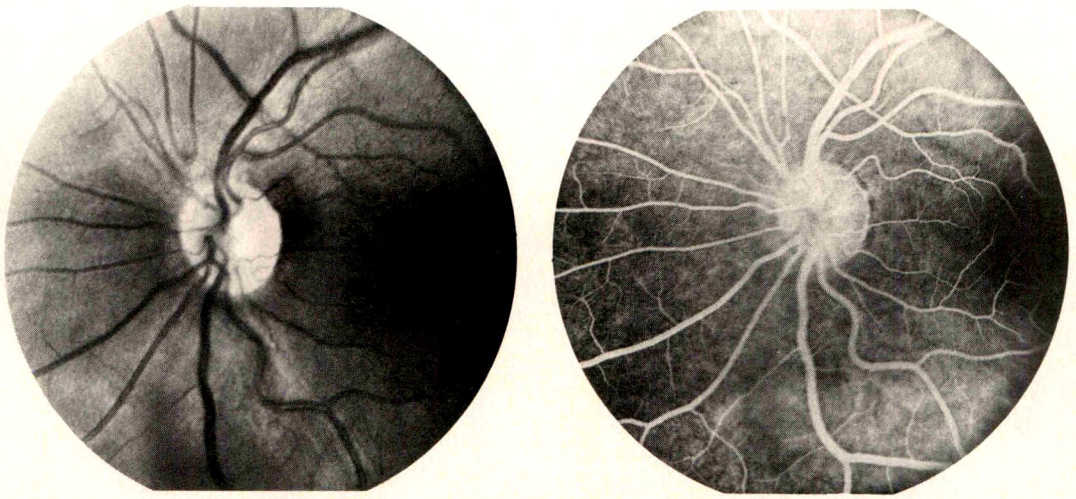


Fig. 3 (Kelly and Weiter). Case 1. Left eye after three months of therapy. Left, Resolution of neovascularization and subhyaloid hemorrhage. Right, Fluorescein angiogram also shows complete resolution.

(20/200) and L.E.: 6/6 (20/20). We administered a subconjunctival injection of 4 mg of dexamethasone and she began taking 100 mg/day of prednisone for seven days. This dosage was rapidly reduced over the next two weeks to 10 mg/day.

On subsequent examination, three months after institution of systemic corticosteroids, her anterior chamber reaction had decreased to trace flare and cells and only 1+ cells in the vitreous. A regressed fibrovascular network was now visible on the inferior portion of the optic disk in the right eye. The retinal vascular sheathing was still present but was not as extensive as on prior examinations.

Case 3—A 12-year-old girl came to us for evaluation of chronic uveitis in her right eye. She had a three-month history of blurred vision and photophobia in the right eye. There was no history of arthritis or other systemic illnesses. Her mother denied any prior history of pica and the family had never had pets.

On initial ophthalmologic examination, the visual acuity was R.E.: finger-counting at 1 foot and L.E.: 6/6 (20/20). The conjunctiva was minimally injected and we noted early band keratopathy in the cornea of the right eye. There were 2+ cells and flare in the anterior chamber of the right eye. The anterior segment examination of the left eye was normal. We saw early posterior subcapsular changes of the lens and 4+ cells and debris in the vitreous of the right eye on biomicroscopic examination. The optic nerve appeared edematous, with congestion of the vessels, and we noted cystoid macular edema in the right eye. We saw cells and thick exudates (snowbanking) inferiorly in the vitreous over the pars plana in the right eye. Detailed examination of the optic nerve, macula, and peripheral retina of the left eye gave normal results.

Subsequent laboratory evaluation disclosed a total eosinophil count of 624/mm^3 and a positive enzyme-linked immunoserum titer for *Toxocara canis* of 1:64. Westergren sedimentation rate, complete blood cell count, antinuclear antibody titer, and chest roentgenogram were normal.

We diagnosed *T. canis* uveitis and retinitis in the right eye. The patient began using topically applied prednisolone acetate, four times a day, and atropine 1%, once a day, for her anterior chamber inflammation.

The intraocular inflammation persisted during follow-up evaluations. Three months after her initial examination, we noted a vitreous hemorrhage in the right eye. This cleared over the ensuing six months, gradually allowing a view of the posterior pole which disclosed a neovascular frond on the optic nerve. We administered a subconjunctival injection of 4 mg of dexamethasone, and she continued to use atropine 1% once a day in the right eye. There were no longer cells or flare in the anterior chamber but 4+ vitreous cells and snowbanking persisted in the right eye.

Three months later, the vitreous cells and snowbanking had decreased markedly and the neovascular frond was regressing. Her visual acuity was finger-counting at 5 feet in the right eye and there were marked posterior subcapsular lenticular changes and residual vitreous debris.

Discussion

The optic disk appears to be more susceptible to developing neovascularization than is the peripheral retina.[2-4] This may be related to the thinness of and occasional gaps in the internal limiting membrane in this area,[5] drainage of vaso-

proliferative factors through the vitreopapillary pathway, resulting in increased exposure of this area,[2] involvement of the ciliary circulation in producing optic disk neovascularization,[6] or a combination of these factors. It is usually associated with conditions leading to an ischemic retina. Wise, Dollery, and Henkind[7] suggested that this is secondary to release of a vasoproliferative factor that may be a product of the switch to anaerobic metabolism found in hypoxic states. Removal of this hypoxia-induced neovasculogenic stimulus with panretinal photocoagulation results in regression of the neovascularization. The mechanism by which panretinal photocoagulation works is related to removal of hypoxic retina, either by destruction[2,6] or by increasing the oxygen levels.[8]

Previous reports have also associated optic disk neovascularization with inflammation.[1-3,9] This suggests that the inflammatory cells, the inflamed intraocular tissue, or other products of inflammation, are capable of inciting optic disk neovascularization, presumably, as in the case of hypoxic retina, by liberating a substance that diffuses back to the optic disk. In our cases, the neovascularization, associated with chronic uveitis, ocular sarcoidosis, and ocular *T. canis* organisms, regressed with resolution of the inflammation. This strongly suggests that the inflammation is causally related to the neovascularization and that reduction of the inflammation, by any means, will result in a regression of the neovascularization. However, additional cases are needed to confirm this association definitely.

Henkind[2] noted a similar process in other ocular tissues such as the cornea, iris, and choroid. Neovascularization appears to be an adaptive healing mechanism used by the body in response to various tissue insults. It is a dynamic process that requires a continuing stimulus, and will usually regress with the cessation of the stimulus. The present cases, in addition to those of retinal hypoxia-induced disk neovascularization, stress that removing the neovasculogenic factors (inciting causes) is the way to treat optic nerve head neovascularization.

Summary

Three cases of optic disk neovascularization associated with intraocular inflammation demonstrated progressive regression of the neovascularization with resolution of the inflammation, suggesting a causal relationship. These are the first reported cases, to the best of our knowledge, of regression of optic disk neovascularization in this situation.

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VITRECTOMY IN TERSON'S SYNDROME

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Terson[1,2] was the first to describe the association of vitreous hemorrhage, probably caused by a sudden increase in venous pressure that ruptures epipapillary and peripapillary capillaries, and subarachnoid hemorrhage. Recent reviews have indicated that roughly 20% of patients suffering subarachnoid or subdural hemorrhages develop associated intraocular hemorrhages.[3,4] Although most of these patients show evidence of intraretinal or preretinal hemorrhage, vitreous hemorrhage may also occur. As Khan and Frenkel[5] observed, most of these hemorrhages clear spontaneously and no ocular treatment is necessary. However, when the hemorrhage does not clear, significant reduction of visual function can result.

Our purpose is to report the results of pars plana vitrectomy in six eyes of five patients with Terson's syndrome who underwent surgical removal of nonclearing vitreous hemorrhage that had persisted for more than six months.

CASE REPORTS

Case 1—A 36-year-old woman was involved in an automobile accident on July 4, 1976. She underwent surgery twice for a right acute subdural hematoma on the day after the injury. She was comatose until Aug. 1, when she regained consciousness to find that she was virtually blind. Visual acuity had always been poor in her left eye; in 1974 she had undergone cosmetic strabismus surgery for left-sided esotropia. Because of the significant reduction in visual acuity in her right eye after the accident, she came here for examination.

Ocular examination on Aug. 20, 1976, disclosed a visual acuity of hand motions in the right eye and light perception in the left eye. Pupillary examination uncovered an afferent defect in the left eye. The anterior segment was within normal limits in both eyes. She had a dense vitreous hemorrhage in the right eye which obscured all fundus detail. Ophthalmoscopic examination of the left eye disclosed a hypoplastic optic nerve with a large macular scar. No vitreous hemorrhage was present in the left eye and the peripheral fundus was unremarkable.

Because we anticipated spontaneous resorption of the hemorrhage, we did not recommend treatment. When we re-examined her five months later, her visual acuity was still hand motions at 3 feet in the right eye and the vitreous hemorrhage was unchanged. Results of bright-flash electroretinography in the right eye were normal. Because the vitreous hemorrhage had not cleared and she was functionally blind, we recommended a vitrectomy in the right eye.

We performed a pars plana vitrectomy of the right eye on Jan. 28, 1977. During the operative procedure we found a clear lens but a densely hemorrhagic vitreous with a posterior vitreous detachment. After vitrectomy, the fundus in the right eye appeared normal. On the first postoperative day, the patient's vision improved to 6/12 (20/40); when we examined her five months postoperatively her visual acuity had improved to 6/6 (20/20+1) in the right eye. Although we have not examined her since May 1977, she told us three years after surgery that she has maintained normal visual acuity in her right eye.

Case 2—A 56-year-old man had had poorly controlled hypertension for 15 years before ocular examination in September 1979. In 1973 he had suffered a cerebral vascular accident that caused a coma which lasted for three days. Upon regaining consciousness, the patient noted reduced visual acuity in both eyes; this worsened over the following week to almost total blindness. An ophthalmologist noted bilateral vitreous hemorrhages and recommended observation. However, visual acuity did not improve, and the patient came here for examination several years later.

Ocular examination six years after the onset of visual symptoms disclosed visual acuity of hand motions at 1 foot with accurate light projection in both eyes. Pupillary examination uncovered a 1+ afferent defect in the left eye. A 15-prism diopter alternating exotropia was present. The anterior segment was normal in both eyes. Examination with the eyes dilated disclosed dense vitreous hemorrhages that obscured all fundus detail in both eyes.

Bright-flash electroretinography showed a normal

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a-wave and moderately abnormal b-wave in the right eye, and a two-thirds normal a-wave and normal b-wave relative to the a-wave in the left eye. The visually evoked response was interpreted as normal in both eyes.

When he entered the hospital, his blood pressure was 220/130 mm Hg. He was treated with methyldopa, furosamide, and propranolol HCl which kept his blood pressure within the normal range.

We performed an uncomplicated pars plana vitrectomy on his right eye on Sept. 11, 1979, and a similar procedure on his left eye on Sept. 17. At surgery we found a dense white vitreous hemorrhage with a posterior vitreous detachment in each eye. Ophthalmoscopic examination after vitrectomy showed marked narrowing of the retinal arterioles but an otherwise normal fundus in the right eye. In the left eye there was a zone of atrophy of the retinal pigment epithelium temporal to the fovea, but no other unusual findings. There was a clear lens in each eye.

Postoperatively, the patient did well and his visual acuity improved to 6/8 (20/25) in the right eye and 6/12 (20/40) in the left eye.

Case 3—A 30-year-old man had been in good health until Jan. 4, 1976, when he was involved in an automobile accident and was in a coma for 21 days. While hospitalized, he underwent a right-sided craniotomy for subdural hematoma. He had hemiplegia, a fractured clavicle, and a lacerated spleen that required abdominal surgery. He later underwent a cranioplasty to repair a skull defect. The hemiplegia largely resolved over a period of six months. When he regained consciousness after the accident, he noted a loss of the left peripheral visual field and stated that he had to turn his head to the left in order to see.

Ocular examination on July 25, 1978, disclosed a best corrected visual acuity of R.E.: 1/60 (3/200) and L.E.: 6/6 (20/20). Confrontation visual fields confirmed a left-sided hemianopia. Pupillary examination did not uncover an afferent defect. Examination with the eyes dilated disclosed a very dense vitreous hemorrhage which obscured posterior fundus detail in the right eye; we saw the nasal retina peripherally and found that it was attached. In the left eye, the media were clear; the disk, vessels, macula, and periphery were normal. Results of ultrasonographic examination of the right eye were consistent with a vitreous hemorrhage without retinal detachment. Bright-flash electroretinogram in the right eye showed normal a- and b-waves.

Because the reduction in visual acuity had lasted so long, we recommended a vitrectomy in the right eye. We performed a pars plana vitrectomy on Sept. 15, 1978. His visual acuity improved postoperatively to 6/12 (20/40) within two weeks of surgery. Ocular examination in August 1979 disclosed a visual acuity of 6/9 (20/30) in the right eye and 6/6 (20/20) in the left eye. He was able to see better as a result of the improvement in the temporal field of vision in his right eye, as well as his improved visual acuity.

Case 4—A 47-year-old woman with hypertension came here complaining of poor visual acuity in both eyes of two years' duration. In September 1977 she was delirious, raving, and diaphoretic. While she was hospitalized, a lumbar puncture showed bloody cerebrospinal fluid. Computed axial tomography showed a hematoma in the right temporal area, but arteriography failed to pinpoint a cause for the bleeding. The examining neurologist could not see the fundus in the right eye and described a subhyaloid hemorrhage in the left eye. When her general medical condition improved, she was discharged from the hospital. There was no ophthalmologic follow-up. However, her visual acuity did not improve. Our examination in July 1979 disclosed a visual acuity of 6/60 (20/200) in the right eye and 1.2/60 (4/200) in the left eye. The anterior segments were normal. We found no pupillary defect. Examination with the eyes dilated showed bilateral dense white vitreous hemorrhages. We performed a vitrectomy in the left eye in August 1979. Visual acuity in her left eye improved postoperatively to 6/6 (20/20) within one month of surgery.

Case 5—A 45-year-old man had been involved in a motorcycle accident in June 1977 that resulted in a subdural hematoma. He underwent neurosurgical drainage of the subdural hematoma shortly after the injury. Subsequently he developed bilateral vitreous hemorrhages which obscured vision in both eyes. The hemorrhage in the right eye cleared spontaneously, and visual acuity improved. However, blurred vision persisted in the left eye, and he was referred here for evaluation in December 1978.

At ocular examination, his visual acuity was 6/6 (20/20) in the right eye and 1.2/60 (4/200) in the left eye. Confrontation visual fields were full in both eyes. Entoptic phenomena were present in the left eye but were less distinct than in the right eye. There was also a possible afferent pupillary defect in the left eye. Examination of the right eye while it was dilated showed clear media except for some old vitreous blood inferiorly. Ophthalmoscopy showed a normal, clearly visible fundus. Examination of the dilated left eye disclosed a dense vitreous hemorrhage obscuring all posterior detail. We saw the peripheral retina and found that it was attached.

Because of the reduction in visual acuity and useful visual field in the left eye, the patient underwent a vitrectomy on Dec. 22, 1978. At the time of surgery the vitreous was filled with dense degenerative hemorrhagic residue and there was a posterior vitreous detachment. The disk appeared normal, but there was some pigmentary mottling in the macula. The peripheral retina appeared normal.

Postoperatively he did well, and visual acuity was 6/60 (20/200) on the first postoperative day. He had maintained this level when last examined six months after vitreous surgery.

Discussion

Other investigators have reported intraocular hemorrhage in 10% to 40% of adults and 70% of children with subarach-

noid or subdural hemorrhage.[3,4,6,7] These hemorrhages were usually limited to the retina. Before 1962, there were only 16 case reports of vitreous hemorrhage caused by subarachnoid hemorrhage (Terson's syndrome).[8] In their retrospective review of 320 patients with spontaneous subarachnoid hemorrhage, Shaw and Landers[9] uncovered only seven cases of documented vitreous hemorrhage (2.6%). Fortunately, most of these hemorrhages clear spontaneously and visual acuity improves. However, nonclearing vitreous hemorrhages do occur. Castren[10] was, to our knowledge, the first to recommend treatment of these nonclearing vitreous hemorrhages. He aspirated the vitreous from three eyes of two patients in a procedure that he called a vitreous transplantation; visual acuity improved in all three eyes. Shaw and Landers[9] described a 4-month-old child with bilateral vitreous hemorrhages; they performed a vitrectomy in one eye.

All of our patients suffered vitreous hemorrhage in association with traumatic or spontaneous subarachnoid or subdural hemorrhage. The hemorrhages did not clear spontaneously. Three of the five patients were functionally blind, two because of bilateral involvement with vitreous hemorrhage and the third because of a large macular scar and optic nerve hypoplasia affecting the fellow eye. In all cases, there was a dramatic improvement in visual function after pars plana vitrectomy with the techniques described by Machemer.[11] The operative procedures were facilitated technically by the presence of a posterior vitreous detachment.

Five of these six eyes had visual acuities in the range of 6/6 (20/20) to 6/12 (20/40) after vitreous surgery. In the sixth eye, although visual acuity was only 6/60 (20/200) postoperatively, this represented a significant improvement in visual acuity and visual field, compared with the preoperative visual acuity of 1.2/60 (4/200) and reduced functional visual field secondary to the dense vitreous hemorrhage (Table). In all six eyes the lens was preserved and remained clear postoperatively.

We have been favorably impressed with the results of vitreous surgery in our patients. We recommend vitrectomy for treating significant, nonclearing vitreous hemorrhage associated with subdural or subarachnoid hemorrhage. Some precautions are, of course, necessary. There must be a thorough preoperative ophthalmalogic examination, including assessment of the pupils and visual fields and echographic and electroretinographic studies. There should be an observation period of at least six months before surgery is considered. If the fellow eye is functional, the observation period should be increased to one year.

TABLE

RESULTS OF VITRECTOMY IN TERSON'S SYNDROME

Case No., Age (yrs), Sex	Involved Eye	Duration of Hemorrhage (mos)	Visual Acuity	
			Preoperative	Postoperative
1, 36, F	Right	6	Hand movements	6/6 (20/20)
2, 56, M	Right	72	Hand movements	6/7.5 (20/25)
	Left	72	Hand movements	6/15 (20/40)
3, 30, M	Right	30	1/60 (3/200)	6/9 (20/30)
4, 47, F	Left	24	0.3/60 (1/200)	6/6 (20/20)
5, 45, M	Left	18	1.2/60 (4/200)	6/60 (20/200)

Summary

Five patients with nonclearing vitreous hemorrhage associated with subarachnoid or subdural hemorrhages (Terson's syndrome) underwent pars plana vitrectomy. All six eyes of the five patients had postoperatively improved visual acuity and visual function. We recommend treating this rare syndrome with vitrectomy after careful preoperative ophthalmologic examination and a minimum observation period of six months to one year.

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VISUAL ACUITY AND BINOCULARITY IN UNILATERAL HIGH MYOPIA

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Patients with unilateral high myopia have a poor prognosis for satisfactory binocular function and visual acuity in the more ametropic eye.[1,2] They are sometimes compared to individuals with unilateral, spectacle-corrected aphakia who have significant aniseikonia. The subject of binocularity has not been adequately studied. We have studied and analyzed the various components of the binocular relationship in patients with unilateral high myopia.[3]

SUBJECTS AND METHODS

Thirty-eight patients were selected. To qualify for inclusion, each had to have at least 5 diopters of myopic anisometropia, normal fundi, and sufficient maturity to respond to motor, sensory, and visual acuity tests. Four of the patients had been previously treated elsewhere with appropriate spectacle correction. There were 18 males and 20 females.

All patients had ophthalmologic and orthoptic evaluations at the beginning and throughout the course of the study. All were treated with full-spectacle correction. Additionally, most were treated with occlusion of the nonamblyopic eye. Some received other active orthoptic treatment, including amblyopia and fusion therapy. Six patients had strabismus surgery.

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RESULTS

The range of anisometropia was from 5 to 19 diopters with a median of 8 diopters. The more ametropic eye was the right in 20 cases and the left in 18 cases. The astigmatism in the more myopic eye ranged from 0 to 6 diopters with a median of 1.25. Twenty-five patients had an astigmatic difference between the eyes of 1.25 diopters or less. Of these, eight either had no astigmatism or equal astigmatism in both eyes. Thirteen patients had an astigmatic difference of 1.5 diopters or more.

The patients' ages when they were first seen ranged from 3 to 31 years with a median of 7 years. Thirty-four patients were first seen at age 12 years or younger. The median follow-up was two years.

Twelve patients attained a final visual acuity of 6/12 (20/40) or better. Four of these had initial visual acuities of 6/15 to 6/22 (20/50 to 20/70) and six had initial visual acuities of 6/24 to 6/60 (20/80 to 20/200). No patients with initial visual acuities of less than 6/60 (20/200) attained 6/12 (20/40) or better.

Many patients had unsteady central or wandering fixation when first examined. As treatment progressed, fixation improved until all but two patients developed central fixation. Neither of these two patients showed visual acuity improvement beyond 6/30 (20/100).

The direction of the initial and final deviation showed only a few changes during the course of the study. The final examination revealed that 18 patients (47%) had an exodeviation, 11 patients (29%) had an esodeviation, seven patients (19%) were orthotropic, and two

patients (5%) had a combination esodeviation-exodeviation. Of the six patients who had surgery, direction was changed in only one. This patient became esotropic after surgery for an exotropia.

Control of deviation is an important consideration in an investigation of binocularity. When last examined, the eyes of 29 patients (76%) were phoric, six (16%) were intermittent, and three (8%) were tropic. Eight patients showed a change during the sutdy. Four of the six patients showing better control had had surgery. Both patients showing worse control had also had surgery.

The initial sensory status was determined by testing patients immediately after they began to wear spectacles but before any other treatment. The patient was considered to have peripheral fusion if he fused the near Worth Test, the Titmus fly stereotest, or both. Central fusion was recorded if the patient fused the distance Worth Test, achieved a score of 100 seconds of arc on the Titmus dot or animal stereotest, or both.

When initially examined, four patients (12%) had central fusion and 19 patients (56%) had peripheral fusion. The final sensory examination revealed that of the 17 patients (45%) with central fusion, four had previously been in this category and nine improved from peripheral to central fusion. When last examined, ten patients (26%) had peripheral fusion; eight of the ten were in this category when initially seen. Of the six patients (17%) who had no fusion when initially seen, four remained unchanged, one developed peripheral fusion, and one developed central fusion postoperatively.

The relationship between age at the start of treatment and final visual acuity was studied. Four of eight patients (50%) treated at age 5½ years or younger achieved visual acuities of 6/6 to 6/12 (20/20 to 20/40), as did four of 15 patients (27%) treated between the ages of 6 and 8 years, and three of 11 patents (27%) treated at age 8½ years or older.

An examination of the relationship between final visual acuity and degree of anisometropia revealed that six of 16 patients (38%) with anisometropia of 5 to 7.75 diopters attained visual acuities of 6/6 to 6/12 (20/20 to 20/40). Five of 17 patients (29%) with anisometropia of 8 to 10.75 diopters and one of five patients (20%) with anisometropia of 11 or more diopters also attained visual acuities of 6/6 to 6/12 (20/20 to 20/40).

An examination of the relationship between anisometropia and final sensory status showed that 12 of 15 patients (80%) with anisometropia of 5 to 7.75 diopters, four of 15 patients (27%) with anisometropia of 8 to 10.75 diopter, and one of four patients (25%) with anisometropia of 11 or more diopters had central fusion. No patients without fusion had less than 8 diopters of anisometropia.

When evaluating final degree of control in relation to the amount of anisometropia, we found that 15 of 16 patients (94%) with anisometropia of 5 to 7.75 diopters were phoric. The eyes of 11 of 17 patients (65%) with anisometropia of 8 to 10.75 diopters and three of five patients (60%) with anisometropia of 11 or more diopters were also phoric.

Nine of 11 patients (82%) with visual acuities of 6/12 (20/40) or better, five of ten patients (50%) with visual acuities of 6/15 to 6/22 (20/50) to 20/70), and three of eight patients (38%) with visual acuities of 6/24 to 6/60 (20/80) to 20/200) had central fusion. No patient with less than 6/60 (20/200) visual acuity achieved central fusion.

Four of seven patients (57%) treated at age 5½ years or younger achieved central fusion, as did seven of 14 patients (50%) treated between the ages of 6 and 8 years and five of ten patients (50%) treated at 8½ years or older.

Discussion

Recent studies[4-7] have shown that many individuals with unilateral high myopia can achieve good visual acuity with treatment consisting of full-spectacle correction and occlusion.

Of our patients 32% achieved a final visual acuity of 6/12 (20/40) or better in the more ametropic eye. This is slightly less than the 41% mean of previous studies.[4-7] Lesser degrees of amblyopia correlated with better final visual acuity. However, an initial visual acuity of 6/24 to 6/60 (20/80 to 20/200) did not preclude good visual acuity. Four patients improved with spectacle correction only. Others[4,5,7] have found that a small percentage of patients improve with spectacles alone. It thus seems best to start treatment with spectacles only. If visual acuity does not reach a satisfactory level after a reasonable period of time, occlusion should then be started.

Neither the astigmatism in the more myopic eye nor the astigmatic difference between the eyes adversely affected the final visual acuity level. Of 12 patients who attained a final acuity of 6/12 (20/40) or better, six had astigmatism of 1.25 diopters or less and six at least 1.5 diopters in the more myopic eye. A final visual acuity level of at least 6/22 (20/70) was attained by 14 to 25 patients (56%) with an astigmatic difference between the eyes of 1.25 diopters or less and eight of the 13 patients (62%) with a difference of 1.5 diopters or more.

The likelihood of acquiring 6/12 (20/40) or better decreases as age at the start of treatment increases. However, some patients were able to reach this level when treatment started at an age generally considered to have a poor prognosis. This supports the observations of others,[5,8] that patients with high anisometropia can respond to amblyopia treatment as late as age 14 years.

Three of the four patients (75%) whose eyes were initially tropic had an initial visual acuity of less than 6/60 (20/200). This compares with 26% of the total group with an initial visual acuity of less than 6/60 (20/200). This suggests that strabismus may have contributed to the amblyopia in these four patients. None had more than 8.75 diopters of anisometropia. By contrast, previous reports[4,7] found no relation between manifest strabismus and the degree of amblyopia in unilateral high myopia. The strabismic patients had gains in visual acuity similar to those of other patients. This agrees with other reports,[4,5,7] which showed that strabismus was not a poor prognostic sign for visual acuity improvement. However, it differs from the finding of Curtin and Schlossman[6] that strabismus has an adverse affect on visual improvement.

The degree of anisometropia was inversely related to final visual acuity. However, even high degrees of anisometropia did not preclude good visual acuity. One patient with 11.75 diopters of anisometropia improved to 6/6 (20/20).

There are several possible explanations for the inverse relationship between anisometropia and fusion. The patients with lower degrees of anisometropia had better visual acuity than patients with higher degrees. Better visual acuity correlates with better fusion. The eyes of patients with lesser degrees of anisometropia were phoric more often than those of patients with higher degrees. There is a better prognosis for binocular function without manifest strabismus.

Aniseikonia is considered to be a concomitant of anisometropia and an obstacle to binocularity.[1,2] A consideration of the two types of anisometropia and their relation to aniseikonia is important.

In refractive anisometropia, such as unilateral aphakia, spectacle correction will produce significant aniseikonia. The

image size difference is lessened with a contact lens. In axial anisometropia, such as unilateral high myopia, correction with a spectacle lens at the anterior focal point of the eye should not produce aniseikonia. The retinal image should be the same size as in an emmetropic eye. This principle is known as Knapp's Rule.[9] Contact lens correction should induce aniseikonia in these patients. If the spectacles are not at the anterior focal point, aniseikonia will also be induced. It will be roughly proportional to the anisometropia. This can also explain the inverse relationship between anisometropia and fusion, since we never know the precise position of the anterior focal point of the eye.[9] Additionally, spectacles do not stay in precisely the same position all the time.

Furthermore, the applicability of Knapp's Rule has been questioned. One study found large amounts of aniseikonia in four cases of spectacle-corrected unilateral high myopia.[4] This may be the result of retinal stretching and secondary micropsia. There may also be a small refractive component in unilateral high myopia. Thus, there is no clearcut optical advantage to either spectacle or contact lens correction in terms of reducing aniseikonia. However, aniseikonia may not be as serious an obstacle to fusion as often thought.

Lubkin and Linksz[10] recently reported that 27% of a group of patients with monocular aphakia fitted with phakic-aphakic spectacles adapted totally and 24% adapted almost totally. They overcame both the aniseikonia and the diplopia in different directions of gaze caused by the variable-induced prism. This ability to overcome both aniseikonia and anisophoria may explain why people with unilateral high myopia have been reported to have normal binocular vision with either spectacle or contact lens correction.[4]

Four of our patients had contact lens correction. Their fusion was similar to that achieved when they had spectacle correction. No patient with either spectacle or contact lens correction described aniseikonia or diplopia. Full-spectacle correction was well tolerated by 24 (63%) of our patients and moderately well by an additional six (16%). Perhaps, those patients who did not tolerate full-spectacle correction could not accurately describe the aniseikonia or anisophoria which they experienced. A significant refractive error in the less ametropic eye encouraged acceptance of the spectacles. Contact lenses often presented tolerance problems in these young children. Therefore, we recommend beginning treatment with full-spectacle correction. Contact lenses can be tried later, if desired.

Age at the start of treatment is not crucial to the development of central fusion. The median age at the start of treatment was 7 years for those classified as having either central or peripheral fusion and 9 years for those having no fusion when last examined. Approximately one half of the patients in the various age groups studied had central fusion on the last examination. The gains in binocularity achieved during the treatment period were maintained in the follow-up period of more than six years for 12 of the patients. After the desired results were attained, constant use of the full correction was not essential to maintenance of a good binocular relationship. Several patients have maintained good binocularity and good visual acuity with part-time use of spectacles or a balance lens for the more myopic eye.

Summary

Thirty-eight patients with unilateral high myopia ranging from 5 to 19 diopters were followed up for a median of two years. All were treated with full-spectacle correction and most had occlusion. When

initially examined one patient (3%) had visual acuity of 6/12 (20/40) or better, four patients (12%) had central fusion, and 24 patients (63%) were phoric. At the conclusion of the study 12 patients (32%) had visual acuities of 6/12 (20/40) or better, 17 patients (45%) had central fusion, and 29 patients (76%) were phoric. Age at the start of treatment was not a factor in the attainment of central fusion but did adversely affect the response to amblyopia treatment. The degrees of anisometropia was inversely related to both final fusion and final visual acuity.

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CLINICAL ANOPHTHALMIA ASSOCIATED WITH CONGENITAL CYTOMEGALOVIRUS INFECTION

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The ocular manifestations of congenital cytomegalovirus infection include chorioretinitis (the most common, seen in 5% to 29% of selected cases[1-3]), optic nerve hypoplasia and coloboma,[4] optic nerve atrophy,[2,5-7] uveitis,[6] cataract,[8,9] Peters anomaly,[7] and microphthalmia.[4,10,11] We describe three cases of clinical anophthalmia associated with congenital cytomegalic inclusion infection. We have published selected studies from two of the cases as part of a larger series.[7] To our knowledge, these are the first published reports of congenital clinical anophthalmia associated with congenital cytomegalovirus infection.

CASE REPORTS

Case 1—A 1,950-g boy was born two weeks prematurely to a 26-year-old woman who had had a chronic cold during the first trimester of her pregnancy. His gestational age was estimated to be 35 weeks and the placenta was small. He had marked postnatal respiratory distress secondary to meconium aspiration and congenital heart disease. He had anophthalmia of the right eye and horizontal eyelid fissures of 10 mm in the right eye and 15 mm in the left eye (Fig. 1). The left eye was normal. We noted mild thrombocytopenia with infrequent petechial skin hemorrhages. Neurologic evaluation disclosed microcephaly and nonspecific electroencephalographic abnormalities without periventricular calcification. We saw no hepatosplenomegaly or jaundice. Infant and maternal urine cultures grew cytomegalovirus. The infant's serum antibody titers for cytomegalovirus were positive (1:256) at birth by indirect hemagglutination and less than 1:8 by indirect immunofluorescent-IgM studies. A maternal indirect hemagglutination antibody titer done at the same time was 1:64. A rising indirect hemagglutination antibody titer of 1:4096 two months later indicated continued active viral infection in the infant. Serologic studies for herpes simplex virus, rubella, and toxoplasmosis were negative. Results of chromosomal studies were normal. B-scan ultrasonographic examination of the right orbital contents disclosed a severely microphthalmic eye associated with a cyst of equal size (Fig. 2).

Case 2—We examined a boy with bilateral anophthalmia when he was 5 months old. His 21-year-old mother had had an uncomplicated pregnancy. He was a well-developed, mesocephalic child without demonstrable neurologic deficits. Viral urine cultures grew cytomegalovirus and cytomegalovirus antibody titers were postitive at 1:1024 (indirect hemagglutination) and 1:4 (indirect immunofluorescent-IgM).

Case 3—We examined a boy with anophthalmia of the right eye when he was 11 months old. Anophthal-

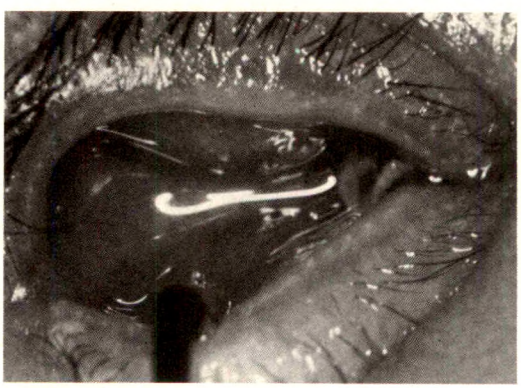


Fig. 1 (McCarthy and associates). Case 1. Anophthalmia of the right eye shown by socket dilation with conformers when the infant was 3 months old.

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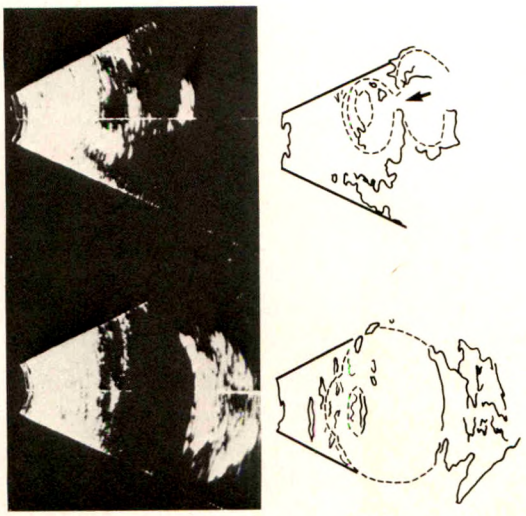


Fig. 2 (McCarthy and associates). Case 1. Top, Severe microphthalmia of right eye with posterior cyst joined by narrow isthmus (arrow). Bottom, Normal left eye.

mia was confirmed when the boy was examined under anesthesia. Antenatal history included hyperemesis gravidarum and cystitis in the first trimester with later polyhydramnios and premature rupture of the membranes. Birthweight was 2,400 g. On the first day after birth, the infant underwent surgical correction of pyloric atresia and microgastrium. By the age of 11 months, marked psychomotor retardation, microcephaly, and seizure activity were evident. An electroencephalographic examination later confirmed diffuse cerebral dysfunction. We noted optic nerve atrophy with roving movements of the left eye. A urine culture for cytomegalovirus was positive. Cytomegalovirus serum antibody titers were positive at 1:16 (indirect hemagglutination) and 1:4 (indirect immunofluorescent-IgM).

Discussion

Seroepidemiologic surveys have found that approximately 6% of pregnant women acquire cytomegalovirus infection during pregnancy.[12] Cytomegalovirus can be cultured from the urine in up to 3.5% of pregnant women.[13] Some prospective studies have shown that neonatal congenital infection rates range from 0.5% to 2% of all live births.[14] However, despite the relatively high rate of congenital infection, the rate of congenital defects is considerably smaller, in the range of 0.01% to 0.1% of all live births, depending upon the thoroughness of the investigation. In one prospective study, 20% of congenital cytomegalovirus infections caused congenital defects.[15] This discrepancy between infection and defect rates may be explained by maternal cell-mediated immune defects that allow more severe intrauterine damage.[16]

Although two of our three cases were not diagnosed at birth, they fullfill the diagnostic criteria for congenitally acquired cytomegalovirus infection: stigmata consistant with the TORCH syndrome (toxoplasmosis, rubella, cytomegalic virus, herpes simplex), persistant cytomegalovirus isolation from the urine, persistantly high serum titers against cytomegalovirus, and the absence of serologic and virologic evidence for other causes of the stigmata.[7]

The severely affected neonate with congenital cytomegalovirus infection may have the whole range of symptoms: low birthweight for gestational age, hepatosplenomegaly with jaundice, petechiae, thrombocytopenia, pneumonia, nephrosis, gastroenteritis, microcephaly, cerebral calcifications, deafness, psychomotor retardation, seizures, and electroencephalographic abnormalities. The degree of ocular involvement is highly variable. McCracken and associates[1] discovered only one case of chorioretinitis without other ocular abnormalities in 20 severe cases of cytomegalovirus affliction. Conversely, ocular involvement does not necessarily imply central nervous system or visceral abnormalities.[4] The teratogenic effects of the virus have been suggested by abnormalities of the first embryonic arch.[1] Previous reports of ocular teratogenesis have been restricted to colobomas of the optic nerve and microphthalmia.

The term clinical anophthalmia denotes the absence of clinically detectable ocular structures.[16] The differentiation of true anophthalmia from clinical anoph-

thalmia with severe microphthalmus requires histopathologic examination. However, ultrasonographic studies may be helpful, as shown by Case 1. In severe microphthalmia, involution of formed structures occurs between the third and eighth weeks of gestation, after development of the optic vesicle.[17] True anophthalmia indicates failure in the development or early involution of the primary optic vesicle in the second or third week of gestation.[18] The extreme variability of the virus and its protean manifestations may allow for unequal involvement of the ocular tissues.

Although severe intrauterine infection during the first trimester of pregnancy may cause spontaneous abortion, some pregnancies are carried to term. This has been well documented in studies of rubella epidemics.[19,20] Because maternal cytomegalovirus infections tend to be sporadic rather than epidemic, often without specific clinical signs, there are few reports of cytomegalovirus infection in the first trimester of pregnancy.[21,22] Indirect evidence of first-trimester infection comes from the presence and pattern of dystrophic intracranial calcification which suggests, based on the chronology of central nervous system development, that intrauterine infection occurs before or during the third month of gestation.[23,24] A prospective study indicated that second-trimester infections may result in congenital defects, whereas third-trimester infection produced no demonstrable abnormalities.[25] These data suggest that first-trimester infection causing severe ocular abnormalities does not necessarily result in spontaneous abortion.

The frequent occurrence of neonatal cytomegalovirus infection and the rarity of clinical anophthalmia may mean that these two findings were coincidental in our three cases. However, since the cause of clinical anophthalmia has been established only rarely,[17] our findings indicate that complete viral cultures and serologic studies should be carried out in both neonate and mother when severe ocular anomalies are discovered. Only by thorough evaluation of infants with anophthalmia can the role of cytomegalovirus infection be definitively established.

Summary

Three cases of congenital clinical anophthalmia associated with congenital cytomegalovirus infection were diagnosed by positive viral cultures and immunologic studies. Ultrasonography in one infant with clinically detected anophthalmia showed the presence of severe microphthalmia with cyst. These findings suggest that maternal infection occurred within the first trimester of gestation.

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SEPARATION OF THE IRIS PIGMENT EPITHELIUM

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Schmitt first described splitting of the iris stroma in 1922.[1] Since then more than 40 articles about stromal iridoschisis have been published.

I report a case of iris pigment epithelium separation with complete retraction of the anterior epithelial layer from the posterior epithelial layer.

CASE REPORT

A 36-year old woman came to the hospital from a tavern after a heavy drinking glass struck her face. On examination visual acuity was R.E.: light perception with projection, and L.E.: 6/6 (20/20) and 4 pt. By slit-lamp examination a right corneoscleral laceration with iris prolapse was present at 7:30 o'clock. The anterior chamber was shallow and contained dispersed pigment, fibrin, and cells. A thick mat of consolidated fibrin completely covered a small, irregular pupil and obscured visualization of the lens and the posterior pole.

Results of a general physical examination were unremarkable. Orbital X-rays were normal.

After administering general anesthesia, I explored the injury more thoroughly. I found a triangular-shaped lamellar corneal flap. Its base abutted the corneoscleral limbus at 5:30 and 7:30 o'clock. The medial margin was only a partial-thickness tear, but the lateral edge was a full-thickness penetration through which uveal tissue prolapsed. The lateral laceration extended 2 mm past the corneoscleral limbus into sclera. The small area of lens visible under the iris defect appeared to be clear.

I excised prolapsing iris and closed the corneal and scleral lacerations with eight interrupted 10-0 nylon sutures. I closed the conjunctiva with 7-0 chromic sutures.

Results of a microscopic examination of sections of excised iris tissue, stained with hematoxylin and esoin and bleached, were unremarkable.

Postoperative slit-lamp examination disclosed a separation of the pigment epithelium extending 300 degrees from both margins of the surgical sector iridectomy. The pigment epithelial pupillary margin was tightly adherent to the lens capsule by posterior synechiae and a dense pupillary occlusion. The posterior pigment epithelium was stretched taut over the lens capsule. It was intact except for the surgical iridectomy and a small oval tear at 3 o'clock (Fig. 1). Irregular pupillary ruff remnants surrounded the 3-mm pigment epithelium pupillary margin. The iris stroma and the dilator pupillae and sphincter pupillae muscles were retracted, forming a 7-mm pupil diameter. Both the anterior iris stromal surface and the stromal pupillary margin were smooth. The anterior iris pigment epithelium was not visible under the iris stroma. The lamellar corneal section lay flat against the cornea. The lens was clear.

When she looked through the small tear in the iris pigment epithelium, the patient's visual acuity was 6/60 (20/200) on the first postoperative day. Once the consolidated fibrin cleared from the visual axis, her visual acuity improved to 6/9 (20/30), correctable to 6/6 (20/20) with +0.50 +0.75 × 30. After several weeks the posterior pigment layer retracted and curled back upon itself as a large ectropion uvea. It did not reattach to the back of the iris (Fig. 2).

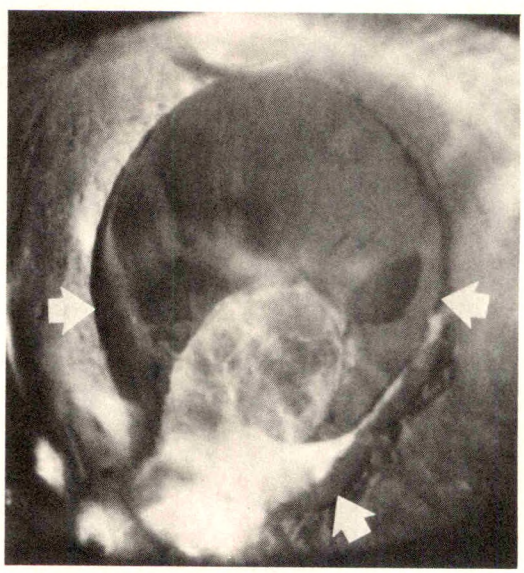


Fig. 1 (Sidrys). Slit-lamp photograph of the anterior segment on the first postoperative day. Posterior pigment epithelial layer is bound to the lens by an occlusion of the pupil. An oval tear is present at 3 o'clock. Arrows indicate the retracted anterior border of the pupillary ruff.

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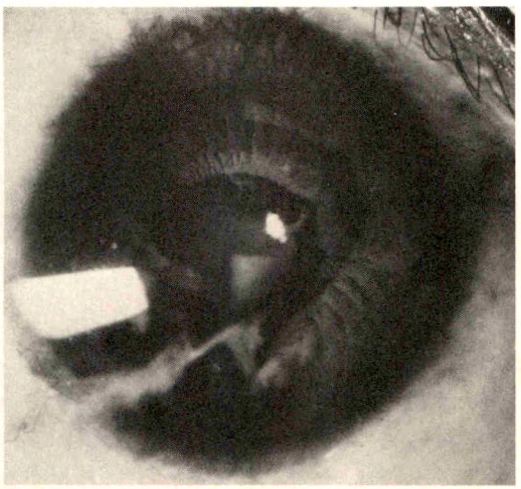


Fig. 2 (Sidrys). Four months after surgery the visual axis has cleared and the posterior pigment layer has retracted and curled.

Discussion

The neuroectodermal layer of the globe is derived from an outpouching of the lateral wall of the primitive forebrain, which forms the embryonic cup. The apposed layers form the sensory retina and the pigment epithelium. Anteriorly, the apposed layers come together as the double-layered epithelium of the ciliary body and the iris and fuse at the pupil to form the pigment seam, or pupillary ruff. Failure of the lumen to close completely in the pupillary area creates a space known as the marginal sinus of von Szily. In the normal iris pigment epithelium, small spaces are frequently present between the two epithelial layers.[2] These spaces are also remnants of the lumen of the optic vesicle and persist throughout life. The apical villi of the apposed cell layers project into these spaces.

The dilator pupillae muscle originates in the neuroepithelium and remains in anatomic continuity with the underlying anterior layer of the pigment epithelium by cytoplasmic processes.[2] The posterior pigment epithelium, however, has very weak attachments to the anterior layer and may even separate spontaneously from the anterior layer to form free-floating or fixed pigmented cysts within the anterior chamber.[3] In most cases of pigment epithelium separation, the posterior pigment epithelium remains hidden behind the rest of the iris and is not visible clinically.

In this 36-year old patient sequential trauma, inflammatory synechiae, surgical iridectomy, and mydriatic agents created unusual lines of force within the iris. The retracting dilator pupillae muscle tore the anterior pigment epithelium layer loose from the adherent pupillary margin while the posterior layer remained stretched flat against the lens capsule. This provided a rare view of the inner surface of the posterior pigment epithelial layer, the inside surface of the embryonic cup.

Many cases of stromal iridoschisis have been documented. It typically occurs in elderly individuals after trauma, long-term iridocyclitis, or the use of miotics.[4] The posterior stromal tissue remains attached to the pigment epithelium while the free ends of the atrophic anterior stromal fibers float freely in the aqueous humor.[4] In this case the anterior stromal surface remained smooth and unaffected.

Separation of the iris pigment epithelium may not be uncommon. It becomes clinically visible, however, only if the pupillary ruff is torn and the posterior pigment epithelium layer remains adherent to the lens when the anterior layers of the iris retract.

Summary

Surgical repair of traumatic corneal and scleral lacerations with uveal prolapse postoperatively disclosed a separation of the pigment epithelium. Posterior synechiae and a dense occlusion of the pupil tightly bound the papillary margin to the

lens capsule. Traumatic, surgical, and mydriatic agents tore the anterior pigment epithelium layer loose from the adherent pupillary margin and induced a complete separation of the iris along the original lumen of the embryonic cup, between the two epithelial layers. Once the consolidated fibrin cleared, the posterior pigment layer retracted from the visual axis but did not reattach to the iris.

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Fifty years ago this month in THE JOURNAL:

Of late there have appeared in the continental papers various articles concerning the replacement of the usual ophthalmic lenses, in spectacle or pince-nez form, by contact glasses. In due course of time we may expect some similar articles, but on a more sensational basis, in our own press. These which have appeared in Europe were inspired by Heine of Kiel, who spoke at the last international congress, in Amsterdam, on such usage and demonstrated a few patients whose vanity led them to the use of the inconspicuous contact glass to replace the usual spectacles. Undoubtedly such individuals do exist, but fortunately they are few in number.

R. Von Der Heydt and Harry Gradle,
Concerning contact glasses.
Am. J. Ophthalmol. 13:867, 1930

BROWN TUMOR OF THE ORBIT ASSOCIATED WITH PRIMARY HYPERPARATHYROIDISM

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Tumors containing giant cells rarely involve the orbit. We were able to find only seven cases of brown tumor involving the orbital bones in published reports (Table).[1-6] In only one case was there involvement of the frontal bones.[1] Our patient was, to our knowledge, only the second example of frontal bone involvement in brown tumor.

The external, radiographic, and histologic appearances of the various giant-cell-containing tumors are sufficiently similar to warrant a review of the differential diagnoses of these lesions.

From the Eye Pathology Laboratory, Wilmer Ophthalmological Institute (Mr. Naiman, Mr. d'Heurle, and Drs. Green and Iliff); the Departments of Medicine (Dr. Benedict) and Pathology (Dr. Eggleston), Johns Hopkins Medical Institutions; and the Pathology Department, Sinai Hospital of Baltimore (Dr. Dorfman), Baltimore, Maryland.

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CASE REPORT

A 70-year-old woman had a two-year history of vague scalp tenderness and a one-year history of ankle and knee tenderness, but she was otherwise in good health. In September 1976, she noticed a nontender, slowly enlarging mass on the left superior orbital rim. Roentgenograms of this area disclosed an osteolytic lesion and the patient was referred to one of us (W. J. I.).

The patient was asymptomatic except for the mass above her left eye. She denied any history of trauma to this area. Physical examination revealed a smooth, firm, fixed mass, 24 mm in diameter, in the mid portion of the left superior orbital rim (Fig. 1). There was 3 mm of blepharoptosis but no proptosis of the left eye. Visual acuity with correction was 6/7.5 (20/25) in the right eye and 6/12 (20/40) in the left eye. Laboratory tests done postoperatively gave the

TABLE

BROWN TUMORS OF THE ORBIT

Study	Patients' Age (yrs), Sex	Location of Tumor	Symptoms	Primary or Secondary Hyperparathyroidism	Treatment of Bone Lesion
Siedenbiedel[1]	22, M	Right frontal bones	Mass	Primary	Surgical excision
Bedard and Nichols[2]	48, M	Right maxillary bone	Nasal obstruction; swelling	Primary; adenoma	Surgical excision
Friedman, Pervez, and Schwartz[3]	21, F	Right maxillary bone	Pain; swelling; tearing; proptosis	Secondary; familial nephritis	Surgical excision
Gay, Viskoper, and Chowers[4]	41, M	Left maxillary bone	Swelling; proptosis	Secondary; renal tubular acidosis	Intraoral biopsy
Hamlin and Lund[5]	37, F	Left maxillary bone	Mass; pain; swelling	Primary; adenoma	Biopsy; radiation
Ferry[6]	17, F	Right maxillary and ethmoid bones	Proptosis	Secondary; familial nephritis	Surgical excision
Ferry[6]	24, F	Right ethmoid bone	Proptosis; diplopia; pain	Secondary; chronic glomerulonephritis	Surgical excision
Present study	70, F	Left frontal bone	Mass	Primary; carcinoma	Surgical excision

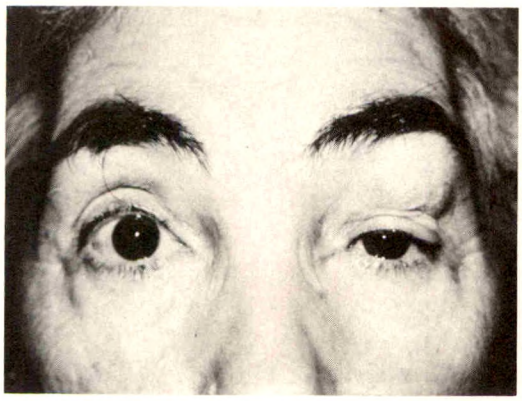


Fig. 1 (Naiman and associates). External appearance of lesion of frontal bone with orbital and eyelid involvement.

following values: serum calcium, 12 mg/100 ml (normal, 9 to 11 mg/100 ml); phosphorus, 2.7 mg/100 ml (normal, 3.0 to 4.5 mg/100 ml); and alkaline phosphatase, 46 IU/liter (normal, 10 to 32 IU/liter). Repeat X-ray films of the frontal bone disclosed a lytic lesion in the area of the left superior orbital ridge, with destruction of the roof of the orbit (Fig. 2). There had been a slight enlargement of the lytic lesion since the previous X-ray films had been taken two months earlier. The bone density was normal in other areas of the skull. Retrospective review, however, showed features suggestive of resorptive changes.

With the patient under general anesthesia, we removed, with some difficulty, a 20 × 15 × 15-mm soft gray mass from the left orbital plate of the frontal bone and frontal sinus through a brow approach on Nov. 12, 1976. We also removed similar tumor material extending into the left ethmoid and nasal sinuses. The frontal bone in the area of tumor was eroded and friable. The dura mater was exposed in one area. We submitted a portion of the specimen for frozen sections at the time of surgery. These were

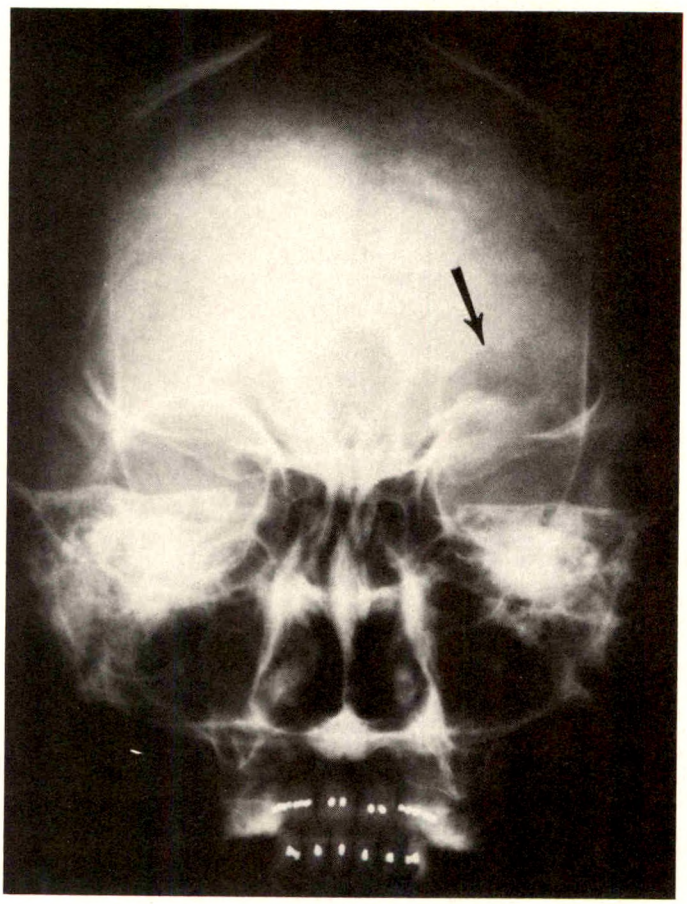


Fig. 2 (Naiman and associates). Osteolytic lesion (arrow) of left frontal bone.

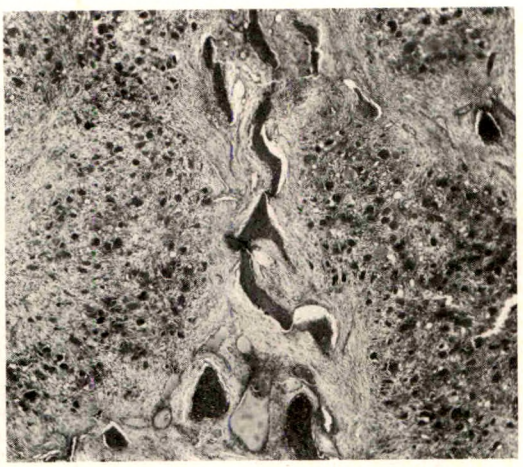


Fig. 3 (Naiman and associates). Microscopic appearance of brown tumor with a nodular distribution of giant-cell aggregates separated by fibrous tissue stroma and spicules of reactive new bone (hematoxylin and eosin, ×40).

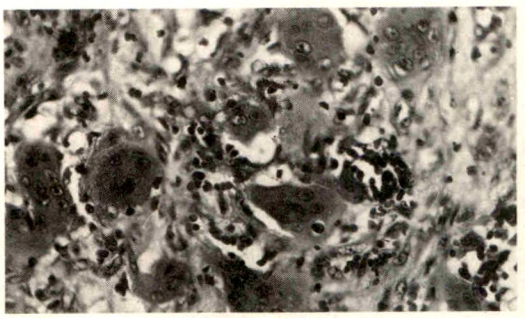


Fig. 4 (Naiman and associates). Higher magnification of brown tumor showing a prominent cluster of giant cells separated by moderately pleomorphic spindle-shaped cells (hematoxylin and eosin, ×400).

interpreted as showing a giant-cell tumor. After the extensive surgery, we believed that we had removed all of the tumor.

Grossly, the specimen consisted of several firm brownish fragments of tissue, the largest measuring 28 × 18 × 12 mm. Histologic examination disclosed an encapsulated tumor composed of nonuniform nodular aggregates of giant cells, separated by a fibrous stroma and spicules of bone (Fig. 3) . The giant cells contained between five and 30 spindle-shaped and oval nuclei (Fig. 4). Most nuclei had clear nucleoplasm and one nucleolus; we saw no evidence of mitoses. Hemosiderin pigment was present in the mononuclear cells, which were located in the collagenous stroma of the tumor and occasionally in the multinucleated cells. Some of these pigment-laden cells were in the neighborhood of giant-cell clusters. Additionally, there was prominent osteoid formation in the stroma that was not related to the reactive bone spicules previously mentioned. Although there was some pleomorphism of the spindle-shaped cells in the stroma, there was no evidence to suggest malignancy. At one side of the specimen, tumor material was present at the surgical margin. Our initial impression was that the tumor was probably a true giant-cell tumor. A consultant (H. D. D.) first suggested that it was a brown tumor.

After excision of the orbital tumor, we found that the patient had increased parathyroid hormone levels. On selective catheterization of the left inferior thyroid vein, this level was as high as 3,609 ng/ml (normal range, 163 to 347 ng/ml), and we diagnosed primary hyperparathyroidism.

On Jan. 20, 1977, the patient underwent a cervical exploration, and we discovered a parathyroid lesion adhering to the left side of the thyroid gland. The mass was a dark red, smooth, elongated, oval nodule weighing 12.2 g and had a relatively homogeneous light tan cut surface.

Microscopic examination showed a parathyroid carcinoma consisting of large chief cells with distinct cell borders and uniform, large, slightly irregular nuclei containing a moderate amount of chromatin. Scattered tumor cells were in mitosis, and we found as many as three mitotic figures per high-power field (Fig. 5). Irregular fibrous bands extended from the capsule into the tumor, and we identified both capsular and vascular invasion. A small amount of residual normal parathyroid was present adjacent to the tumor.

Follow-up in August 1979 showed the patient to be in good health with no evidence of recurrence of the

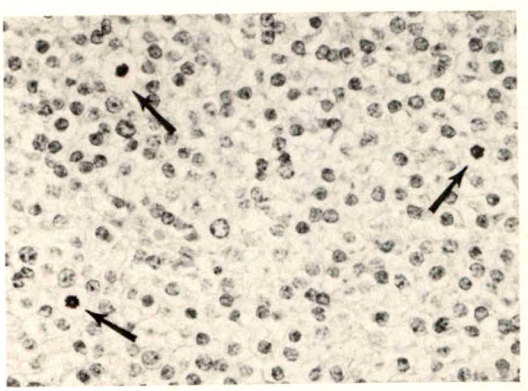


Fig. 5 (Naiman and associates). Area of parathyroid carcinoma showing large chief cells with distinct cell borders and slightly irregular nuclei. Three mitotic figures (arrows) are present in this field (hematoxylin and eosin, ×350).

orbital or parathyroid tumors. The patient has been asymptomatic since the initial procedure.

Discussion

Differential diagnoses of giant-cell-containing tumors of the orbital bones include giant-cell tumor, giant-cell reparative granuloma, brown tumor of hyperparathyroidism, and aneurysmal bone cyst. The radiographic and histologic appearance of these various lesions is often nonspecific,[2,5,7,8] and diagnosis requires histologic samples, serum analysis, and a knowledge of the clinical appearance.[9-11] Because the lesions are heterogeneous, biopsy must be sufficient to ensure sampling of all representative areas of the tumor.

Giant-cell tumor—Giant-cell tumors characteristically occur at the ends of long bones.[7-9,11,12] The incidence in males and females may be equal[8] or may be greater in females.[7] The lesion usually occurs in patients more than 20 years old.[7-9] Radiographic examination of giant-cell tumors discloses osteolysis (usually not penetrating through the cortex) with expansion and thinning of the cortex, and little or no new bone formation.[8,13] The usual occurrence of giant-cell tumors in the epiphyseal region has led to speculation that giant cells arise from osteoclasts known to be present in this region.[14] For this reason the tumor has frequently been called osteoclastoma[13-16] Jaffe, Lichtenstein, and Portis,[8] however, disagree, and state that the giant cells and stromal cells develop from the same cell line. Authors of more recent ultrastructural studies[16,17] give conflicting opinions about the osteoclastic origin of the giant cells in both true giant-cell tumor and giant-cell reparative granuloma.

Histologic examination has disclosed regularly dispersed giant cells[9] with a stroma of oval or spindle-shaped cells.[11] The nuclei of the stromal cells and giant cells are similar[7] or identical.[13] Osteogenesis is rare.[11,13] Jaffe, Lichtenstein, and Portis[8] classified giant-cell tumors in grades I to III on the basis of the degree of atypism of the stromal cells, compactness of these cells, and degree of collagenous differentiation. Grade III was the most malignant with metastases to the lungs being typical.[8,11] Thus, they believed that different grades required different treatments. The usefulness of this grading system has been questioned.[7,13]

Dahlin, Cupps, and Johnson[7] described a 19-year-old woman with recent onset of blindness who underwent subtotal removal of a giant-cell tumor of the sphenoid bone after receiving radiation treatment at another institution. Two years later she had a giant-cell tumor in the upper tibia and no clinical evidence of hyperparathyroidism. These authors did not mention the cause of the blindness.

In another case reported as giant-cell tumor, a 24-year-old man had exophthalmos of the left eye, progressing over six months. Visual acuity and eye movements were normal. At surgery the tumor, originating in the frontal sinus, was found to be depressing the roof of the orbit.[10]

In another report,[18] a 41-year-old man had had pain in the infratemporal region for one month. On X-ray examination the authors noted obscuration of the left innominate line and destruction of the posterior half of the left lateral wall of the orbit and the floor of the left middle cranial fossa. The bony destruction was sharply marginated. On biopsy, they found that the mass consisted of multinucleated giant cells and a loose stroma of strap-shaped mononuclear cells. They diagnosed a probable malignant giant-cell tumor. The patient received extensive radiation therapy but the outcome was uncertain when the report was published.

It has been difficult to determine the best treatment of giant-cell tumors, because many primary therapies and combinations of therapies have been used in the past.[7,19] However, in one series of 195 cases, 20 patients had either en-bloc resections or amputation of the tumor, and none of these patients had recurrences.[7] Since total resection of tumor from skull bones may be surgically difficult, radiation following partial resection has been recommended.[13] The recurrence rate may be as high as 45% in totally excised lesions with or without radiation, but this rate decreases when bone grafting is combined with surgical excision.[7]

Giant-cell reparative granuloma—This is a giant-cell-containing lesion that has been confused with true giant-cell tumor in the past.[5,9,11] This lesion is a reactive process which follows traumatic or infectious injury.[3,11] In contrast to true giant-cell tumors, giant-cell reparative granulomas occur in patients less than 20 years old who frequently give a history of trauma preceding the development of tumor.[9] The radiographic appearance is nonspecific.

Hirschl and Katz[9] described the histologic differences between giant-cell tumor and giant-cell reparative granuloma. In the latter there is an irregular distribution of the giant cells and hemorrhagic zones,[11] creating a granulomatous appearance.[9] Osteoid formation and spicules of new bone may be present.[5] The areas with fewer cells have collagen, oval cells, and spindle-shaped fibroblasts.[9] New and old hemorrhages may be prominent.

The reparative granulomas are more common in the skull bones than are giant-cell tumors.[5] Surgical excision is the preferred treatment.[5,9,14] Because of the nature of these lesions, radiation therapy should not be used unless surgical removal is impossible. Some investigators have reported malignancies developing after supposedly successful radiation treatment.[5,20]

A review of published reports disclosed two cases of possible giant-cell reparative granulomas involving the orbit:

1. A 45-year-old woman with a three-year history of "turning up" of the right eye had a 10-cm mass involving the anterior and posterior ethmoid sinuses, and destroying the lateral and medial walls of the right maxillary sinus as shown by X-ray examination. A biopsy diagnosis of giant-cell tumor was followed by radiation therapy. There was improvement of symptoms until 14 years later, when proptosis accompanied by photophobia and blurred vision occurred. Surgical decompression was done, but the authors reported a progressive increase in both the size of the tumor and severity of associated symptoms.[5]

2. A 21-year-old man had been struck in the left eye six months before admission and had had slow progressive proptosis of the eye, with intermittent diplopia, during the two months before admission. The surgeon removed an ethmoid mass that had broken into the orbit. Microscopic examination disclosed a giant-cell-containing lesion. One month later the patient had 7 mm of nonprogressive proptosis, with normal eye movements.[19]

Brown tumor—Brown tumor, which is synonymous with von Recklinghausen's disease of bone,[21,22] is not considered a true tumor, but rather a reactive process of hyperparathyroidism.[8,13] The osteolytic appearance of the lesions on X-ray film may be similar to those of giant-cell reparative granuloma, giant-cell tumor, and aneurysmal bone cyst. However, decreased bone mineralization secondary to the disease process may be present in association with a brown tumor. The histologic appearance of the brown tumor may be identical to that of a giant-cell

reparative granuloma.[2,5,9,11] Laboratory determinations of serum calcium, phosphate, alkaline phosphatase, and parathyroid hormone levels are useful in diagnosing brown tumor.[9,13]

Brown tumors with orbital involvement are rare; we found only seven published case reports.[1-6] Possibly, brown tumors of the orbital bones are not as rare as this suggests. Many cases with skull involvement in hyperparathyroid bone diseases are observed radiologically but never reported. The area of involvement in these seven cases was as follows: maxillary bone and adjacent areas in four,[2-5] ethmoid bone and sinus in one,[6] maxillary and ethmoid bones in one,[6] and frontal bone in one.[1] Four of the tumors were associated with primary hyperparathyroidism and four with secondary hyperparathyroidism. Proptosis and nasal obstruction were the predominating symptoms. Only one case was similar to our case, with tumor involving the frontal bone.[1] In all seven cases treatment was surgical excision of the tumor; in the cases of primary hyperparathyroidism, the parathyroid tumors were removed. One patient died from electrolyte imbalance after removal of a parathyroid adenoma.[5] On biopsy, the parathyroid glands of the patients with secondary hyperparathyroidism were grossly enlarged but showed no evidence of adenoma or carcinoma.[4]

In all four cases of primary hyperparathyroidism, including our own, the brown tumor disclosed the parathyroid disease. Parathyroid carcinoma is a much less frequent cause of hyperparathyroidism than are parathyroid adenoma or parathyroid hyperplasia of secondary hyperparathyroidism.

Aneurysmal bone cyst—This is a giant-cell-containing lesion of unknown origin that usually affects the ends of long bones or vertebrae. The typical patient has a fracture or enlarging mass during adolescence or childhood.[22,23] The radiographic appearance is a cystic mass with some calcification, expanding the bone from within.[13] Histologically, the lesion contains irregular cavities not lined by endothelium but filled with fresh blood.[11] Our review of published reports disclosed an unusual case of an intracranial aneurysmal bone cyst involving the superior orbital roof.[24] A 14-year-old girl had had vomiting and persistent headaches for one month. A bilateral sixth nerve palsy and papilledema and a slight right hemiparesis were present. Five years after surgical excision of the lesion, the patient was in excellent health.

A 10-year-old boy had optic nerve compression caused by an aneurysmal bone cyst.[25] He lost central vision in both eyes and was found to have an expanding mass arising in the sphenoid and ethmoid sinuses. Visual acuity had returned to nearly normal in both eyes one year after partial excision of the intracranial and extracranial portions of the lesion.

Although experience with these bone cysts in the skull is limited, the preferred treatment is probably surgical excision[11] with radiation reserved for inaccessible lesions.[13]

Other cases—Several other reports of giant-cell tumors involving the orbit have been published, but these reports preceded detailed classification of the tumors. Thus, tumors reported to be giant-cell tumors might be classified differently today.[12,26] In one instance, for example, a 5-year-old boy underwent orbital exenteration. Histologic examination disclosed a giant-cell-containing tumor, but the patient was lost to follow-up before von Recklinghausen's disease of bone (brown tumor) could be ruled out.[15]

Our patient with a brown tumor had a favorable outcome. Of the seven previously described patients, six recovered

and one died from postoperative complications of parathyroid surgery. We recognize, however, that the diagnosis of hyperparathyroidism should have been suspected and established before, or even without, orbital surgery. The appropriate serum chemistry tests should be done in all patients with an obscure lytic lesion of an orbital bone. The best treatment is either amelioration or eradication of the cause of the hyperparathyroidism.

SUMMARY

A 70-year-old woman had swelling of her left superior orbital ridge. A tumor was surgically removed, and histologic examination disclosed a tumor containing giant cells. Subsequent tests showed primary hyperparathyroidism caused by a carcinoma of the parathyroid. This rare instance of brown tumor of the orbit led us to review the differential diagnoses of giant-cell lesions of the orbit and to examine published case reports. Laboratory determinations of serum calcium, phosphorus, and alkaline phosphatase levels for all patients with lytic orbital bone lesions can establish whether they have hyperparathyroidism, a diagnosis making orbital surgery unnecessary.

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NOTES, CASES AND INSTRUMENTS

OCULAR INFLAMMATION IN REITER'S SYNDROME ASSOCIATED WITH *CAMPYLOBACTER JEJUNI* ENTERITIS

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Sebald and Véron[1] proposed the term *Campylobacter* as a generic name for microaerophilic *Vibrio* organisms which can cause diseases in domestic animals and enteritic infections in humans.[2] By selective culture, we can now isolate *Campylobacter* organisms from patients with diarrhea.[3] We describe a patient who developed bilateral conjunctivitis as a part of Reiter's syndrome after enteritis caused by *C. jejuni*, a bacterium which, to our knowledge, has not been previously described as a causative agent in Reiter's syndrome.

CASE REPORT

An 18-year-old woman became ill on Sept. 23, 1979, with fever, abdominal pain, and diarrhea lasting for one week and followed by asymmetric, acute polyarthritis that affected her fingers, toes, large weight-bearing joints, and sternoclavicular joints for two months. Two weeks after the onset of the fever she developed bilateral, acute conjuctivitis with smarting and burning sensation, lacrimation, mucopurulent exudation, and redness. The conjunctivitis resolved spontaneously in seven days. Conjunctivitis coincided with asymptomatic, sterile pyuria. We isolated *C. jejuni* from the feces. The serum of the patient showed agglutination antibodies against an autologous strain of *C. jejuni* in titers of 1:200 during the active stage of the illness and 1:50 during the convalescent period. Stool cultures for *Salmonella*, *Shigella*, and *Yersinia* organisms, as well as serological test titers for *Salmonella*, *Yersinia*, and *Chlamydia* organisms, were negative. The peak erythrocyte sedimentation rate was 95 mm/hr. The highest leukocyte count was 10,800/mm^3. Latex, Waaler Rose, and antinuclear antibody titers were negative. Antistreptolysin 0 titer was 200. The HLA phenotypes were A1, A2, B27, w4, w6, and Cw2. X-ray examination of the lumbosacral spine showed nothing abnormal.

DISCUSSION

To our knowledge, this is the first study of Reiter's syndrome associated with *C. jejuni* infection. In our patient, bilateral mucopurulent conjunctivitis, acute polyarthritis, and sterile pyuria developed one or two weeks after the onset of *C. jejuni* enteritis. We excluded other known causes of Reiter's syndrome. Ocular inflammation associated with *Yersinia* infection[4] and after *Salmonella* enteritis in Reiter's syndrome[5] occurred in patients with HLA-B27 antigen, which was also present in our case. Berden, Muytjens, and van de Putte[6] reported reactive arthritis associated with *C. jejuni* enteritis without conjuctivitis and dysuria in a patient with HLA-B27 antigen. These findings suggest that *C. jejuni* infection may occasionally cause ocular inflammation in Reiter's disease in HLA-B27-positive patients in the same way as it does certain other infections.[4,5,7]

SUMMARY

An 18-year-old woman developed acute polyarthritis one week and bilateral, acute, mucopurulent conjunctivitis and sterile pyuria two weeks after onset of *Campylobacter jejuni* enteritis. The conjunctivitis resolved spontaneously in one week and the arthritis in two months.

From the Departments of Ophthalmology (Dr. Saari) and Rheumatology (Dr. Kauranen), Institute of Clinical Sciences, University of Tampere, and Central Hospital, Tampere, Finland. This study was supported in part by the Finnish Rheumatism Association and the Medical Research Council of the Academy of Finland (Dr. Saari).

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The patient had HLA-B27 antigen. *Campylobacter* organisms may lead to Reiter's syndrome in a patient with HLA-B27 antigen.

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A NEW METHOD FOR FIXATION OF THE EYELID DURING TARSORRHAPHY

Karim F. To'mey, M.D.

Beirut, Lebanon

I have always found it exceedingly difficult to steady the eyelid edge while producing a raw surface on its margin in tarsorrhaphy. Moreover, unless some pressure is applied on the eyelid, there is always enough bleeding to obscure the small operative field. If a regular mosquito clamp is used to hold the eyelid, it has to be taut to prevent slipping and to achieve adequate hemostasis, and thus may crush the tissues. A chalazion clamp does not provide proper exposure of the eyelid margin and also slips frequently.

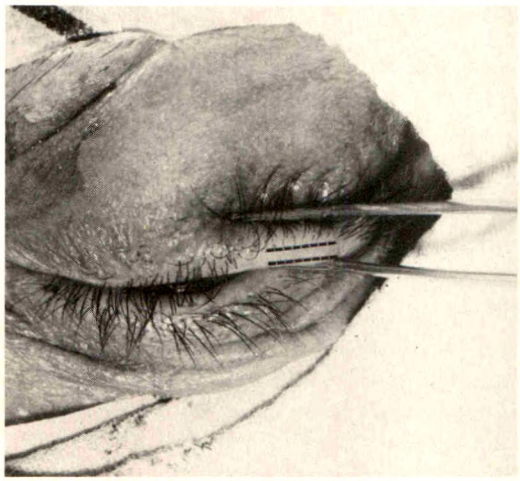


Figure (To'mey). The Reese muscle forceps applied over the eyelid exposes the area of incision (dotted lines) and holds the eyelashes out of the way.

I have discovered that the Reese muscle forceps is ideally suited for holding the eyelid during tarsorrhaphy. It provides a firm grip, even when applied close to the eyelid margin, because of its many fine teeth. It holds the eyelid as close as possible to its margin, preventing movement of the eyelid while it is being incised, and achieves perfect hemostasis. Moreover, the eyelashes are easily retracted as they are included in the grip of the forceps (Figure).

Summary

I have found the Reese muscle forceps to be the ideal instrument for holding and steadying the eyelid margin during tarsorrhaphy. Not only does it provide a firm grip, but it also prevents bleeding and retracts the eyelashes.

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This work was supported by grant No. 18-5213 from the Faculty of Medicine Research Committee, American University Medical Center.

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MEETINGS, CONFERENCES, SYMPOSIA

EDITED BY THOMAS CHALKLEY, M.D.

THE TWENTY-SECOND ANNUAL RESEARCH MEETING OF THE DEPARTMENT OF OPHTHALMOLOGY, UNIVERSITY OF TORONTO

The 22nd Annual Research Meeting of the Department of Ophthalmology, University of Toronto, Toronto, Ontario, Canada was held June 6, 1980, at the Medical Sciences Building, University of Toronto. The guest of honor was Dr. J. R. Trevithick, professor of biochemistry, University of Western Ontario; the opening speaker was Dr. P. Seeman, professor of pharmacology, University of Toronto. Dr. Trevithick presented two papers: (1) "Tissue Culture Modelling of Cortical Cataract Formation" and (2) "Cataract Modelling Experiments Indicate Some Future Possibilities for Clinical Treatment and Prevention of Cortical Cataracts." Dr. Seeman spoke on "Retinal Dopamine Receptors."

Abstracts of scientific papers presented at the meeting are as follows:

IN VITRO CORRELATE OF SUTURE REACTION

M. Avaria and P. K. Basu

A constant initial feature in tissue reaction caused by sutures is the mobilization of leukocytes around them. We wished to determine if there was a relationship between the degree of leukocyte attachment to a suture and the degree of suture reaction in vivo. Different kinds of 6-0 sutures were dipped in a human leukocyte suspension. Using light and scanning electron microscopy, we determined the numbers of cells attached to the sutures (per unit area) after a given period of incubation at 37°C. We found that the nylon sutures which produce the least tissue reaction in vivo had the lowest number of leukocytes attached to them. On the other hand, chromic collagen and braided silk sutures, which usually produce moderate to severe tissue reaction, had the greatest number of leukocytes attached to them.

THE EFFECTS OF DRYING OF DONOR CORNEAS

M. Avaria and P. K. Basu

The standard eye bank procedure is to keep donor eyes in a moist chamber at 4°C as soon as they are enucleated. Due to human error, some eyes reach the eye bank laboratory in dry bottles. In this study we wished to determine the effects of dryness on the viability and ultrastructure of the corneal cells. We used 12 rabbits. One eye of each rabbit was kept in a regular moist chamber as a control and the other experimental eye in a dry chamber at 4°C for 48 hours. Most of the experimental corneas, as compared to the controls, were thicker and had more epithelial and endothelial cell damage (as tested by vital staining and by transmission and scanning microscopy). Three experimental corneas looked normal, with minimum epithelial defects, but showed significant damage to the endothelial cells.

SIGNIFICANCE OF FAILURE OF INITIAL RADIOTHERAPY TO CONTROL INTRAOCULAR RETINOBLASTOMA

A. M. Drysdale and B. L. Gallie

Radiation therapy is the standard treatment for intraocular retinoblastoma when retaining visual acuity in the affected eye

is possible. We reviewed 31 patients treated with radiation for intraocular tumor between 1958 and 1977 to determine the incidence and significance of failure of initial radiation therapy to control intraocular tumor growth. In 15 patients (48%) no further therapy was required. In 16 patients (52%) intraocular tumor growth continued, requiring further treatment (removal of the eye, photocoagulation cryotherapy, or repeat radiation therapy). Radiation therapy failed to control all eyes with many large tumors (Reese Ellsworth groups III B, IV, and V). The failure rate for the smaller tumors (Reese Ellsworth groups I, II, and III) was 44%. Failure of initial radiation therapy resulted in significantly increased blindness and mortality as compared to those eyes controlled by initial radiation, even in the smaller tumors (Groups I, II, and IIIa). Failure of initial radiation therapy to control intraocular retinoblastoma is a bad prognostic sign. Efforts to improve radiation therapy for intraocular retinoblastoma are indicated.

Comparative Study of Dexamethasone and Indomethacin in the Suppression of Corneal Neovascularization

P. Harvey and P. M. H. Cherry

We wished to compare the effects of indomethacin and dexamethasone on the suppression of corneal vascularization after injury.

In our study, 7.0 silk suture was placed in the corneas of three groups of rabbits (N = 18) as a vasogenic stimulus. The first group received a 10.3 mg/ml suspension of indomethacin and the second group a 0.1% suspension of dexamethasone (Alcon) in the traumatized eyes three times daily. One group (control) received no medication. There was a statistically significant difference between the rate of neovascularization in the untreated group and the treated groups. Vascularization was suppressed in the drug-treated groups. There was, however, no measurable difference between the group treated with indomethacin and the group treated with dexamethasone in rate or quality of neovascularization.

Herpes Zoster Ophthalmicus Treatment Trial

M. J. Howcroft, J. Rodgers, and C. Breslin

We designed the protocol of an ongoing double-masked prospective clinical treatment trial designed to assess the value of high-dose systemic corticosteroids in the prevention of complications secondary to herpes zoster ophthalmicus. Patients are assessed at the time of diagnosis and at weekly intervals while receiving treatment. Ocular involvement, including post-herpetic neuralgia, is noted and graded at each visit. Follow-up of at least six months to one year is anticipated.

Referred patients are welcomed into the study provided that they (1) are more than 55 years old; (2) are referred within five days of the skin eruption; (3) can use high-dose corticosteroids; and (4) give informed consent.

Jones Pyrex Tubes and Assessment of 40 Patients

M. Howcroft and J. Hurwitz

The Lester Jones Pyrex glass tube remains the most effective method of lacrimal system bypass. When all methods of mucosal reconstruction of the lacrimal drainage pathways fail, the insertion of a permanent bypass tube becomes the only way of eliminating epiphora.

Of 40 patients reviewed, 80% are doing extremely well with a Jones tube in place.

There have been some complications such as extrusion, membrane formation, migration, turbinate hypertrophy, and intermittent tearing.

Effects of Zinc, Vitamin A, and Combined Deficiency on the Electroretinogram in the Rat

S. P. Kraft, J. A. Parker, Y. Matuk, and A. V. Rao

To assess the relative importance of zinc and vitamin A in photoreceptor function, a group of 36 Wistar rats were raised in dim illumination and deprived of both zinc (0-5 ppm) and vitamin A for 90 days (Phase I) until they lost weight and showed corneal changes of hypovitaminosis A. Scotopic electroretinograms (ERGs) were performed on nine rats on Day 83. Twelve rats that died at this stage had femurs analyzed for zinc content, livers assayed for vitamin A content, and eyes enucleated for histologic study.

For Phase II, the remaining 24 rats were randomly placed into three treatment groups: one remained vitamin A deficient (A−), one remained zinc deficient (Z−), and one received both zinc and vitamin A (Z+A+). ERGs were performed four and eight weeks after randomization. All animals were then killed so we could measure femur zinc, plasma zinc, and liver vitamin A levels and perform histologic studies of pancreas, gut, testes, and eye.

The ERGs of the Z−A− rats showed moderate deterioration compared to a normally fed group, implying a slow deterioration in rod function. Subsequently, the Z+A+ rats' ERGs markedly improved while the Z− rats showed almost equivalent improvement. The ERGs of the A− rats deteriorated and there were abnormalities in cornea, photoreceptor outer segments, and gut mucosa.

It appears that vitamin A can significantly reverse receptor degeneration from the Z−A− state even with a marginal zinc status, and zinc supplementation alone does not prevent and may accelerate deterioration from hypovitaminosis A.

Testing the Phagocytic Activity of the Retinal Pigment Epithelium

S. Markowitz, P. K. Basu, and N. S. Ranadive

The phagocytic activity of the retinal pigment epithelium was tested using the bovine eye cup model developed in our department. Latex particles and bovine outer rod segments were used as objects for phagocytosis. Various substances known to influence phagocytosis by polymorphonuclear leukocytes and macrophages were used as test agents. Iodoacetate and a mixture of penicillin with streptomycin reduced significantly the phagocytic activity of the retinal pigment epithelium. Concanavalin A enhanced the phagocytic activity. Lymphokines showed a tendency to increase phagocytosis slightly. Dimethyl sulfoxide had no effect on the phagocytic activity of the retinal pigment epithelium.

An Animal Model of Demyelinating Disease of the Optic Nerve

G. Martin and J. Czarnecki

Demyelinating lesions were produced in cat optic nerves. Each cat was anesthetized and 2.5 μl of lysolecithin, a demyelinating agent, was injected transclerally by a Hamilton syringe directly into the optic disk. The contralateral eye was injected by the same technique with normal saline to serve as a control. Cats were killed at one week, two weeks, four weeks, and eight weeks. The eyes were

examined histologically for demyelination. Demyelination was present in all specimens injected with the enzyme but significant disorganization of myelination was also present in control eyes. This was attributed to needle tract trauma. A modified technique using glass micropipettes and stereotact micromanipulator apparatus is now being investigated in an effort to lessen artifactual changes in myelination.

The Alternating Horner's Syndrome—Is It or Isn't It?

E. Moniz and J. Czarnecki

We studied two patents with so-called alternating Horner's syndrome. We performed pupillary testing on these patients, including cocaine and hydroxyamphetamine tests.

The accepted explanation was that these lesions were a true interruption of the sympathetic pathway, first on one side and then on the other. We offer an alternate explanation. There may be only a one-sided lesion which is at times hypoactive, thus producing ipsilateral Horner's syndrome. At other times, it may be irritative and hyperactive, simulating Horner's syndrome on the opposite side.

Toleration of Artificial Tears. The Effect of pH and Osmolarity

M. Motolko and C. Breslin

The effects of altering the pH and osmolarity of an artificial tear preparation on the tolerance of 15 patients with keratoconjunctivitis sicca was studied. Our results confirmed the value of both a neutral hypotonic tear substitute and an isotonic alkaline tear substitute in the management of patients with keratoconjunctivitis sicca. A commercially available alkaline tear substitute is needed.

Parameters of Normal Optokinetic Nystagmus in Man

D. J. Murphy, C. D. Sherret, and J. A. Sharpe

Optokinetic nystagmus was quantified in 15 normal subjects, aged 20 to 46, with infrared reflection oculography and a surrounding striped drum that enveloped the visual field. A multiparameter nystagmus algorithm was used for digital computer analysis of data. Parameters of optokinetic nystagmus gain (the ratio of mean optokinetic nystagmus slow phase velocity to drum velocity) and frequency were examined at stimulus speeds from 20 to 100 degrees per second.

Mean optokinetic nystagmus gains approached unity at stimulus velocities of less than 60 degrees per second. After 60 to 90 seconds of stimulation, optokinetic after-nystagmus was examined in complete darkness. Normal subjects often exhibited optokinetic after-nystagmus. The gain of optokinetic after-nystagmus was consistently lower (<0.25) than that previously obtained in the monkey. The occurrence of optokinetic after-nystagmus appeared to correlate with illusionary self-rotation (circularvection) during optokinetic nystagmus stimulation. In contrast to the slow build-up of optokinetic nystagmus reported in the monkey, human optokinetic nystagmus slow phases showed step increments in gain with changes in stimulus speed. This behavior may reflect foveal smooth pursuit superimposed upon the genuine optokinetic nystagmus elicited by panretinal stimulation.

Augmentation Keratoplasty—Possible Treatment for Aphakia

J.R. Oswald and W. S. Dixon

We set out to determine if silicone buttons could be inserted intracorneally

to increase the curvature of rabbit corneas and induce myopia. Fourteen rabbits were examined preoperatively for refractive error and keratometry readings. The rabbits were then anesthetized and underwent lamellar separation intracorneally, followed by insertion of trephined silicone buttons. Postoperative refraction and keratometry were carried out and repeated at one week. Results were similar in all rabbits, demonstrating an increase in curvature and induced myopia. The cornea overlying the silicone button, however, melted in all rabbits; 12 of the 14 melted within one week, while two lasted for two weeks. Gross infection was associated with corneal melting in six of the 14 rabbits. This paper outlines the technique devised for this procedure as we suspect that the corneal melting could be caused by excessive thickness of the silicone button. We plan to investigate whether this technique could be used for microlathed donor corneal buttons for intracorneal insertion.

Leukemic Infiltrations of the Optic Nerve

M. Ridley and J. R. Buncic

Abnormalities of the ocular system were detected in all patients treated with central nervous system leukemia at the Princess Margaret Hospital and the Hospital for Sick Children. Of these, more abnormalities involved the disk. Five patients had optic nerve infiltrates which were initially thought to be papilledema.

The clinical entity of optic nerve infiltration is discussed with regard to appearance, course, prognosis, and treatment.

We emphasized sensitivity to radiation therapy and agree with previous authors that the eye may be a pharmacologic sanctuary. We propose including the eye and orbit in prophylactic cranial radiation protocols.

P.K. Basu

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ALLVAR GULLSTRAND
A TRIBUTE ON THE 50TH ANNIVERSARY OF HIS DEATH

Allvar Gullstrand (1862-1930) was born in Landskrona, Sweden, and was the son of the town's physician. Gullstrand was a brilliant student in mathematics who, largely through his own efforts, mastered the discipline of differential geometry before entering the University of Lund. He followed his father's example and chose a career in medicine, but his interest in mathematics led him to enter the field of ophthalmology. He studied under Erik Johann Widmark of the Serafimer Hospital in Stockholm. Throughout his life, his primary interest remained the study of optics and his work expanded the earlier studies of William Rowan Hamilton (1805-1865).

Gullstrand was recognized, first in Sweden and then internationally, for his brilliant clarifications of refractive errors such as astigmatism, aberration, and coma. At the age of 38, he was appointed to the newly created chair of ophthalmology at the University of Uppsala, a position designed to permit the continuation of his scientific work. In October 1911, the faculty of the Caroline Medico-Surgical Institute in Stockholm awarded to Gullstrand the Nobel Prize for Medicine and Physiology for "work on the diffraction of light by lenses applied to the eye." As a result of this honor, Gullstrand was appointed in 1914 to a chair in physical and physiological optics at Uppsala which he filled until his retirement in 1927.

Gullstrand's brilliance in physiologic optics combined with his knowledge of the examination of the human eye led to the development of the slit-lamp biomicroscope. Others, including Koeppe, Gallemaerts, and Vogt, had attempted to design such an instrument before Gullstrand's success. Gullstrand's principal contribution was the design and construction of the light source: a filament enclosed in a light-proof covering and using

a system of lenses in a tiny aperture in the wall of the covering. This light source, which came to be known as the diaphragm lamp, provides a small, intensive beam of light and emits the light in only one direction out of a completely dark background. By combining this lamp with an aberration-free convex lens, Gullstrand constructed a small slit lamp for examining the eye's transparent structures, especially the cornea, aqueous humor, and lens. Professor Henker of the Zeiss works in Jena transformed Gullstrand's initial instrument into a stationary instrument with a binocular microscope, the basis of the modern slit-lamp biomicroscope.

Gullstrand's diaphragm lamp also permitted reflexless ophthalmoscopy. Gullstrand was able to use the central part of the pupil, where the eye's refractive errors cause the least distortion, to produce the image of the fundus while the light is projected through a peripheral part of the pupil. The diaphragm lamp and reflexless ophthalmoscope were the basis of the ophthalmoscope ophthalmologists use today.

During his long and productive career, Gullstrand made many other contributions. He was involved in a long and controversial study of the nature of the yellow color of the macula, a question which still needs further investigation. Gullstrand remained convinced that the macula was not yellow in the living state and that the leakage of pigment from the choroidal cells into the retina caused yellowing.

Gullstrand was a reserved and aloof individual whom few of his contemporaries knew well. He died on June 30, 1930, of a brain hemorrhage and is buried in the North Cemetery in Stockholm.

English-speaking scientists and physicians may not be familiar with Gullstrand's work because he wrote in Swedish. After Hemholtz's death in 1894, however, Gullstrand became one of the editors of the third edition of Hemholtz's "Handbuch der physiologischen Optik." He added five chapters of his own, and these contain a comprehensive review of his work. These appeared in English in James C. Southall's edition (Menasha, Wisconsin, Optical Society of America, 1924; reprinted, Dover Press, 1962). Although Granit, Hartline, and Wald shared the Nobel Prize in Physiology and Medicine for their work involving the retina, Gullstrand holds the distinction of being the only ophthalmologist ever to receive this award. Further, his development of the slit lamp provided one of the cornerstones of modern ophthalmology.

DANIEL M. ALBERT

CORRESPONDENCE

Letters to the Editor must be typed double-spaced on 8½ × 11-inch bond paper, with 1½-inch margins on all four sides, and limited in length to two manuscript pages.

Flying After Vitreous Injection

Editor:

Patients who have had air or sulfur hexafluoride injected into the vitreous cavity after vitrectomy surgery for traction retinal detachment are usually warned not to undertake air travel until the gas bubble has resorbed. The experience of one of my patients underscores the importance of this.

The patient, a commercial airline pilot who had become interested in ocular surgery after undergoing successful repair of a rhegmatogenous retinal detachment, was notified by a flight attendant that a passenger was complaining of severe ocular pain. He spoke to the passenger personally and found that she had had an aphakic detachment repaired several days previously and that she was flying

home from the referral center at which the operation had been done. The patient stated that air had been injected into the eye to help hold the retina in place. She had had no trouble until moments before when her eye suddenly became severely painful. The pilot deduced that depressurization had caused the gas bubble to expand and created the pain. The plane was at 7,000 feet cabin altitude when the pain occurred. The pilot increased cabin pressure to 5,500 feet, at which point the patient's pain disappeared.

Although vitrectomy surgeons have long recognized in theory the possibility that a gas bubble might expand and cause problems during air travel, this is the first case I am aware of where it has actually occurred. Certainly, all patients who undergo such a procedure should be warned not to fly until the gas bubble has resorbed.

JAMES R. BRINKLEY, JR., M.D.
Laguna Niguel, California

Lens Implant Follow-up

Editor:

We fully support the recommendations made by K. Hoffer in his letter "Food and Drug Administration implant folow-up" (Am. J. Ophthalmol. 89:877, 1980), concerning patients lost to follow-up in the clinical study of intraocular lenses. Dr. Hoffer referred to a tracing system maintained by the Intraocular Lens Manufacturers Association. In this paragraph, Dr. Hoffer refers to the clinical study as beginning on Feb. 9, 1977. This date is incorrect and should actually be Feb. 9, 1978.

We wish to commend clinical investigators for their diligent attempts to reduce the number of patients who are lost to follow-up.

ROBERT HUBBELL, President
Intraocular Lens Manufacturers Association,
Seal Beach, California

Scleral Buckling Without Thermal Adhesion

Editor:

I read with interest the article, "Scleral buckling without thermal adhesion" (Am. J. Ophthalmol. 89:662, 1980), by C. L. Fetkenhour and T. L. Hauch.

At a Retina Society meeting in 1967, I described a patient who had a retinal detachment treated with an encircling silicone band. The patient died within 48 hours of the operation, and we were fortunate enough to obtain the eye for microscopic sectioning. We were surprised to find an inflammatory reaction under the band in the area where no cryocoagulation lesions had been placed. The pathologic report read: "choroid: in the region under and near to the silicone band and implant, the choroid is congested, edematous, and heavily infiltrated, more so in the outer layers with inflammatory cells consisting predominantly of neutrophils. This inflammatory reaction is more marked near the horizonal meridians and is just as marked beneath the band nasally where there is no hole as it is temporally in the region of the hole, and extends posteriorly to midway between the equator and the posterior pole. Reaction beneath the hole superiorly is slightly less intense and has a larger lymphocytic component."

I believe that the presence of a band by itself creates an inflammatory reaction in the choroid, and this may account for the successful treatment of retinal detachments without cryocoagulation or heat.

The sections are available to anyone who wishes to study the effect of the scleral band on untreated choroid and sclera.

M. W. FRIEDMAN, M. D.
San Francisco, California

Reply

The pathologic findings reported in Dr. Friedman's letter suggested that a

silicone band might incite an underlying choroidal inflammation. In 1969, Curtin[1] described similar pathologic changes after retinal detachment surgery in a patient who died three days later. Curtin observed choroidal engorgement and dilated vessels overlying the buckle, and also on the opposite side of the eye. Furthermore, his extended review of microscopic slides of other cases in which silicone implant material had been used disclosed a remarkable lack of reaction around silicone implants. Curtin stated that if inflammatory reaction was present, it was around suture material and not the implants. These considerations suggest a possible initial diffuse, rather than localized, choroidal inflammatory response to cryotherapy, and one in which silicone has little influence. Also in Dr. Friedman's case, the possibility of a preexisting, that is, pretreatment, choroidal inflammation associated with the retinal detachment cannot be ruled out.

As far as the postoperative retinal adhesion is concerned, however, it is probably more important to recognize post-treatment changes in the retinal pigment epithelium and sensory retina than in the substance of the choroid itself, inasmuch as these layers, and not the choroid, are the ones primarily involved in reestablishing the retinal adhesion. The pathologic changes reported in Dr. Friedman's letter might be related to choroidal vascular changes after placement of an encircling silicone band.

Our own study of the effect of encircling silicone bands on ocular circulation demonstrated a marked decrease in both ocular pulse amplitude and ocular rigidity (unpublished data). Alterations of this sort could change the choroidal vascular supply and contribute to choroidal congestion and edema on the basis of a relative insufficiency of the choroidal circulation. Even so, this probably would not create the adhesion between the sensory retina and retinal pigment epithelium.

CARL L. FETKENHOUR, M.D.
THOMAS L. HAUCH, M.D.
Chicago, Illinois

REFERENCE

1. Curtin, V. T.: Pathological changes following retinal detachment surgery. In Symposium on Retina and Retinal Surgery. St. Louis, C. V. Mosby Co., 1969.

BOOK REVIEWS

Epidemiology and Statistics for the Ophthalmologist. By Alfred Sommer. New York, Oxford University Press, 1980. Softcover, 86 pages, 26 tables.

This brief, succinct text was written to provide the ophthalmologist with a simple, clear presentation of epidemiologic and statistical techniques relevant to conducting, interpreting, and assimilating the most common type of clinical research.

The first part deals with epidemiologic definitions, the use of controls, random sampling, bias, sample size, standardization, and reproducibility. The second portion of the text is devoted to the selection and use of statistical manipulations and testing the statistical significance of results. Dr. Sommer provides a number of examples of good and bad uses of statistical analysis from published reports.

He emphasizes sampling bias, which leads to such conclusions as cataracts are more common in patients with diabetes, whereas it is cataract extraction that is more common in diabetics rather than the incidence of cataracts.

Since severe degrees of astigmatism or irregular astigmatism are treated with hard, rather than soft, contact lenses, it is more likely that these patients will devel-

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op keratoconus than those using soft contact lenses. Thus the two groups are not comparable.

Dr. Sommer emphasizes the value of prospective studies with adequate randomization. Thus, in one sampling scheme patients admitted on Monday, Wednesday, and Friday received one form of therapy; those admitted on Tuesday, Thursday, and Saturday received another. Referring physicians quickly learned which therapy was given on which days and arranged for their patients to arrive the day the therapy the physician preferred was being given.

With Dr. Sommer's book in hand one can certainly interpret medical reading with far greater sensitivity. It is highly recommended for all who see patients and read about them in medical journals.

FRANK W. NEWELL

Ocular Pathology. By C. H. Greer. Oxford, Blackwell Scientific Publications, 1979. Hardcover, 281 pages, preface, index, 90 black and white figures. $35.75

This textbook is felicitously written and admirably succeeds in its objective of encompassing the basics of ophthalmic pathology within a manageable length. Up-to-date concepts of inflammation, the role of the neural crest, and the latest World Health Organization classification of eye tumors provide a firm gridwork for an authoritative discussion of these topics. The book is designed for those preparing for examinations, residents in training, and fully trained ophthalmologists who may want to review the subject of opthalmic pathology. It would be particularly useful for residents who are doing a rotation in ophthalmic pathology, because its length allows reading and rereading over a four- to six-week period. While there are large treatises on ophthalmic pathology for those who want in-depth treatments of selected topics, the present book fills the void for the casual reader who wants an overview of the field. I heartily recommend this book to those who would like to whet their appetites on ophthalmic pathology.

FREDERICK A. JAKOBIEC

Repair and Reconstruction in the Orbital Region, 2nd ed. By John Clark Mustardé. New York, Churchill Livingstone, 1979. Hardcover, 400 pages, index, over 200 black and white figures. $85

This is the second edition of a book first published in 1966. It follows the same format as the first edition with the addition of chapters on hypertelorism and coloboma. The first five chapters deal with reconstruction of partial-thickness defects of the eyelids and conjunctiva. The next six chapters deal with full-thickness eyelid loss and surgery at the medial and lateral canthus. The basic principles of skin surgery are presented in a clear and understandable manner, especially those relating to grafts and flaps. Each case is well illustrated so that the reader can understand why the author chose a particular method, how to perform the surgery, and the final result. Photographs and illustrations are used in combination to clarify the techniques. The author emphasizes his own techniques for the repair of small defects all the way up to reconstruction after loss of both eyelids.

Unfortunately, the chapter on lacrimal disease has not been updated to include the newer techniques of canicular reconstruction such as the Quickert-Dryden method of silicone intubation. The chapter on socket reconstruction emphasizes older techniques requiring external pressure on the socket and does not include the technique of fixation to the orbital rim such as presented by Callahan. Although most surgeons no longer recommend the Morel-Fatio spring for paralytic

ectropion, the chapter on ectropion describes it without mentioning that erosion into the eyeball is possible. The author's interesting techniques of split-level eyelid resection and levator palpebrae superioris muscle hitch are well presented in the chapter on blepharoptosis. Methods of repairing congenital epicanthus, telecanthus, and blepharophimosis are presented in a clear and understandable manner. He also describes the newer methods of repairing hypertelorism by repositioning the orbital-nasal skeleton. In the chapter on coloboma, severe orbital anomalies associated with coloboma of the eyelid are discussed. The last chapter is an excellent summary of the fine points of tissue transfer using skin grafts, mucous membrane and cartilage grafts, and forehead flaps.

Any surgeon dealing with eyelid tumors by frozen-section techniques must be prepared to reconstruct a defect larger than the external appearance of the tumor would indicate. The author gives a step-by-step discussion of the surgery by which one can reconstruct a defect of any size in the orbital region. Although this book describes the techniques used by Dr. Mustardé, there are other appropriate procedures which can be used for eyelid reconstruction. Even if one prefers other techniques, an understanding of this book would be useful because these techniques can be combined with other procedures.

FREDERICK MAUSOLF

Intervention Combinée Glaucome Cataracte. By A.-G. Ourgaud and P. Gastaud. Marseille, Diffusion Générale de Librairie, 1980. Paperbound, 151 pages, 44 black and white figures. $34.45

The introductory section of this monograph contains an excellent description of the macroscopic and microscopic appearance of the chamber angle. Next, the authors review the problem of the combined presence of cataracts and glaucoma. They make the obvious statement that if the glaucoma is mild and the cataract severe, the latter should be removed; if the situation is reversed, then the glaucoma should be treated. However, the bulk of their publication deals with those cases that require both a filtering operation and cataract extraction and they recommend that this be treated by a combined trabeculectomy and cataract extraction. They recognize that there are various techniques for performing this but give their own in great detail.

Essentially it consists of a scleral flap under a large conjunctival flap, a trabeculectomy done with scissors, and then extension of the wound nasally and temporally to allow for extraction of the cataract. The wound is then closed with interrupted 7-0 or 8-0 Vicryl sutures and the conjunctiva with either a running suture or interrupted sutures. The final section lists their results and complications.

Perhaps the most valuable portion of this monograph is the complete bibliography on surgical intervention in the presence of glaucoma and cataracts.

DAVID SHOCH

Pediatric Ophthalmology Practice. By Eugene M. Helveston and Forrest D. Ellis. St. Louis, C. V. Mosby Co., 1979. Hardcover, 303 pages, index, bibliography, 422 black and white figures. $43.50.

This book gives a practical account of the more common topics encountered in pediatric ophthalmology, in a concise, easy-to-read, atlas-like style. The authors, who are associates, do not intend to provide an exhaustive exposition on

these topics, but rather an informative, updated review of the subjects based on their experience and expertise.

Beginning with chapters on visual acuity testing, strabismus, and amblyopia, the authors go on to give their current ideas about diagnosis and treatment in chapters on the nasolacrimal system, glaucoma, blepharoptosis, the orbit, and trauma. A chapter on the retina includes an excellent summary of the history and pathophysiology of retrolental fibroplasia. This chapter also includes a succinct, thorough discussion of retinoblastoma and an excellent table summarizing many retinitis pigmentosa-like disorders. In the chapter on infections, inflammations, and developmental corneal abnormalities, the great quantity of material covered necessitates less specificity in discussion; the chapter includes an encyclopedic summary of the developmental corneal abnormalities and dystrophies. There are short chapters on genetics and anesthesia. In the last of the 14 chapters the authors give an excellent short review of dyslexia, an often perplexing problem for the ophthalmologist.

The book is well illustrated with good correlation between the text and the figures. The type is large and clear. In a few instances the use of black and white instead of color photographs makes the abnormality difficult to see. Typographical and editorial errors are few, and undoubtedly will be corrected in subsequent editions. The bibliography at the end of the book is small and selective but serves as a good basis for additional reading.

Inevitably a few statements in the book will give rise to some disagreement or question by other ophthalmologists. For example, it is not clear to me why the authors use Neo-Synephrine HCl along with Cyclogyl for cycloplegic refractions. Neo-Synephrine HCl is a mydriatic and not a cycloplegic agent and Cyclogyl produces both cycloplegia and excellent mydriasis. Also, refraction 30 to 40 minutes after the instillation of Cyclogyl may be too soon for full cycloplegia. Studies have shown that full cycloplegia in some patients is not reached for 90 minutes, but 60 minutes is a practical interval for most patients. In the treatment of infections associated with nasolacrimal duct obstruction in infants, I avoid the use of sulfacetamide drops (recommended by the authors) because the antibacterial action of sulfonamides is inhibited by purulent material. Ketamine anesthesia for intraocular pressure measurements in children is not used by many ophthalmologists because ketamine may temporarily increase the intraocular pressure. For the most part, however, I agree with the authors' sensible, middle-of-the-road approach to the subjects covered.

The pediatrician will find this book a useful reference source; the pediatric ophthalmologist will enjoy reading it "to compare notes." The book will prove most useful, however, to the ophthalmology resident, the pediatric ophthalmology fellow, and the general ophthalmologist who will find it a concise, current overview of pediatric ophthalmology.

LEONARD APT

Ophthalmology. A Short Textbook, 9th ed. By Fritz Hollwich. Translated by Gerhard Cibis. Chicago, Year Book Medical Publishers, 1980. Paperbound, 336 pages, index, 23 color plates, over 200 black and white photographs. $18.50

This small pocket book was originally written in German by Professor Fritz Hollwich, former Director of the University Eye Clinic in Munster, Germany. Dr. Hollwich was one of the leading German ophthalmologists of his day, and had the delightful knack of conveying in a

few sentences the important clinical and histopathologic features of the diseases which he discusses. His ability to convey these essentials concisely and clearly benefits beginning students of ophthalmology as well as the more advanced or postgraduate students who wish to refresh their knowledge quickly. The photographs, diagrams, and arrangement of the text superbly convey the pertinent information. The author cleverly included a differential diagnosis of each condition as well as a discussion of the histopathologic features and clinical findings and complications and therapy. This arrangement certainly makes the book tremendously valuable as a handbook, particularly for young residents and medical students. The author has covered concisely all aspects of ophthalmology with the 336 pages of text, and the small size of the book makes it ideal for slipping into the pocket of the busy medical student or resident in the clinic. It is difficult to cover such a large field without being sketchy, but the author has avoided this common pitfall. The color plates are superbly arranged and described, and this book will become classic for students. The publishers are to be congratulated for translating this book into English, particularly in regard to publishing a complete revision of the text of the previous edition. The book has been updated to include the use of fluorescein angiography and has new chapters on the treatment of fungal disease of the cornea, the correction of unilateral aphakia with contact lenses and intraocular lenses, and newer surgical procedures. It is highly recommended for medical students and residents.

H. DWIGHT CAVANAUGH

SYMPOSIA

The Blood-Retinal Barriers, NATO Advanced Study Institutes Series, Series A: Life Sciences, Vol. 32. Edited by Jose G. Cunha-Vaz. New York, Plenum Publishing Corp., 1980. Hardcover, 319 pages, index, 144 black and white figures. $39.50

P. CARVALHO: **Structure and function of cell membranes**

J. J. P. DE LIMA: **Biophysical aspects of membrane permeability**

K. DOROVINI-ZIS, J. J. ANDERS, AND M. W. BRIGHTMAN: **Cerebral endothelium, blood-brain barrier and the astrocyte membrane**

S. I. RAPOPORT: **Cerebrovascular permeability in the normal brain and following osmotic opening**

J. CUNHA-VAZ: **Sites and functions of the blood-retinal barriers**

A. LAJTHA AND H. SERSHEN: **Alterations of amino acid transport in the central nervous system**

L. BITO AND C. J. DEROUSSEAU: **Transport functions of the blood-retinal barrier system and the microenvironment of the retina**

D. M. MAURICE: **Drug exchanges between blood and virtreous**

A. BILL: **Drainage of intraocular fluids**

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A. LATIES, P. A. GRIMES, AND S. I. RAPOPORT: **Acute hypertension and experimental diabetes. Evaluation and their effects on blood-ocular barriers**

D. FINKELSTEIN AND A. PATZ: **Experimental retinal angiogenesis**

F. GOLDBERG: **Diseases affecting the inner blood-retinal barrier**

A. F. DEUTMAN: **Significance of alteration of the outer blood-retinal barrier**

ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

Acta Anaesthesiologica Scandinavica

EPIDURAL ANALGESIA AND PERINATAL RETINAL HAEMORRHAGES. Maltau, J. M., and Egge, K. (Depts. Obstet. and Gynecol., and Ophthalmol., Rikshospitalet, Univ. Oslo, Norway). Acta Anaesthesiol. Scand. 24:99, 1980.

The incidence of magnitude of retinal hemorrhages in a group of neonates delivered spontaneously (n=50), whose mothers received epidural analgesia during labor, has been compared to those of a spontaneously delivered control group (n=50) who received conventional analgesia. The second stage of labor was significantly longer in the epidural group ($P<0.001$), which contained a higher number of primiparas than did their controls. Still, a lower incidence of retinal hemorrhages was noted in the epidural group ($P<0.01$). The study indicates that epidural analgesia offers a certain degree of protection against perinatal retinal hemorrhages in the spontaneously delivered neonate. (2 tables, 10 references)—Authors' abstract

Archives of Ophthalmology

FUNGAL ENDOPHTHALMITIS FOLLOWING INTRAOCULAR LENS IMPLANTATION. A SURGICAL EPIDEMIC. Pettit, T. H., Olson, R. J., Foos, R. Y., and Martin, W. J. (Jules Stein Eye Inst., Univ. California, Los Angeles, Calif.). Arch. Ophthalmol. 98:1025, 1980.

Thirteen cases of fungal endophthalmitis were caused by *Paecilomyces lilacinus*. In all cases an intraocular lens was inserted that had been sterilized in sodium hydroxide and neutralized in sodium bicarbonate. In 12 of the 13 cases, it was absolutely determined that a manufacturer's lot 128 was the neutralizing solution used. The same fungus that caused the endophthalmitis was cultured from several of the neutralizing solutions from lot 128. Eight of the 13 eyes eventually required enucleation. Of the remaining five eyes, one eventually recovered 6/8 (20/25) vision, one recovered 6/24 (20/80) vision, one had light perception, and two lost light perception (one of these became phthisical). This surgically induced epidemic of fungal endophthalmitis clearly shows the major consequences of a breakdown in quality control for any substance or material used intraocularly. (15 figures, 7 tables, 48 references)—Authors' abstract

NONGLAUCOMATOUS EXCAVATION OF THE OPTIC DISC. Trobe, J. D., Glaser, J. S., Cassady, J., Herschler, J., Anderson, D. R. (Dept. Ophthalmol., Univ. of Florida, Gainesville, Fla.). Arch. Ophthalmol. 98:1046, 1980.

In order to test the hypothesis that glaucomatous and nonglaucomatous optic disk cupping can be distinguished ophthalmoscopically, three ophthalmologists experienced in assessing optic disks were asked to view fundus stereophotographs as "unknowns". Of 29 eyes with nonglaucomatous optic atrophy, 13 (44%) were misdiagnosed as showing glaucoma by at least one observer. Of four optic disk features specifically analyzed, neuroretinal rim pallor proved to be 94% specific for nonglaucomatous atrophy, while focal or diffuse obliteration of the neuroretinal rim was 87% specific for glaucoma. These two signs are the most useful in making the distinction. Thinning of the rim was more common in glaucoma than in nonglaucomatous atrophy, but was only 47% specific for glaucoma. Laminar dots were present in both types of excavation. (4 figures, 2 tables, 17 references)—Authors' abstract

VITREOUS LOSS MANAGED BY ANTERIOR VITRECTOMY. LONG-TERM FOLLOW-UP OF 59 CASES. Berger, B. B., Zweig, K. O., Peyman, G. A. (Dept. Ophthalmol., Univ. Illinois, Chicago, Il.). Arch. Ophthalmol. 98:1245, 1980.

Fifty-nine cases of vitreous loss managed by anterior vitrectomy were followed up for an

average of 26 months. Thirty-seven cases were managed by automated anterior vitrectomy and 22 by cellulose sponge anterior vitrectomy. Both techniques, when performed by resident ophthalmologists, give equivalent results. Anterior vitrectomy for vitreous loss gives significantly better results than nontreatment of vitreous loss but significantly poorer results than uncomplicated intracapsular cataract extraction. This is largely caused by the increased incidence of chronic cystoid macular edema after vitreous loss. Vitreous loss treated by anterior vitrectomy is associated with 10% to 15 % greater endothelial cell loss than uncomplicated intracapsular cataract extraction. (5 tables, 15 references)—Authors' abstract

Australian Journal of Ophthalmology

DRUSEN AND THEIR RELATIONSHIP TO SENILE MACULAR DEGENERATION. Sarks, S. H. (14 Norton St., Leichardt, Australia). Aust. J. Ophthalmol. 8:117, 1980.

This beautifully illustrated paper describes the progress of retinal drusen from hard (hyaline), to semisolid, to soft, to regressing. The hyaline material changes to pale staining amorphous material shown by electromicroscopy as vesicles. Membranous debris appeared beneath the pigment epithelium in normal aged eyes, and its accumulation led to widespread shallow separation of the basement membrane. Softening of the drusen was most evident with subretinal neovascularization. Pigment epithelium proliferates, then degenerates, and calcium accumulates in the drusen. Small areas of geographic retinal atrophy gradually expand and coalesce. (23 figures, 9 references)—Ronald Lowe

British Journal of Ophthalmology

INFANTILE CYSTOID MACULOPATHY. Trese, M. T., and Foos, R. Y. (Depts. Ophthalmol. and Pathol., Jules Stein Eye Inst., Univ. California, Los Angeles, Calif.). Br. J. Ophthalmol. 64:206, 1980.

Three premature infants with no family history of eye disease showed bilateral cystoid maculopathy on post-mortem examination. These macular lesions are indistinguishable macroscopically from the macular lesions of sex-linked juvenile retinoschisis. Cystoid change was observed at various vertical retinal levels. The oldest child had a schisis cavity within the nerve fiber layer. All cases had reduced numbers of retinal ganglion cells and central nervous system abnormalities. The authors believe these are the first reported cases of a cystoid macular lesion present at birth and the first description of the pathological features of a cystoid macular lesion in infants. (5 figures, 12 references)—Authors' abstract

MACULAR CHANGES RESULTING FROM PAPILLOEDEMA. Morris, A. T., and Sanders, M. D. (Dept. Neuro-Ophthalmology, National Hospital for Nervous Diseases, Queen Square, London). Br. J. Ophthalmol. 64:211, 1980.

Six cases are presented with macular changes in association with papilloedema; four suffered permanent visual loss. The macular changes consisted of hemorrhages situated in front, within, or behind the retina, and occasionally the results of neovascular membrane formation produced secondary visual loss. Changes in the pigment epithelium were seen in three cases associated with choroidal folds. Macular stars rarely produce visual loss. (5 figures, 14 references)—Authors' abstract

British Medical Journal

PRIMARY HYPERPARATHYROIDISM PRESENTING WITH VISUAL FAILURE AND PALATAL SWELLING. Carr, D., Hall-Parker, B. J. P., Summersgill, B., and Thomson, R. W. (North Tees General Hosp., Cleveland, England), Br. Med. J. 280:1505, 1980.

A 47-year-old woman had a mass in her palate of ten weeks' duration, bilateral calcification of her corneas, and loss of vision in the right eye. The right optic foramen showed a region of radiolucency with no clear margin. Biopsy of the palatal mass disclosed a giant-cell lesion rather typical of the lesions described with primary hyperparathyroidism.

Serum calcium and parathyroid hormone levels were also increased and surgical investigation disclosed a tumor of the left inferior parathyroid gland. This was removed and within two weeks vision in the right eye showed improvement and subsequent tomography showed that the optic foramen became sharply defined and normal in size. After five months visual acuity was 6/5 (20/16). It is assumed that there was probably a giant-cell tumor present in the optic foramen that compressed the blood supply to the optic nerve. (3 references)—David Shoch

Cancer

CARCINOMA METASTATIC TO THE ANTERIOR OCULAR SEGMENT. RESPONSE TO CHEMOTHERAPY. Sierocki, J. S., Charles, N. C., Schafrank, M., and Wittes, R. E. (Dept. Med., Memorial Sloan-Kettering Cancer Ctr., New York, N.Y.). Cancer 45:2521, 1980.

Generally, metastatic lesions involve the posterior segment of the eye and, less commonly, the anterior segment. The patient reported here had a small carcinoma of the lung. About one year after the diagnosis was made and after treatment with radiation and various chemotherapeutic agents, the patient developed a nodule in the iris of the right eye. He was started on a new agent called Vindesine and this resulted in complete resolution of the lesion. Vindesine is a synthetic vinca alkaloid derived from vinblastine sulfate and has a mechanism of action similar to other vinca alkaloids. (3 figures, 12 references)—David Shoch

RETINITIS PIGMENTOSA AND COLON CANCER. Winzelberg, G. G., Greenstein, R., and Ferrucci, J. T. (Dept. Radiol., Massachusetts General Hosp., Boston, Mass.). Cancer 45:2897, 1980.

Retinitis pigmentosa has been associated with numerous diseases. The authors report the first association between it and colon cancer. This occurred in three of 53 hospitalized patients with retinitis pigmentosa for an incidence of 5.7%. The incidence of colon cancer in the general population is 0.047%. This would seem to be a statistical difference. Of particular interest is the fact that in two of the patients the cancers occurred at the age of 24 and in a third at the age of 37. (9 references)—David Shoch

Experimental Eye Research

ROD AND CONE DISC SHEDDING IN LIGHT-ENTRAINED TREE SQUIRRELS. Tabor, G. A., Fisher, S. K., and Anderson, D. H. (Dept. Biol. Sci., Univ. California, Santa Barbara, Calif.). Exp. Eye Res. 30:545, 1980.

Adult tree squirrels (*Sciurus carolinensis*) entrained to a daily lighting cycle of 12L:12D, display a biphasic rhythm of disk shedding. One peak occurs one to two hours after light onset. The second peak occurs during the middle of the dark period, about three-quarters of a cycle out of phase with the morning peak. Rod disk shedding predominates in the morning, and cone shedding predominates during the dark, but both cell types shed disks throughout the cycle. These findings indicate that rod shedding in this species is temporally linked to light onset in a way consistent with earlier reports on a variety of other species. On the other hand, cone shedding is not temporally related to the onset of darkness in the same way that rod shedding is related to the onset of light. (21 figures, 12 references)—Authors' abstract

Laryngoscope

CENTRAL AUDITORY TESTING AND DYSLEXIA. Welsh, L. W., Welsh, J. J., and Healy, M. P. (Jenkintown, Pa.). Laryngoscope 90:972, 1980.

A group of dyslexic pupils with normal ocular function was studied to determine whether a hearing disability existed. The patients had the characteristic findings of dyslexia, that is, reduced reading, spelling, and writing abilities combined with normal intellectual capacity. Four tests were given to this group: the competing sentence test, binaural fusion, rapidly alternating speech perception, and filtered speech. Fifty percent of the group failed two of the four tests and each of the 77 children in the group failed at least one component. The most sensitive tests appeared to be binaural fusion and filtered

speech. The authors suggest that these findings may indicate a lesion in the temporoparietal cortex and the association fibers. (8 figures, 24 references)—David Shoch

Nature

IS ACTIN IN EYE LENS A POSSIBLE FACTOR IN VISUAL ACCOMMODATION? Kibbelaar, M. A., Ramaekers, F. C. S., Ringens, P. J., Selten-Versteegen, A. M. E., Poels, L. G., Jap, P. H. K., van Rossum, A. L., Feltkamp, T. E. W., and Bloemendal, H. (Depts. Biochem., Cytology and Histol., Univ., Nijmegen, Nijmegen, Netherlands). Nature 285:506, 1980.

Actin is a protein, found in various nonmuscle cells, that is able to polymerize into filaments. It has recently been found in the lens. Since actin has a structural function, it has been suggested that it might have a role in accommodation. It may be present as a skeletal element enabling the lens to restore its shape on accommodation, or it could be actively involved in the process of accommodation, rendering the lens a primitive contractile organ. This study indicates that in species in which there is weak accommodation, such as the calf and the rat, there is a great deal of structured actin. In species in which there is a great deal of accommodation, such as the pigeon, structured actin appears to be scarce in the lens. Therefore it appears that structured actin is involved as a cytoskeletal structure but not in the active process of accommodation. (5 figures, 19 references)—David Shoch

Ophthalmology

EFFICACY OF ALRESTATIN, AN ALDOSE REDUCTASE INHIBITOR, IN HUMAN DIABETIC AND NONDIABETIC LENSES. Chylack, L. T., Henriques, H. F., Cheng, H. M., and Tung, W. H. (Howe Lab. of Ophthalmol., Harvard Med. School, Massachusettes Eye & Ear Infirmary, Boston, Mass.). Ophthalmol. 86:1579, 1979.

Immediately after cataract extraction, lenses from diabetic and nondiabetic patients were collected, classified, and assayed or incubated in high-glucose medium. The distribution of cataract types within the diabetic and nondiabetic groups was almost identical. The aldose reductase inhibitor AY22,284 (Alrestatin) was as effective in blocking sorbitol formation in diabetic as in nondiabetic lenses. While there was no difference in the level of intralenticular glucose, the diabetic lens produced significantly more sorbitol than did the nondiabetic lens. Also, the activity of polyol dehydrogenase was much lower in the diabetic population. While these results are preliminary, they suggest that diabetes, in some way, may confer on the human lens an increased susceptibility to osmotic stress via the sorbitol pathway. (2 figures, 17 references)—Authors' abstract

USE OF SILICONE LENSES IN INFANTS AND CHILDREN. Gurland, J. E. (New York, N.Y.) Ophthalmol. 86:1599, 1979.

A contact lens made from silicone rubber has been fitted successfully for extended wear in children. The lens has been used primarily with aphakic children, but it has also been tolerated by children with anisometropia, high myopia, and keratoconus. The lens can be handled like a hard contact lens and can be fitted by a variety of methods. The eye shows excellent tolerance of the lens and complications or failures to wear the lens have been due primarily to discomfort. After about a year of wear the lens surface can no longer be cleaned. (4 tables, 4 references)—Author's abstract

ULTRASONICALLY GUIDED NEEDLE BIOPSY AND CYTOLOGIC DIAGNOSIS OF SOLID INTRAOCULAR TUMORS. Jakobiec, F. A., Coleman, D. J., Chattock, A., and Smith, M. (Algernon B. Reese Lab. of Ophthalmic Pathology and Ultrasound Lab., Edward S. Harkness Eye Inst., New York, N.Y.). Ophthalmol. 86: 1662, 1979.

Six solid intraocular tumors were reliably diagnosed by needle biopsies and cytologic examination of the aspirates. All of the tumors had an unusual clinical or diagnostic feature that raised the possibility of a nonmelanomatous tumor. Two intraocular lesions

that were obscured by opaque media or a retinal detachment were successfully biopsied under B-scan ultrasonographic guidance of the needle. Cytodiagnosis of narrow spindle B, plump spindle B, and epithelioid cell types, as well as one case of Coats' disease, was possible and correlated closely with the predominant cell types comprising the lesions discovered on histopathologic examination of the globes that were enucleated. The ocular tissues were not significantly disturbed and hemorrhage was not a serious problem. Considerable profit may attend the use of this technique for the diagnosis of possible metastatic lesions and tumors of the ciliary body. The technique, however, should not be routinely employed and should be restricted to extremely difficult diagnostic problems. (10 figures, 40 references)—Authors' abstract

FOLLOW-UP STUDY OF PRESUMED OCULAR HISTOPLASMOSIS SYNDROME. Lewis, M., van Newkirk, M. R., and Gass, J. D. M. (Bascom Palmer Eye Inst., Univ. of Miami, Miami, Fla.). Ophthalmol. 87:390, 1980.

One hundred and sixty-two patients with presumed ocular histoplasmosis syndrome were evaluated. New macular scars not present on initial angiogram developed in 9% of eyes. The risk of developing a symptomatic macular lesion in eyes with asymptomatic macular scars was 23%. In eyes with peripapillary scarring, 3.8% developed a peripapillary neovascular membrane. Follow-up was 5.5 years. When the neovascular membrane was touching or outside the foveal avascular zone, the difference between photocoagulated and control eyes was not statistically significant. When only adequately photocoagulated eyes (20) were compared with control eyes (40), the difference was statistically significant ($P = 0.04$). (2 figures, 7 tables, 33 references)—Authors' abstract

AN EVALUATION OF CURRENTLY AVAILABLE, NONINVASIVE TESTS OF CAROTID ARTERY DISEASE. Sanborn, G. E., Miller, N. R., Langham, M. E., and Kumar, A. J. (Neuro-Ophthalmology Unit, Wilmer Inst., Johns Hopkins Hosp., Baltimore, Md.). Ophthalmol. 87:435, 1980.

A clinical study compared compression ophthalmodynamometry, suction ophthalmodynamometry, oculoplethysmography, modified oculopneumoplethysmography, and Doppler flow studies with graded arteriography for the detection of carotid artery stenosis. Compression ophthalmodynamometry and suction ophthalmodynamometry were the most accurate tests in this series with the results of both types of ophthalmodynamometry almost identical. A new, noninvasive procedure, oculocerebrovasculometry, was also performed in a slightly different group of patients. This procedure has theoretic advantages over the others tested because it measures directly the intraocular pressure as the end point of the test. (7 figures, 3 tables, 17 references)—Authors' abstract

ADVERSE REACTIONS FROM TIMOLOL ADMINISTRATION. van Buskirk, E. M. (Dept. Ophthalmol., Univ. of Oregon, Portland, Ore.). Ophthalmol. 87:447, 1980.

Five hundred forty-seven reports of adverse reactions to timolol maleate received by the National Registry for Drug-Induced Ocular Side Effects during an 11-month period were reviewed. Approximately half the reports concerned systemic reactions affecting the cardiac, pulmonary, central nervous, gastrointestinal, and dermatologic systems. Most serious were depression, anxiety, and confusion; bradycardia and arrhythmias; and pulmonary airway obstruction. Half of the ocular side effects concerned external ocular disease, of which superficial punctate keratitis and corneal anesthesia were the most remarkable. Ten percent of the reports described a visual disturbance which, in many cases, was not associated with refractive error. (1 table, 16 references)—Author's abstract

Pharmacology

GENETIC DIFFERENCES IN CATARACT AND OTHER OCULAR ABNORMALITIES INDUCED BY PARACETAMOL AND NAPHTHALENE. Shichi, H., Tanaka, M., Jensen, N. M., and Nebert, D. W. (Lab. Vision Research, National Eye Inst., Bethesda, Md.). Pharmacology 20:229, 1980.

Two different strains of mice were given daily oral doses of acetaminophen or naphthalene in doses of 60 to 120 mg/kg/day and were concomitantly treated with 3-methylcholanthrene or β-naphthoflavone. In one of the strains—C57BL/6N—cataracts developed and persisted until the animals were killed for examination six to seven months later. Histologic signs of chronic toxicity were seen in the anterior lens cortex, choroid, ciliary body, and iris. In the second strain—DBA/2N—treated in exactly the same fashion, no cataracts developed. These data illustrate the delicate balance between genetic and environmental factors causing ocular toxicity. Because drug-induced cataracts look clinically very similar to senile cataracts, these results may be important to patients receiving either a single large overdose of acetaminophen or high doses over a long period of time. (4 figures, 2 tables, 40 references)—David Schoch

NEWS ITEMS

EDITED BY THOMAS CHALKLEY, M.D.

Send News Items double-spaced to News Editor, American Journal of Ophthalmology, Tribune Tower, Suite 1415, 435 N. Michigan Avenue, Chicago, Illinois 60611

For adequate publicity, notices of postgraduate courses, meetings, and lectures must be received at least four months before the date of occurrence.

INTERNATIONAL AGENCY FOR THE PREVENTION OF BLINDNESS: LASER AND ADVANCED RETINAL MEETING

A Laser and Advanced Retinal Meeting will be held in Penang, Malaysia, March 1-5, 1981. For further information, write Michael S. H. Oh, M.D., c/o Penang Medical Centre, No 1 Jalan Pangkor, Penang, Malaysia.

XII JULES STEIN LECTURE AND POSTGRADUATE SEMINAR

The XII Jules Stein Lecture and Postgraduate Seminar will be presented April 23 and 24, 1981, in Los Angeles, California. The lecture will be delivered by Saiichi Mishima. For further information, write Mrs. Lu Hendricks, Jules Stein Eye Institute, UCLA School of Medicine, Los Angeles, CA 90024.

LONG ISLAND JEWISH-HILLSIDE MEDICAL CENTER: CLINICAL DAY ON CATARACT SURGERY AND CORRECTION OF APHAKIA

The Department of Ophthalmology, Long Island Jewish-Hillside Medical Center and the Health Sciences Center, State University of New York, will sponsor a clinical day of cataract surgery and correction of aphakia, May 13, 1981. For further information, write Continuing Education Coordinator, Long Island Jewish-Hillside Medical Center, New Hyde Park, NY 11042.

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO: COURSE ON CURRENT THINKING IN THE MANAGEMENT OF OCULAR AND ADNEXAL TUMORS

A postgraduate course, "Current Thinking in the Management of Ocular and Adnexal Tumors," will be held in San Francisco, Feb. 4-6, 1981. For further information, write Extended Programs in Medical Education, School of Medicine, Rm. 569-U, University of California, San Francisco, CA 94143.

UNIVERSITY OF TORONTO: ANNUAL REFRESHER DAY COURSE

The Department of Ophthalmology of the University of Toronto will hold its annual refresher day course Feb. 6 and 7, 1981. For further information, write Department of Ophthalmology, University of Toronto, 1 Spadina Crescent, Room 115, Toronto, Ontario, Canada M5S 2J5.

NEW YORK OCULAR TUMOR MEETING

The New York Ocular Tumor meeting will be held Feb. 21, 1981. For further information, write Frederick A. Jakobiec, M.D., Manhattan Eye, Ear and Throat Hospital, 210 E. 64th St., New York, NY 10021.

EYE FOUNDATION OF AMERICA: SYMPOSIUM ON THE ANTERIOR SEGMENT

The Eye Foundation of America will present a symposium on the anterior segment, Feb. 20 and 21, 1981, in New Orleans. For further information, write Kenneth G. Haik, M.D., Symposium Coordinator, The Eye Foundation of America, 823 Maison Blanche Bldg., New Orleans, LA 70112.

CORNELL MEDICAL COLLEGE: MANAGEMENT OF RETINAL DISEASE—ART OR SCIENCE?

The Cornell University Medical College will hold a course, "Management of Retinal Disease—Art or Science?," Jan. 24, 1981, in New York City. Registration fee is $125 for practicing physicans and $50 for residents and fellows. For further information, write Jesse Sigelman, M.D., 515 E. 71st St., New York, NY 10021.

VOLUNTEERS NEEDED IN CAMBODIAN REFUGEE CAMPS

Rotary International has asked Aesculapian International to recruit Rotarian ophthalmologists to serve

as short-term volunteers in the Cambodian refugee camps in Thailand. For further information, write Aesculapian International, Dept. RA, P.O. Box 1319, Santa Barbara, CA 93012.

Hellenic Ophthalmologic Society: 50th Anniversary Celebration and 14th Annual Congress

The celebration of the 50th anniversary of the Hellenic Ophthalmologic Society will be held together with the 14th annual Hellenic ophthalmologic congress in Athens, Greece, May 21-24, 1981. Registration fee is $125. For further information, write John Koliopoulos, Assistant Professor and General Secretary, State General Hospital of Athens, 170 Messogion Ave.-Cholargos, Athens, Greece.

Society for Clinical Trials and Eighth Annual Symposium for Coordinating Clinical Trials: Combined Session

The combined annual scientific sessions of the Society for Clinical Trials and the eighth annual symposium for coordinating clinical trials will be held May 11-13, 1981, in San Francisco. For further information, write Christian R. Klimt, M.D., Secretary, Society for Clinical Trials, Inc., 600 Wyndhurst Ave., Baltimore, MD 21210.

Personals

Cornelius D. Binkhorst

Cornelius D. Binkhorst has been nominated to deliver the second Ridley Medal Lecture during the joint meeting of the International Intra-Ocular Implant Club and the American Intra-Ocular Implant Society during the International Congress of Ophthalmology in San Francisco in November 1982.

Jay M. Enoch

Jay M. Enoch has been appointed dean of the School of Optometry, professor of Physiological Optics and Optometry at the University of California-Berkeley and professor of Ophthalmology, University of California, San Francisco.

Ida Caroline Mann

Ida Caroline Mann was named a Dame of the British Empire on Queen Elizabeth's birthday honors list June 14.

A. Ralph Rosenthal

Professor A. Ralph Rosenthal has been appointed to the Frederick Thorpe Ulverscroft chair of ophthalmology, University of Leicester. The appointment was effective Sept. 1, 1980.

Fight For Sight Awards For Student Fellowship and Travel Grants

The Fight for Sight Scientific Advisory Committee approved the funding of ten Student Fellowships to be activated during the summer of 1980. To assist residents in ophthalmology who have completed a minimum of one year's training, the Fight for Sight funds a limited number of Travel Grants. These awards are intended to help defray travel and per diem costs for residents who are authors or co-authors of a paper to be presented at a scientific meeting or to participate in a symposium or seminar. For further information, write Fight for Sight, Inc., 139 E. 57th St., New York, NY 10022.

Fight for Sight! Student Fellowships
Summer 1980

Investigator and Institution	Direct Supervisor	Project Title	Amount Awarded
James C. Ahn Boston University Boston, Massachusetts	Allen Kupferman, Ph.D.	Quantitative evaluation of the effectiveness of various therapeutic agents used to suppress anterior uveitis	$1,000
Jane H. Broughan* Scheie Eye Institute University of Pennsylvania Philadelphia, Pennsylvania (Burroughs Wellcome Co. Fund)	Herbert Blough, M.D.	Competitive inhibition of herpesvirus complimentary groups	1,050

*Continuation award

Investigator and Institution	Direct Supervisor	Project Title	Amount Awarded
Randall A. Brown Southern Illinois University Carbondale, Illinois	Paul Hargrave, Ph.D.	Transglutaminase labeling of bovine rhodopsin in the disc membrane	$ 900
Dennis M. Defoe* University of California Los Angeles, California (Abraham Silver Memorial Fund)	Dean Bok, Ph.D.	Autoradiographic and radiobiochemical studies of vitamin A metabolism by the vertebrate retina	1,050
Paul G. Fitzgerald University of California Los Angeles, California (Albert G. & Herman B. Mosler Memorial Fund)	Dean Bok, Ph.D.	Studies on the transport of vitamin A from the liver to the retinal pigment epithelium	900
Martin L. Katz* University of California Santa Cruz, California (James Thurber Memorial Fund)	Edward Dratz, Ph.D.	Role of autoxidation in aging and disease of the eye	1,050
Stephen P. Miller Wills Eye Hospital Philadelphia, Pennsylvania	Norman Felberg, Ph.D.	Ocular angiogenesis factor	900
Andrew Mulberg The Mount Sinai Hospital New York, New York (Burroughs Wellcome Co. Fund)	Michael Yablonski, M.D.	Effect of colchicine on aqueous humor dynamics in primates	900
James E. Ramseur, Jr. Cornell Univesity Medical Center New York, New York (Burroughs Wellcome Co. fund)	Stanley Chang, M.D. Jesse Sigelman, M.D.	Profiles of diabetic retinopathy in pregnant diabetic women	700
Uri Shabto The Mount Sinai Hospital New York, New York	Kathryn S. Pokorny, Ph.D.	The effect of cannabinoids on ciliary $(Na^+ + K^+) - ATPase$	900

Fight for Sight! Travel Awards
Spring 1980

Investigator	Institution	Amount Awarded
Penny A. Asbell, M.D.	New York University New York, New York	$450
Emily Chew, Ph.D.	University of Toronto Toronto, Canada	450
Martin Ehrenberg, M.D.	University of Chicago Chicago, Illinois	450
Cynthia J. MacKay, M.D.	Edward S. Harkness Eye Institute New York, New York	450

*Continuation award

INVESTIGATOR	INSTITUTION	AMOUNT AWARDED
Leon G. Partamian, M.D.	University of Chicago Chicago, Illinois	$450
Calvin W. Roberts, M.D.	Edward S. Harkness Eye Institute New York, New York	400
Gareth A. Tabor, Ph.D.	Baylor College of Medicine Houston, Texas	400

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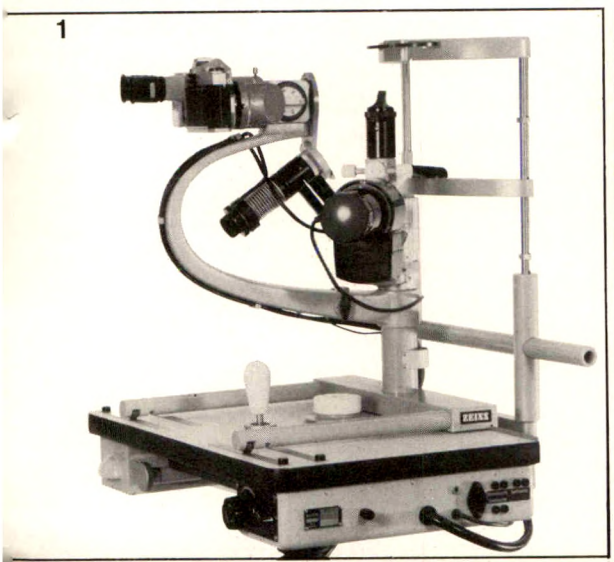
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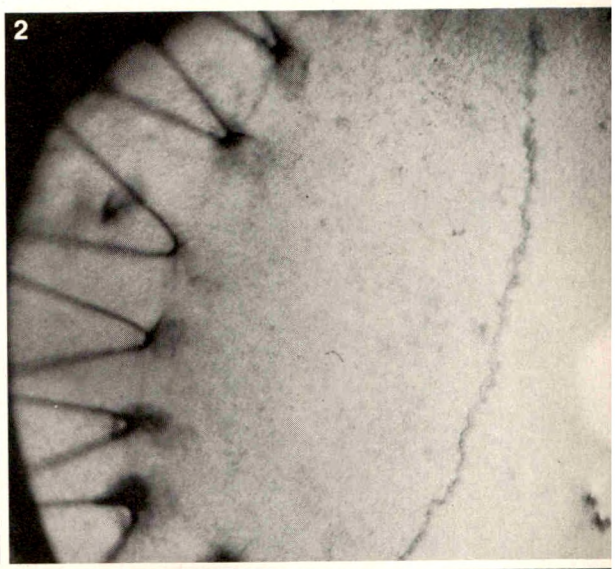
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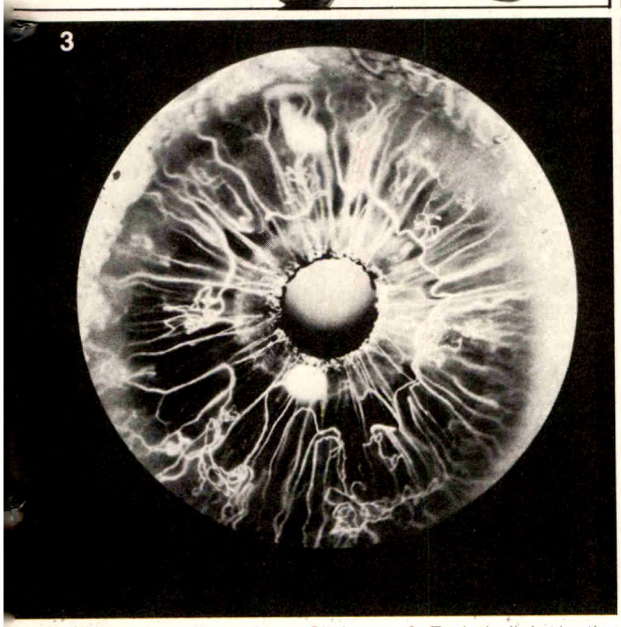
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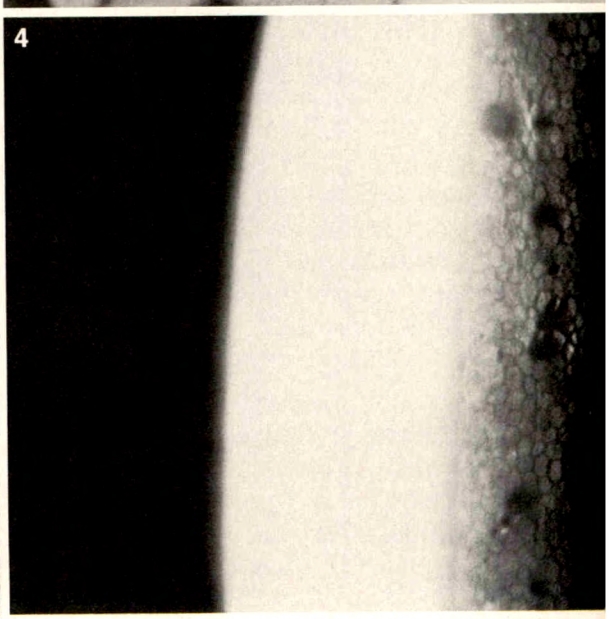
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DIVISION OF OPHTHALMOLOGY, STANFORD UNIVERSITY
announce
THE XVII PALO ALTO COURSE ON DIAGNOSIS & TREATMENT OF RETINAL & CHOROIDAL DISEASES
FEBRUARY 16, 17, 18, 1981
Fairchild Auditorium
Stanford University Medical Center

THE THREE-DAY COURSE WILL PRESENT:
...up-to-date information on retinal & choroidal diseases
...basic knowledge of retinal fluorescein angiography, flurophotometry, ultrasonography, & laser photocoagulation
...indications, results, & complications of photocoagulation
...opportunity to use argon laser photocoagulator (optional)

FACULTY:

Lloyd M. Aiello, M.D.—Joslin Clinic, Boston
Lawrence V. Basso, M.D.—Palo Alto Medical Clinic, Palo Alto
Devron Char, M.D.—University of California, San Francisco
Steven T. Charles, M.D.—University of Tennessee, Memphis
Peter Egbert, M.D.—Stanford University
Robert M. Ellsworth, M.D.—Cornell University, New York
Robert L. Jack, M.D.—Palo Alto Retinal Group & Stanford University
Francis A. L'Esperance, M.D.—Columbia University, New York
Hunter L. Little, M.D.—Palo Alto Retinal Group & Stanford University
A. Edward Maumenee, M.D.—John Hopkins Hospital, Baltimore
Irene Maumenee, M.D.—John Hopkins Hospital, Baltimore
G. Richard O'Connor, M.D.—Proctor Foundation, University of California, San Francisco
Stephen J. Ryan, M.D.—Estelle Doheny Eye Foundation, Los Angeles
Arthur Vassiliadis, Ph.D.—Zweng Memorial Retinal Research Foundation

ADVANCE REGISTRATION IS REQUIRED:

Course Fee: $400 (includes 2 lunches, buffet dinner, & the 4th Zweng Memorial Lecture Banquet)

Registration is payable to: Zweng Memorial Retinal Research Foundation
1225 Crane Street
Menlo Park, California 94025

Course Coordinator: (415) 323-0231

22 Hours Category 1 credit CMA's Continuing Education

NEW YORK OCULAR TUMOR MEETING

Saturday, February 21, 1981

Sponsored by the Combined Ophthalmology Department of the New York Hospital-Cornell Medical Center and Manhattan Eye, Ear and Throat Hospital

A full day course on ocular and adnexal tumors will be organized around a series of 20 case presentations covering the full gamut of benign and malignant tumors of special interest to the practicing ophthalmologist. The audience and panel will participate in the analysis of each case. The course will emphasize diagnosis, operative and therapeutic decisions, and new controversial managements.

The following will be the course participants:

David Abramson, M.D.
James Augsburger, M.D.
D. Jackson Coleman, M.D.
Robert Ellsworth, M.D.
Barrett Haik, M.D.
Paul Henkind, M.D.
Albert Hornblass, M.D.
Frederick A. Jakobiec, M.D.
Ira S. Jones, M.D.
Herbert Katzin, M.D.
Murray Meltzer, M.D.
Jerry Shields, M.D.
James Schutz, M.D.
Frederick Theodore, M.D.
Stephen Trokel, M.D.
Lorenz E. Zimmerman, M.D.

For information and registration please write to:

Frederick A. Jakobiec, M.D.
Manhattan Eye, Ear and Throat Hospital
210 East 64th Street
New York, NY 10021
212-753-7697

University of Miami School of Medicine
Department of Ophthalmology
Bascom Palmer Eye Institute

Announces the

1980 NEURO-OPHTHALMOLOGY COURSE

December 10-13, 1980
Key Biscayne Hotel
Miami, Florida

Topics to be covered include: Optic nerve pathophysiology; New tests of visual function; Radiologic correlations of pituitary and cavernous sinus masses; The child with poor vision; Visual fields; Neuropathology of brainstem and cranial nerves; Ocular myasthenia; Nystagmus diagnosis; Case presentations and discussion; Free paper presentations; Questions and answers; and others.

Faculty for the Course will include:

Douglas R. Anderson, M.D.
Robert B. Daroff, M.D.
Nobel J. David, M.D.
John T. Flynn, M.D.
Joel S. Glaser, M.D.
Richard Lindenberg, M.D.
Judith Donovan Post, M.D.
D. Martin Regan, Ph.D.
Norman J. Schatz, M.D.
Jonathan D. Trobe, M.D.

and others

Registration fee is $275 for practitioners and $150 for residents upon application from their Department Head. Mail registration fee payable to "Continuing Education in Ophthalmology, Inc."; P.O. Box 610326, Miami, Florida 33161. Course hours: 13, Category I, AMA.

Wills Eye Hospital

ANNOUNCES

THE SIXTH ANNUAL OPHTHALMOLOGY REVIEW COURSE

Marriott Motor Inn Hotel
Philadelphia, PA

December 8-12, 1980

GUEST SPEAKER

Jack T. Holladay, M.D.
Director, Vision Unit, Hermann Eye Center, University of Texas, Health Science Center at Houston

HOSPITAL FACULTY

Juan J. Arentsen, M.D.
William E. Benson, M.D.
Vitaliano B. Bernardino, Jr., M.D.
Joseph H. Calhoun, M.D.
David H. Fischer, M.D.
Joseph C. Flanagan, M.D.
Stephen B. Lichtenstein, M.D.
Joseph I. Markoff, M.D.
Michael A. Naidoff, M.D.
Ralph S. Sando, M.D.
Peter J. Savino, M.D.
Jerry A. Shields, M.D.

Peter J. Savino, M.D., Course Director

SUBJECTS

Anatomy and Embryology
Anterior Segment Surgery
Cornea and External Diseases
Glaucoma
Intraocular Inflammation and Immunology
Neuro-Ophthalmology
Ocular Pathology
Ocular Tumors
Optics and Refraction-Contact Lenses
Orbit, Eyelid, Lacrimal System
Pharmacology and Therapeutics
Physiology
Retina and Vitreous
Strabismus
Trauma

An AMA-CME 42-Hour Accredited Course
Category I, Continuing Medical Education

Tuition: $500.00 Residents and Fellows: $250.00
(with identification from their Department of Ophthalmology)

Includes Reception-Dinner and five lunches

FOR REGISTRATION AND INFORMATION CONTACT:

Department of Continuing Medical Education
Wills Eye Hospital
9th and Walnut Streets
Philadelphia, PA 19107

Phone: (215) 928-3258

THE DUKE
ADVANCED VITREOUS SURGERY COURSE
PART I: MANAGEMENT OF TRACTION RETINAL DETACHMENTS

Intended for the vitreo-retinal surgeon with some experience.

Detailed presentations and discussions of surgical techniques, complications and instrumentation used in the management of traction retinal detachments.

Discussion of taped surgery.

Ample time for exchange of experiences.

All this in the beautiful spring environment of North Carolina from April 10 to 11, 1981.

FACULTY
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George W. Blankenship, Miami
Steve Charles, Memphis
Brooks W. McCuen, Durham
Maurice B. Landers, Durham
Robert Machemer, Durham
Ronald Michels, Baltimore

Applications To:
Ms. Cathy E. Moore
Duke University Eye Center
P.O. Box 3802
Durham, N.C. 27710
Phone: 919-684-3891

AMA Category I

Registration Fee: $200.

1981
INTERNATIONAL CATARACT SURGERY SYMPOSIUM

February 16-17-18, 1981

La Posada Resort Hotel — Scottsdale, Arizona

FACULTY

Ronald W. Barnet, United States
D. Peter Choyce, England
Henry M. Clayman, United States
Robert C. Drews, United States
Herbert L. Gould, United States
Kenneth J. Hoffer, United States
Harold A. Stein, Canada

For information please write: The Eye Foundation (AFORE)
421 North 18th Street, Suite 105
Phoenix, Arizona 85006
Phone: (602) 257-1077

XIII CONGRESO PANAMERICANO DE OFTALMOLOGIA ACAPULCO, MEXICO 1981

XIII PAN AMERICAN CONGRESS OF OPHTHALMOLOGY
MAY 10-15, 1981
ACAPULCO, MEXICO

For applications and further information,
please write to the:

XIII PAN AMERICAN CONGRESS
OF OPHTHALMOLOGY
Apartado Postal No. 11—566
México 11, D.F.
MEXICO

OPHTHALMOLOGY BOARD AND CLINICAL REVIEW COURSE

Sponsored By
The Department of Ophthalmology
The University of Texas
Health Science Center at Houston
The Medical School

Course Director
Jack T. Holladay, M.D.

The Ophthalmology Board and Clinical Review Course, an intensive one week course intended to prepare practicing ophthalmologists and recent graduates of residency programs for the OKAP and American Board of Ophthalmology Examinations, will begin at 7:30 a.m. on Monday, November 17th and continue thru Saturday, November 22, 1980.

TUITION including lunch: $500.00

For more information contact:

Department of Ophthalmology
Hermann Eye Center
Hermann Hospital
1203 Ross Sterling
Houston, Texas 77030
Phone: (713) 797-1777

Announcing—

50th ANNIVERSARY MID-WINTER CLINICAL CONFERENCE IN OPHTHALMOLOGY

January 26, 27, 28, 1981

Presented by

The Research Study Club of Los Angeles, Inc.

ANTERIOR SEGMENT PROBLEMS and POSTERIOR SEGMENT COMPLICATIONS

GUEST SPEAKERS

Joseph A. Baldone, M.D. • Miles A. Galin, M.D.
John Hetherington, M.D. • Walter J. Stark, M.D.
William Tasman, M.D.

PANEL MODERATORS

Clarence A. Albaugh, M.D. • S. Rodman Irvine, M.D.
Irving Leopold, M.D., D. Sc. • Stephen J. Ryan, M.D.
Bradley R. Straatsma, M.D.

SURGICAL ANATOMY COURSE

Instructors

Crowell Beard, M.D. • James L. Hargiss, M.D. • Bernice Brown, M.D.
Saturday, January 24, 1981, 9:00 A.M. to 5 P.M. & Sunday, January 25, 9:00 AM—5 PM

FEE

Practicing physicians: $100, if attending Mid-Winter Course—$150, if not attending
Residents: $75, if attending Course; $100, if not attending Course.

Registration Deadline—January 1, 1981

(Category 1 Credit 24 hours, CMA, AMA)

Registration Fee $200.00 for Practicing Physicians
$100.00 for Residents

Fee includes: Reception & Dinner (incl. spouses) and 3 "Round Table" lunches

For Additional Information: Research Study Club, P.O. Box 49412, Los Angeles, California 90049

Emory University School of Medicine
Department of Ophthalmology

Pediatric Ophthalmology and Strabismus: Update

December 1-2, 1980

Course Director:
J. Allen Gammon, M.D., M.P.H., Atlanta, Georgia

Course Faculty:
Frank C. Bell, M.D., Atlanta, Georgia
David G. Campbell, M.D., Atlanta, Georgia
Melvin R. Carlson, M.D., Seattle, Washington
H. Dwight Cavanagh, M.D., Atlanta, Georgia
Howard Eggers, M.D., New York, New York
David L. Guyton, M.D., Baltimore, Maryland
Arthur J. Jampolsky, M.D., San Francisco, California
Henry J. Kaplan, M.D., Atlanta, Georgia
Philip Knapp, M.D., New York, New York
Clinton D. McCord, Jr., M.D., Atlanta, Georgia
Travis A. Meredith, M.D., Atlanta, Georgia
Marshall M. Parks, M.D., Washington, D.C.
James Richard, M.D., Oklahoma City, Oklahoma
William E. Scott, M.D., Iowa City, Iowa
Robert H. Spector, M.D., Atlanta, Georgia
Frankie W. Stegall, B.S., Atlanta, Georgia
David Taylor, F.R.C.S., M.R.C.P., London, England
George O. Waring, M.D., Atlanta, Georgia
John Wright, M.D., Boston, Massachusetts

Core topics of Pediatric Ophthalmology and Strabismus. Visual development, management of congenital cataracts, latest surgical techniques for strabismus. Panel discussion on controversial topics and national state of the art in pediatric ophthalmology and strabismus. AMA Credit Category I.

RESERVATIONS: Write to Colony Square Hotel, Peachtree & 14th Street, Atlanta, Georgia 30361, 404/892-6000, for hotel accommodations.

ADVANCE REGISTRATION: Ophthalmologists $295, Orthoptists, Technicians $150, Residents, Medical Students, Orthoptic students $100 with letter. Checks payable to Emory University. Send to: Course Coordinators, 1339 Clifton Road, N.E., Atlanta, Georgia 30322.

ALSO ANNOUNCING:
Second Annual F. Phinizy Calhoun, Jr., M.D. Lecture - December 1, 1980.
Sixth Annual Emory/Grady Eye Residents Alumni Meeting - December 3, 1980.

7TH ANNUAL
ROCKY MOUNTAIN
NEURO-OPHTHALMOLOGY
COURSE

THE YARROW
PARK CITY, UTAH
JANUARY 21-24, 1981

R. Burde · St. Louis
T. Carlow · Albuquerque
J. Corbett · Iowa City
L. Dell'Osso · Cleveland
J. Glaser · Miami
J. Kennerdell · Pittsburgh
J. McCrary · Houston
G. Sanborn · Salt Lake City
P. Savino · Philadelphia
N. Schatz · Philadelphia
R. Seigel · Albuquerque
S. Thompson · Iowa City
B. Wilson · Denver
B. Younge · Rochester
D. Zauel · Indianapolis

This course will include lectures and seminar sessions covering ocular motility, the pupil and visual system disorders. Ample time between sessions has been provided for leisure activity in Park City and the Park City Ski Area.

Registration is limited.
A separate brochure mailing is not planned.

For information, please contact:

Rocky Mountain Neuro-Ophthalmology Society
302 Juniper Hill Road, N.E.
Albuquerque, New Mexico 87122

SHEETS EYE FOUNDATION
&
TEMPLE UNIVERSITY
DEPT. OF OPHTHALMOLOGY
Presents
Extracapsular Cataract Extraction
&
The Sheets Posterior Chamber Lens

Dates and Locations:

April 18, 1980	Ft. Lauderdale
May 16	Los Angeles
July 25	Kansas City
October 2	Houston
October 24	Philadelphia
October 25	New York City
November 14	Atlanta
January 30, 1981	Phoenix
February 20	Vail

Fee: $150
Category 1 CME Credits: 4 hrs.

For information contact:
Nancy Fraass, Administrator,
McGhan Medical, PO Box 6447,
Santa Barbara CA 93111 or phone
collect 805/964-8711

THE MIAMI COURSE IN PHACOEMULSIFICATION

Sponsored By:

MIAMI EYE FOUNDATION

in cooperation with

BASCOM PALMER EYE INSTITUTE

University of Miami School of Medicine

MIAMI DADE COMMUNITY COLLEGE

and

ST. FRANCIS HOSPITAL

DECEMBER 19-20-21, 1980

Faculty

Henry M. Clayman, M.D.
Mark S. Jaffe, M.D.
Norman S. Jaffe, M.D.
Charles Kelman, M.D.
Lyle Moses, M.D.
C. William Simcoe, M.D.

COURSE DIRECTOR:
HENRY M. CLAYMAN, M.D.

course includes

- practical labs for phaco (Cavitron)
- observation of live surgery
- interworking of phaco machine
- indications, contraindications, complications
- implant labs
- extracapsular cataract surgery

Registration: Lyn Johnson
Miami Eye Foundation
1680 Michigan Avenue-Suite 1104
Miami Beach, Florida 33139
U.S.A.

Tuition $1000
Checks Payable to Miami Eye Foundation

(305)532-1768

THE POSTGRADUATE INSTITUTE OF THE NEW YORK EYE AND EAR INFIRMARY ANNOUNCES THE SIXTEENTH ANNUAL REVIEW COURSE "BASIC SUBJECTS IN OPHTHALMOLOGY"

DECEMBER 1 through 6, 1980

SUBJECTS

PHYSIOLOGY • PHARMACOLOGY • OCULAR MOTILITY
NEURO OPHTHALMOLOGY • MEDICAL OPHTHALMOLOGY
DEVELOPMENTAL ANOMALIES • PEDIATRIC
OPHTHALMOLOGY • ANATOMY and HISTOPATHOLOGY OF
THE EYE • DISEASES of the CORNEA and CONJUNCTIVA
INTERPRETATIONS of POSTERIOR POLE LESIONS
PRINCIPLES of PHYSICAL OPTICS and REFRACTION

COURSE DIRECTOR: JAMES C. NEWTON, M.D.

FACULTY

Ronald Carr, M.D.
D. Jackson Coleman, M.D.
Andrew P. Ferry, M.D.
David S. Friendly, M.D.
John S. Hermann, M.D.
Frank B. Hoefle, M.D.
Thomas Kuhns, M.D.
Peter J. G. Maris, M.D.
Robert C. Mickatavage, M.D.
Richard Ruiz, M.D.
Whitney G. Sampson, M.D.

This six-day intensive course is designed as a comprehensive review of the basic subjects in Ophthalmology and should be especially helpful to those physicians preparing for the American Board Certification Examinations and those having to fulfill requirements for continuing medical education. The course qualifies for 48 credits in Category 1 for the AMA-CME Physicians Recognition Award.

Tuition: $500.00 (Luncheons and Cocktail Party included)

For Application and further information, please write:
Jane Stark, Registrar • Postgraduate Institute
New York Eye and Ear Infirmary 310 East Fourteenth Street New York, New York 10003

The Southern California Permanente Medical Group
and
The University of California, San Diego
present
The Third Annual Ophthalmology Symposium "Ocular Trauma, Uveitis and Retinal Disorders"

January 30 and 31, 1981
at
Little America Westgate Hotel,
San Diego, California

Guest Participants:
James G. Diamond, M.D., Stuart L. Fine, M.D., Morton F. Goldberg, M.D., G. Richard O'Connor, M.D., Stephen J. Ryan, M.D., plus other prominent speakers.

Co-Chairmen:
Lawrence P. Newman, M.D.,
Michael Goldbaum, M.D.

14 hours of Category I Credit will be offered. Registration will be limited. For further information regarding program and hotel arrangements, please contact:
Southern California Permanente Medical Group
Education Office, 4647 Zion Avenue
San Diego, CA 92120, (714) 563-4298

THE ESTELLE DOHENY EYE FOUNDATION AND THE UNIVERSITY OF SOUTHERN CALIFORNIA DEPARTMENT OF OPHTHALMOLOGY PRESENT



ESTELLE DOHENY EYE FOUNDATION

A VITREOUS SURGERY SYMPOSIUM

Director Kenneth R. Diddie, M.D., February 20-21, 1981 • New Otani Hotel Los Angeles

Michael H. Goldbaum, M.D.
Klaus Heilmann, M.D.
Alexander R. Irvine, M.D.
Allan E. Kreiger, M.D.
Robert Machemer, M.D.
James B. Miller, M.D.
Thomas M Aaberg, M.D.
George Blankenship, M.D.
Steven T. Charles, M.D.
James Diamond, M.D.
Kenneth R. Diddie, M.D.
H. McKenzie Freeman, M.D.
Ronald E. Smith, M.D.
Manfred Spitznas, M.D.
Walter H. Stern, M.D.
Felipe Tolentino, M.D.
C. P. Wilkinson, M.D.
Ronald G. Michels, M.D.
Richard R. Ober, M.D.
Conor C. O'Malley, M.D.
Stephen J. Ryan, M.D.
John Scott, M.D.

TOPICS TO INCLUDE: Vitrectomy Instrumentation in the 1980's • Diabetic Retinopathy • Vitrectomy in Retinal Detachment Surgery Massive Periretinal Proliferation, Giant Tears—Silicone Oil, Posterior Holes, Virectomy in Eye Trauma • Virectomy Techniques for the General Ophthalmologist • Controversial Topics in Vitreous Surgery

Registration Fee: $200, For Information: Nancy Call Coordinator—Continuing Education, 1533 San Pablo Street, Box J, Los Angeles, California 90033 (213) 224-7752

Semi-Annual Cadaver Courses
of the
New York Medical College
Westchester County Medical Center

OCULOPLASTIC SURGICAL DISSECTION COURSE—May 13-16, 1981
(40 HRS. CME & CAT I)

FACULTY:
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H. Gould, M.D.
M. Guibor, C.O.
P. Guibor, M.D.
J Hagedoorn, Ph.D
S. Hecht, M.D.
L. Herman, Ph.D.
A. Messina, M.D.
N. Pastorek, M.D.
E. Wiggs, M.D.
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and others.



SUBJECTS:
Cosmetic Blepharoplasty, Blepharoptosis, Trauma Surgery, Lid Reconstruction, Lacrimal Surgery, Orbital Reconstruction, Entropion, Ectropion, Orbital Fractures, Orbital Tumors and Chemosurgery.

SPECIAL FEATURES:
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Clip coupon and mail with registration fee to:

Ms. Paula Tamkin, Registrar
c/o Dr. P. Guibor, 630 Park Avenue
New York, N.Y. 10021 (212) 734-1010

May 13-16, 1981
OCULOPLASTIC REGISTRATION FORM
Registration **$650.00**

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Specialty: Ophth—ENT—Plastic—Derm
(circle one)

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February 14-21, 1981
International Oculoplastic
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Venezuela
March 15, 1981
American Society Contemporary Ophth,
Oculoplastic Section
Disney World—Orlando, Florida

UNIVERSITY OF SOUTH FLORIDA
Department of Ophthalmology
Tampa, Florida

presents

OCULOPLASTIC-ORBITAL UPDATE
MARCH 5, 6, 7, 1981

This course is offered to provide the practicing surgeon with a sound clinical foundation in the current diagnosis and management of orbital disease and other problems of the ocular adnexa. Topics include: cosmetic and cancer surgery of the eyelids, surgical treatment of ptosis, lacrimal surgery, and newer techniques in the evaluation of orbital disease.

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Arthur S. Grove, Jr., M.D. — Boston
Charles R. Leone, Jr., M.D. — San Antonio
Clinton D. McCord, Jr., M.D. — Atlanta
Lawrence R. Muroff, M.D. — Tampa
Jay J. Older, M.D. — Tampa
Allen M. Putterman, M.D. — Chicago
James A. Rush, M.D. — Tampa
Orkan G. Stasior, M.D. — Albany
Richard R. Tenzel, M.D. — Miami
Elise Torczynski, M.D. — Tampa
Robert R. Waller, M.D. — Rochester

Registration fee $250.00
($125.00 Residents and Fellows)

Contact: James A. Rush, M.D., Oculoplastic-Orbital Update, USF College of Medicine
MDC Box 21, Tampa, Florida 33612, (813) 974-2030 or 974-2031

KRESGE EYE INSTITUTE

of

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Presents a five-day intensive continuing education course

OPHTHALMOLOGY: BASIC AND CLINICAL REVIEW

November 28-December 2, 1980

Hyatt Regency Dearborn • Dearborn, Michigan

CORNEA AND EXTERNAL DISEASES (4 hours)	Juan J. Arentsen, M.D., Assistant Professor Jefferson University; Director of Medical Education, Wills Eye Hospital
NEUROANATOMY AND NEURO-OPHTHALMOLOGY (4 hours)	John L. Keltner, M.D., Chairman, Department of Ophthalmology, Associate Professor of Ophthalmology, Neurology and Neurological Surgery, University of California, Davis
PATHOLOGY I & II (8 hours)	Myron Yanoff, M.D., Chairman, Department of Ophthalmology, University of Pennsylvania; Director of Scheie Eye Institute
EMBRYOLOGY AND ANATOMY (4 hours)	David M. Worthen, M.D., F.A.C.S., Professor and Head, Division of Ophthalmology, University of California San Diego
STRABISMUS (4 hours)	Robert D. Reinecke, M.D., Professor and Chairman, Department of Ophthalmology, Albany Medical College of Union University
OPTICS AND REFRACTION (6 hours)	Jack T. Holladay, M.D., Unit Director, Low Vision Unit, Hermann Eye Center, University of Texas Health Science Center at Houston
GLAUCOMA AND METABOLIC DISEASES (4 hours)	H. Saul Sugar, M.D., Clinical Professor, Department of Ophthalmology, Wayne State University School of Medicine; Consultant, Sinai Hospital of Detroit; Consultant Glaucoma, Henry Ford Hospital
PHARMACOLOGY AND THERAPEUTICS (4 hours)	Joel S. Mindel, M.D., Ph.D., Assistant Professor, Department of Ophthalmology, Mt. Sinai School of Medicine
MEDICAL OPHTHALMOLOGY (4 hours)	Paul Henkind, M.D., Ph.D. Professor and Chairman, Department of Ophthalmology, Montefiore Hospital and Medical Center

COURSE DIRECTOR: Maurice Croll, M.D. Co-Director Frank Nesi, M.D.

THIS COURSE IS AN EXCELLENT AID IN
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REGISTRATION FEE: $450.00 (includes syllabus, coffee breaks, evening reception and luncheons)
Residents $250.00

Category I Continuing Medical Education: 42 hours

For more information, write or call:
Wayne State University School of Medicine, Division of Continuing Medical Education,
9B Health Care Institute, Detroit, Michigan 48201,(313) 577-1180

Make checks payable to: WAYNE STATE UNIVERSITY

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West Point, PA 19486

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Scheie Eye Institute
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July 1, 1981

Applications are being accepted for a one-year fellowship at the Scheie Eye Institute of the University of Pennsylvania.

A comprehensive program of Medical Retinal Vascular diseases, Retinal Detachment surgery, Retinal Photocoagulation surgery and Vitreous surgery is being offered.

Interested parties please contact:

Alexander J. Brucker, M.D.
Retina Service
Scheie Eye Institute
51 North 39th Street
Philadelphia, Pennsylvania 19104
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Neuro-Ophthalmology-Glaucoma Fellowship, 1981.

University of Connecticut Health Center. One year, second year optional, clinical and academic training. Three years residency prerequisite. Inquiries:

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Department of Ophthalmology,
University of Pennsylvania
Scheie Eye Institute
Presbyterian-University of Pennsylvania Medical Center

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Myron Yanoff, M.D.
Director, Scheie Eye Institute
51 N. 39th Street
Philadelphia, PA 19104

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DAVID SHOCH, M.D.
Executive Secretary
THE HEED OPHTHALMIC FOUNDATION
303 EAST CHICAGO AVENUE
CHICAGO, ILLINOIS 60611

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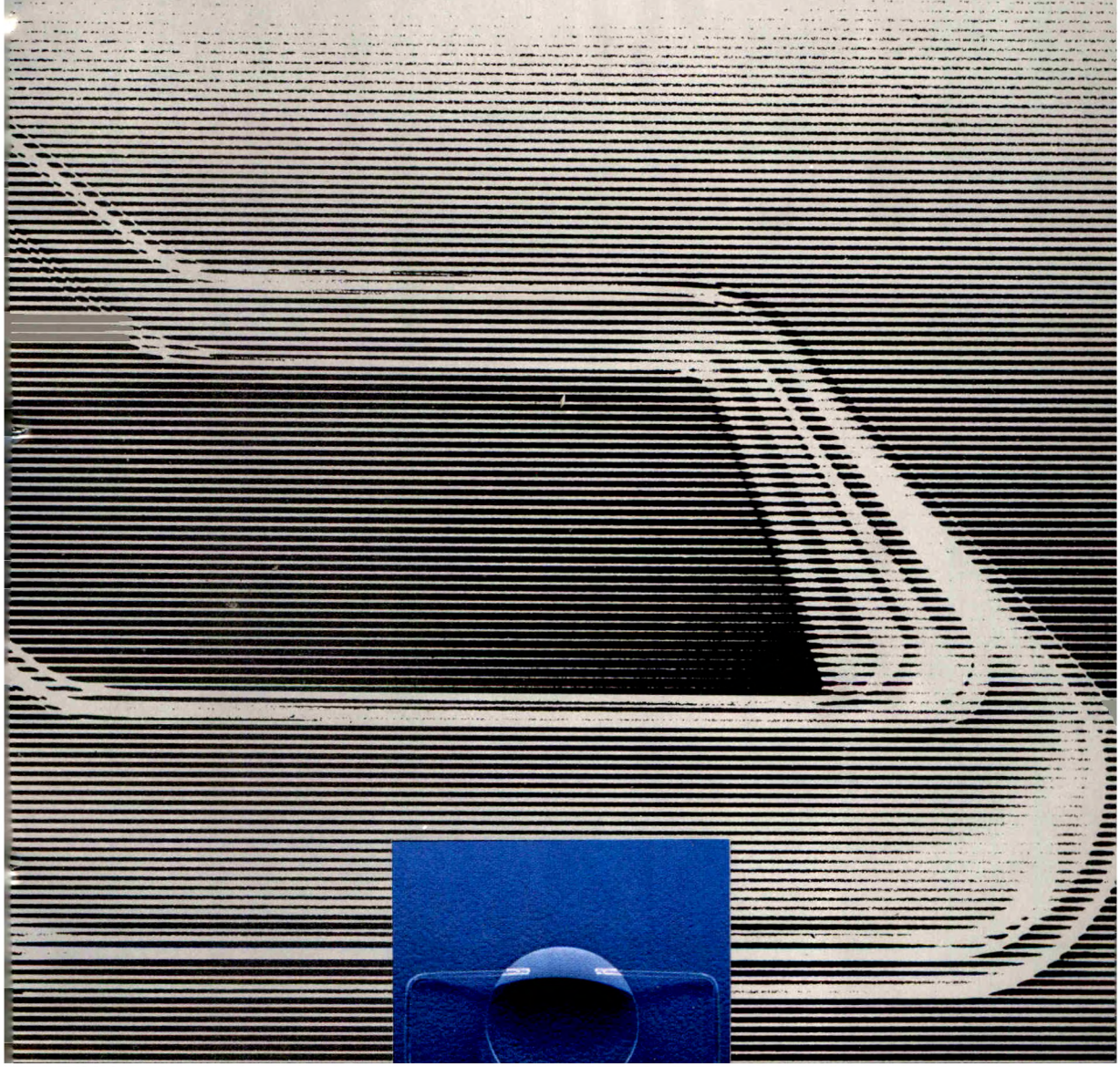
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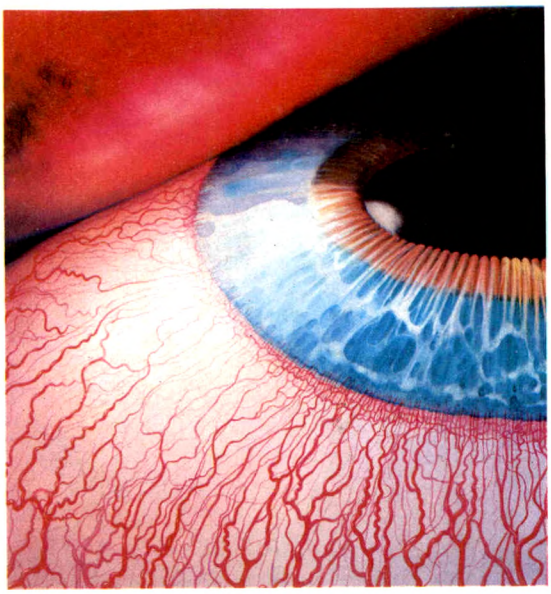
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[1]Leibowitz HM, Kupferman A. Bioavailability and therapeutic effectiveness of topically administered corticosteroids. Trans Am Acad Ophthalmol Otolaryngol 1975;79(1):OP78-OP88.

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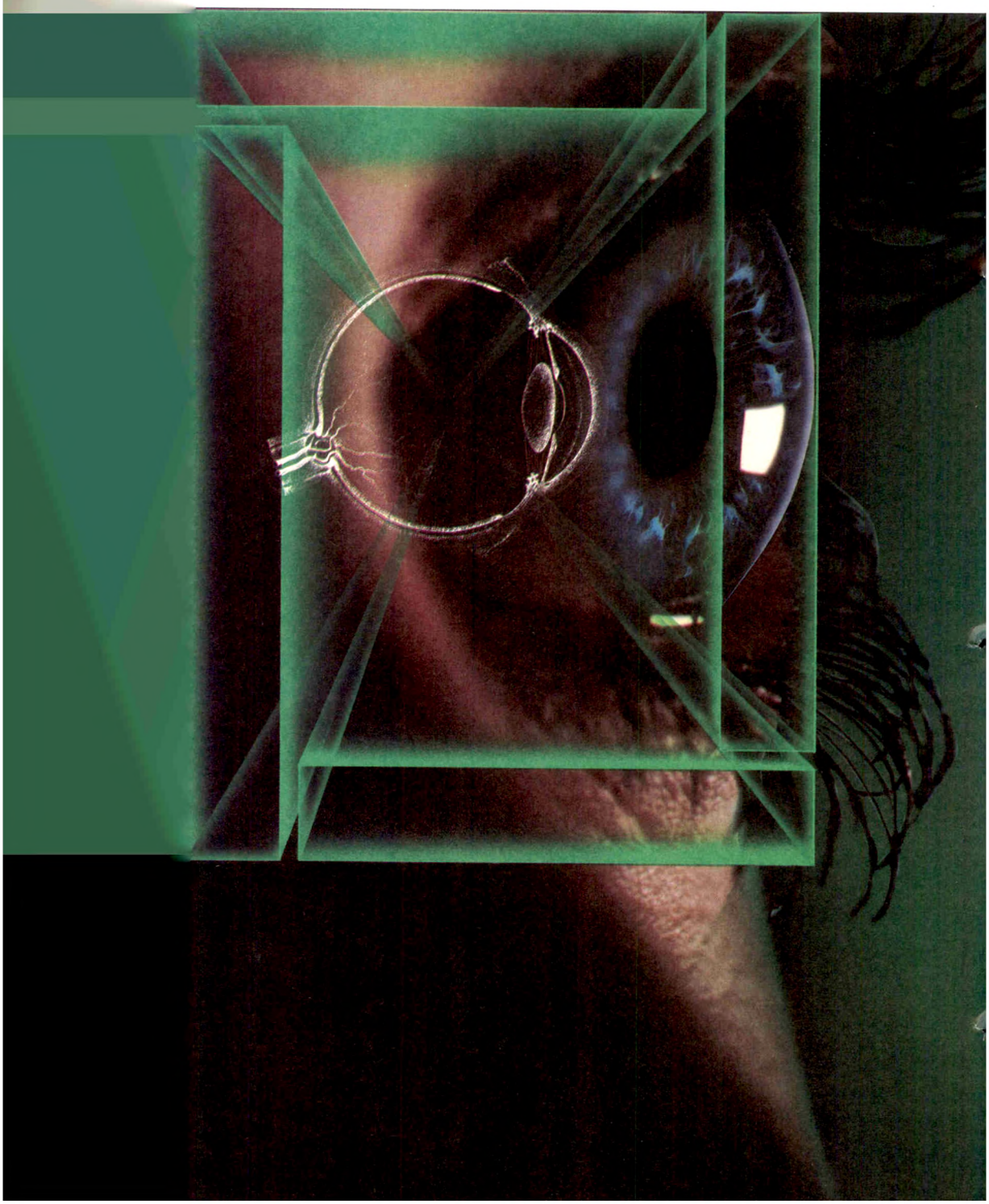
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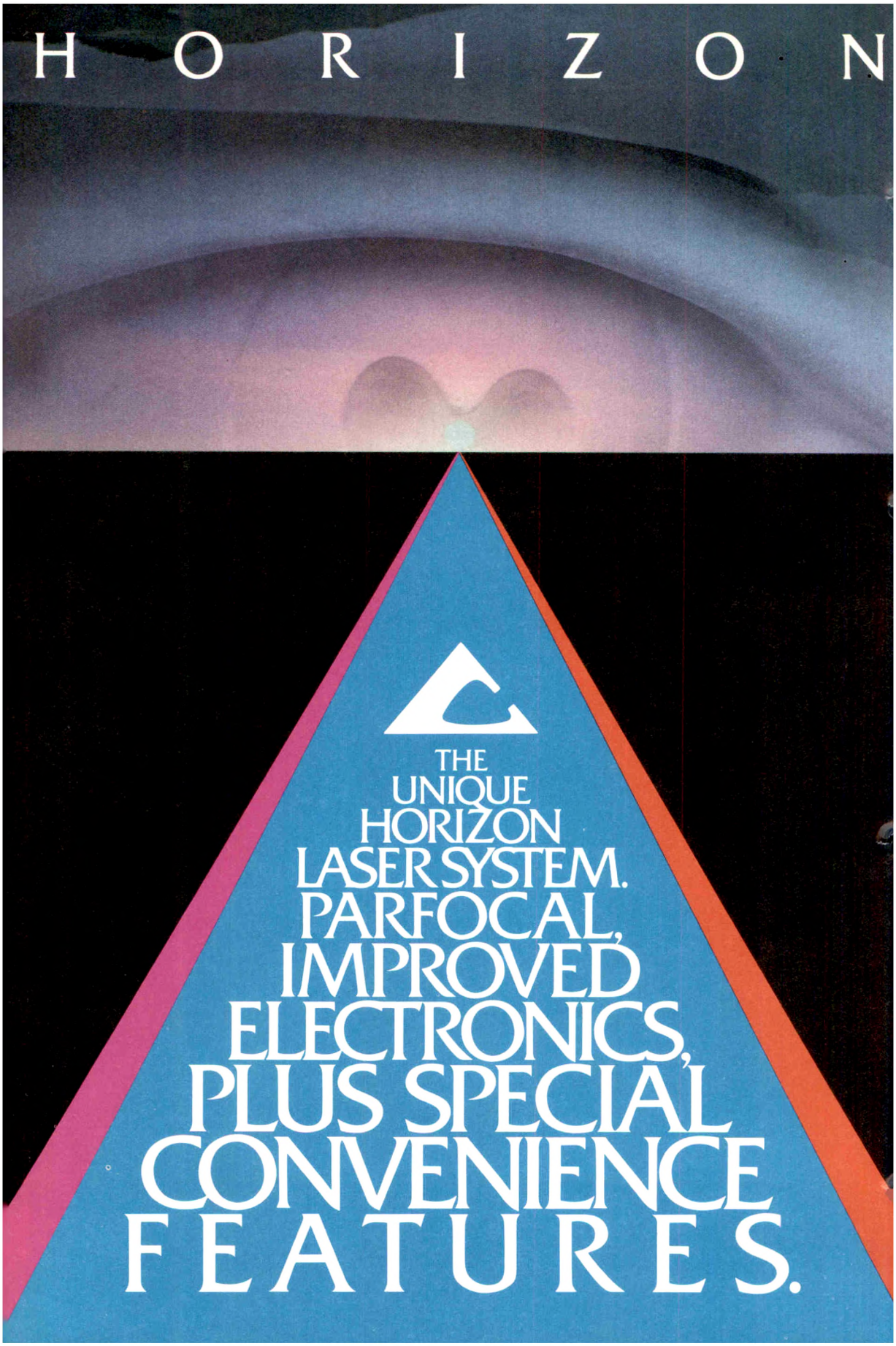
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1. Data on file at Alcon Laboratories.
2. Zimmerman, T. J., Ophthalmology Audio Digest, Vol. 17, No. 1

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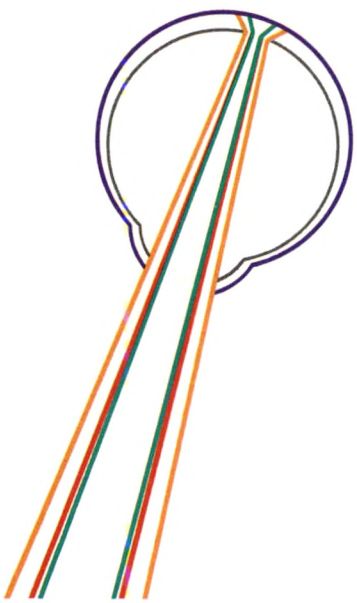
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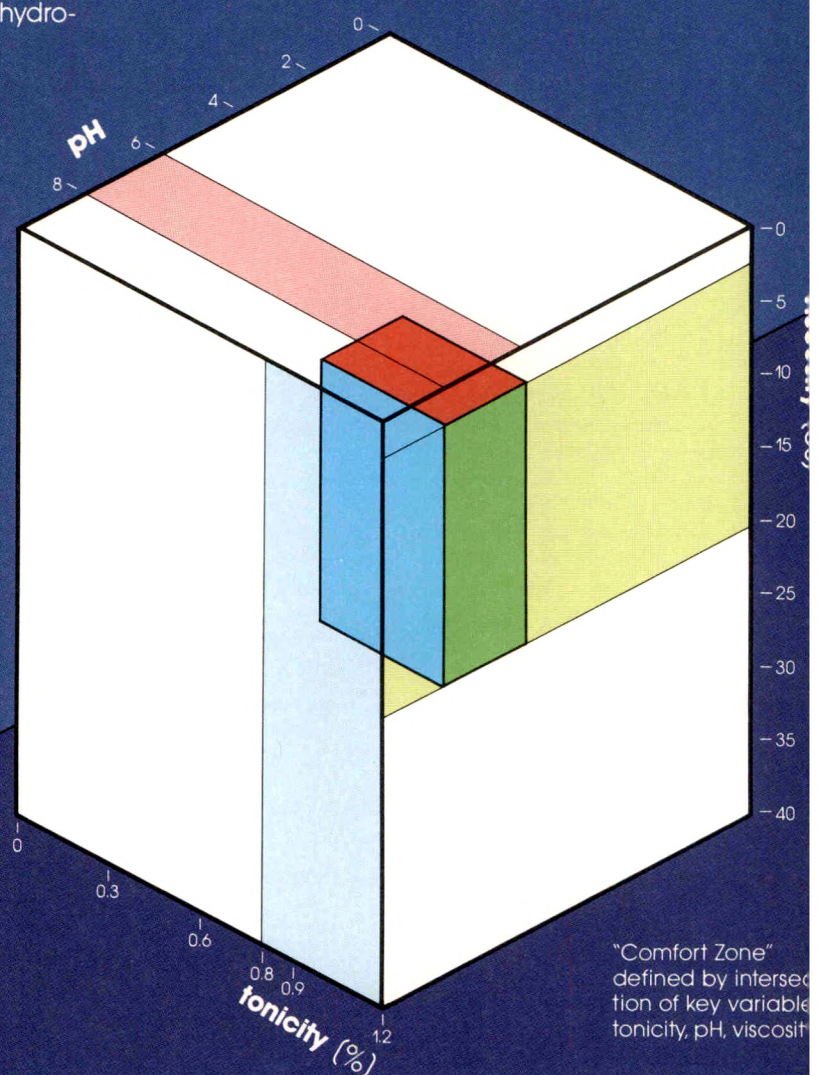
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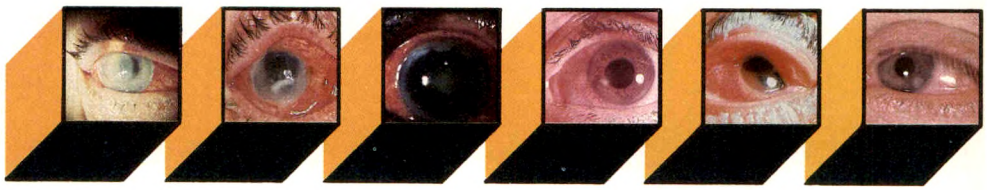
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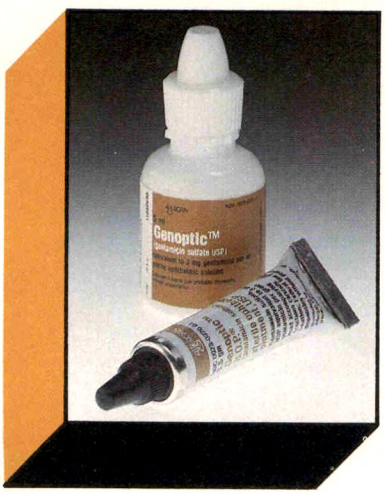
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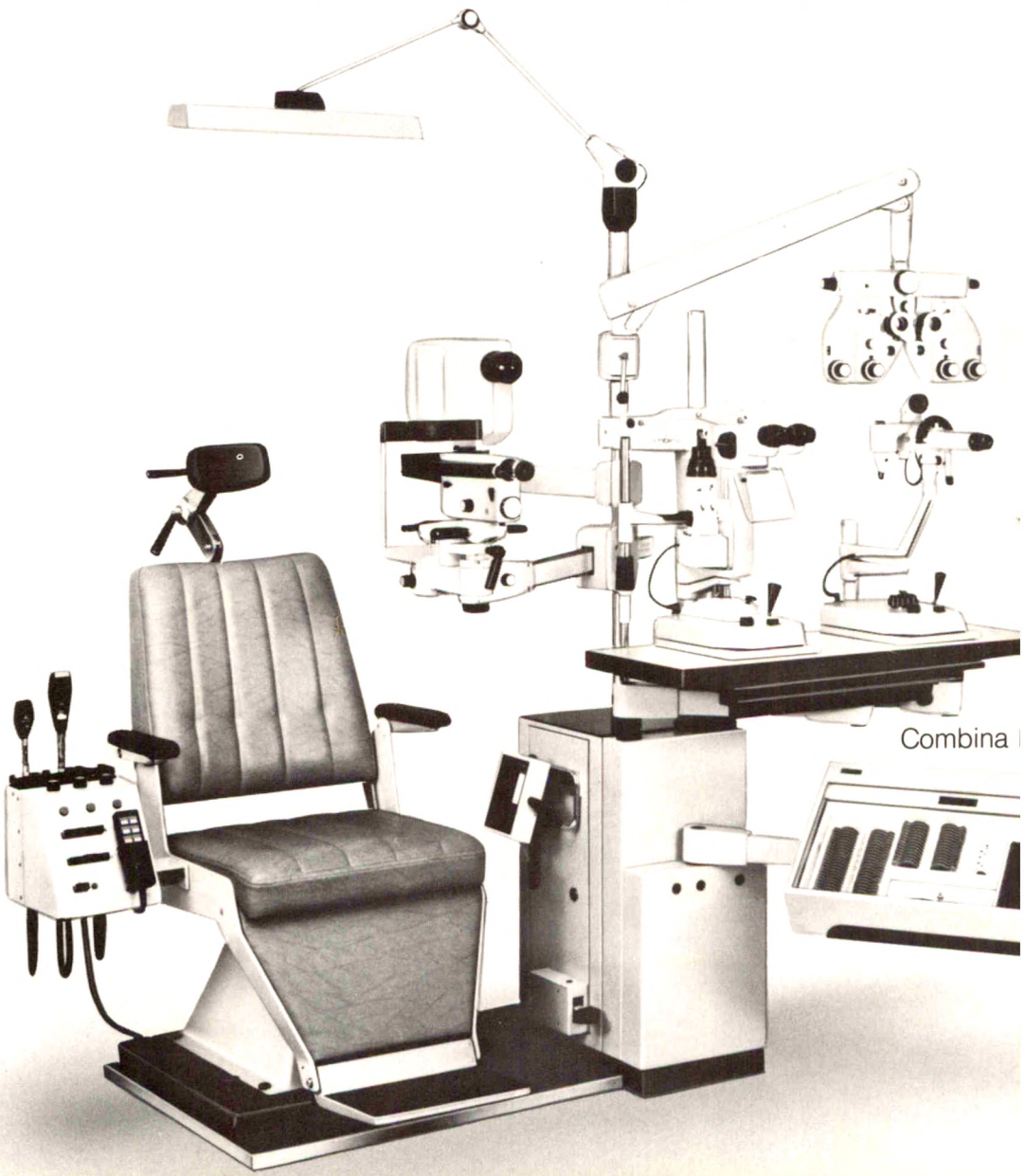
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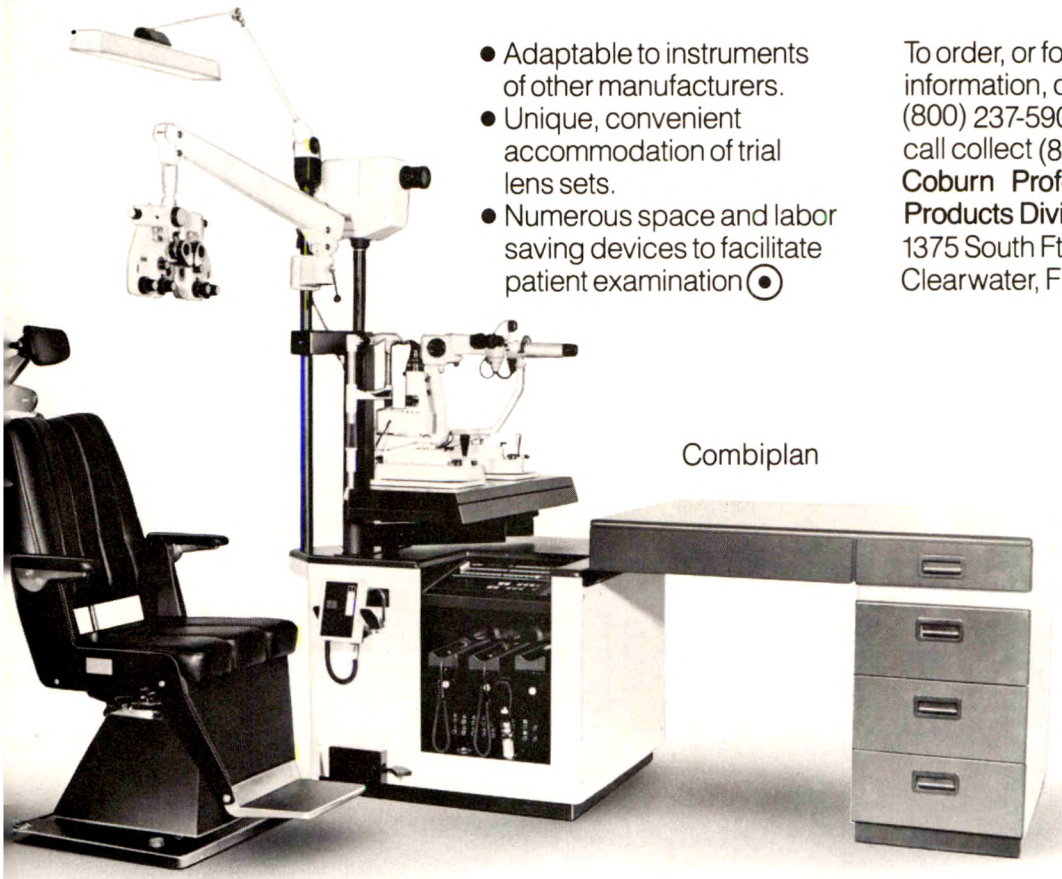
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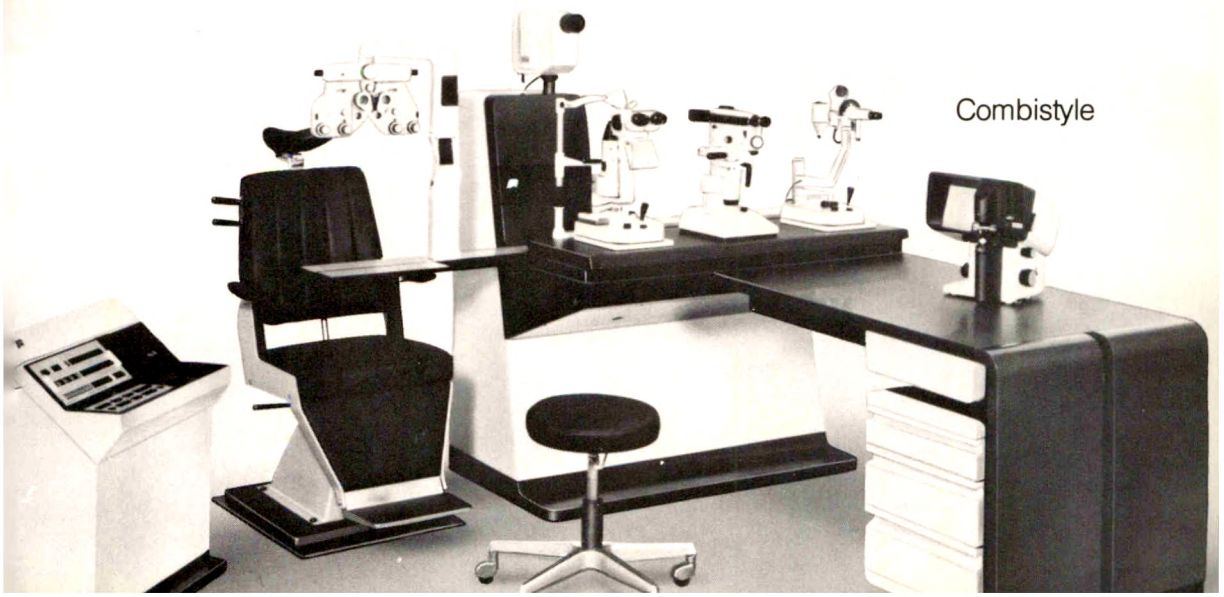


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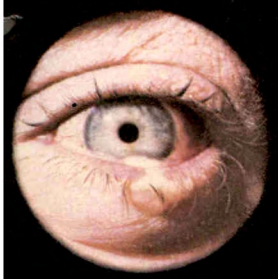
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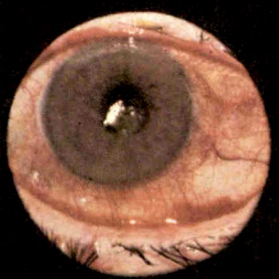
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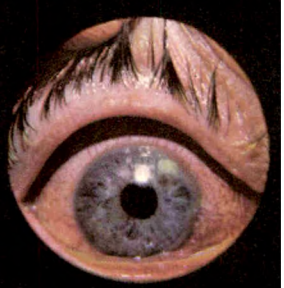
Corneal ulcer. Etiology: S. aureus. Before treatment.



Acute hemorrhagic conjunctivitis. Etiology: Pneumococcus. Before treatment.

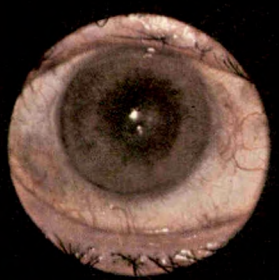


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Do not resterilize.

PRECAUTIONS
The VICRYL suture knots must be properly placed to be secure. Place the first throw in precise position for the final knot, using a double loop; tie the second throw square; using horizontal tension; additional throws are advisable.

Coated VICRYL sutures, which are treated to enhance handling characteristics, require the standard surgical technique of flat and square ties with additional throws if indicated by surgical circumstance and the experience of the surgeon.

Skin and conjunctival sutures remaining in place longer than 7 days may cause localized irritation and should be removed as indicated.

Acceptable surgical practice must be followed with respect to drainage and closure of infected wounds.

ADVERSE REACTIONS
Reactions reported in clinical trials which may have been suture related have been minimal. These include skin redness and induration; rare instances of hemorrhage, anastomotic leakage, wound separation in the eye, and abscesses.

DOSAGE AND ADMINISTRATION
Use as required per surgical procedure.

HOW SUPPLIED
VICRYL sutures are available sterile as braided dyed (violet) strands in sizes 9-0 thru 3 (metric size 0.3—6) and undyed (natural) strands in sizes 8-0 thru 3 (metric size 0.4—6), in a variety of lengths, with or without needles, and on LIGAPAK ligating reels. VICRYL sutures, monofilament, dyed (violet) are available in sizes 10-0 (metric size 0.2) and 9-0 (metric size 0.3), in a variety of lengths with needles.

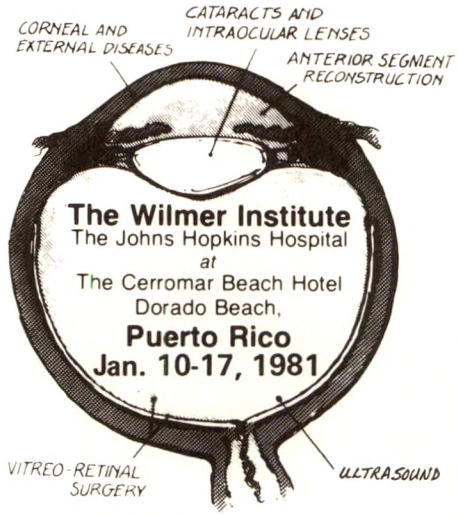
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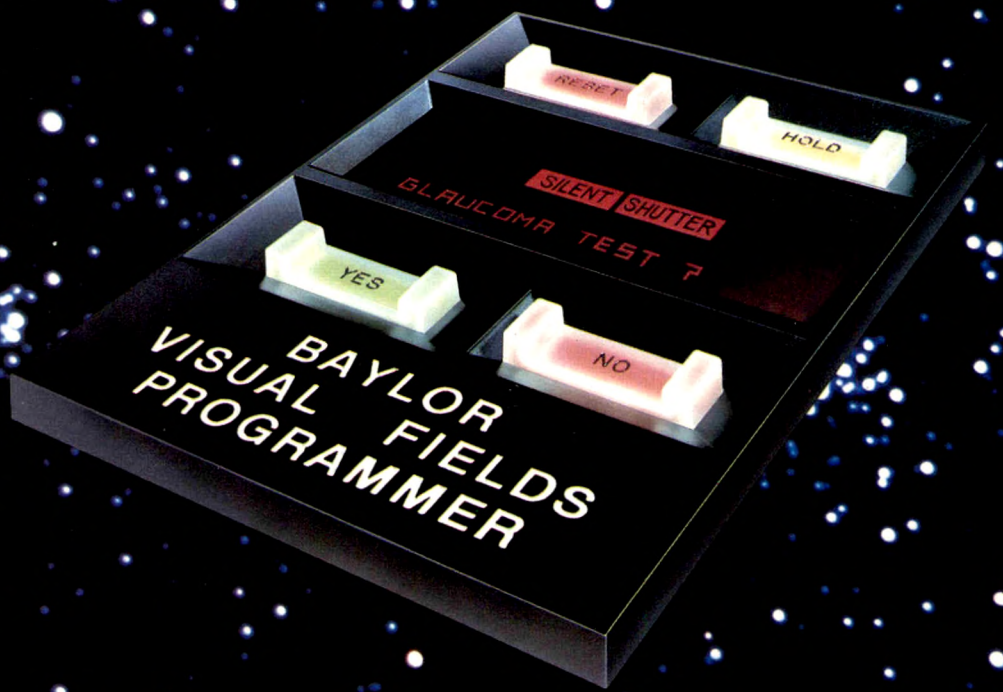
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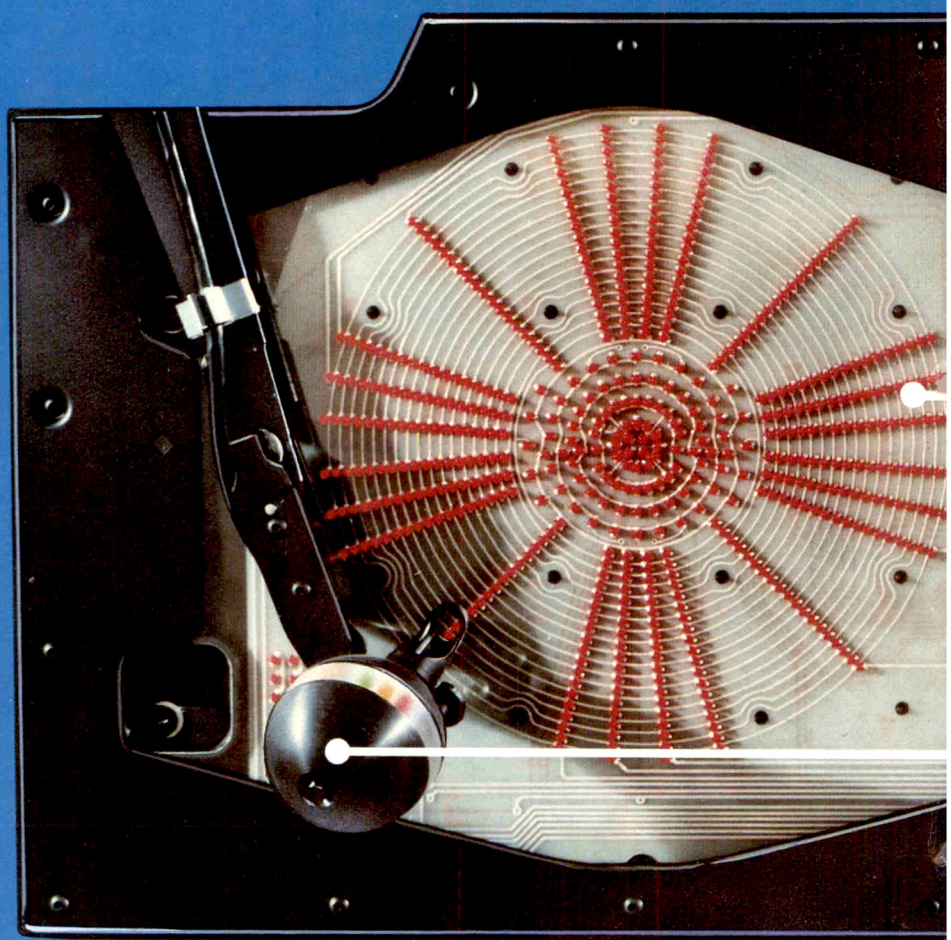
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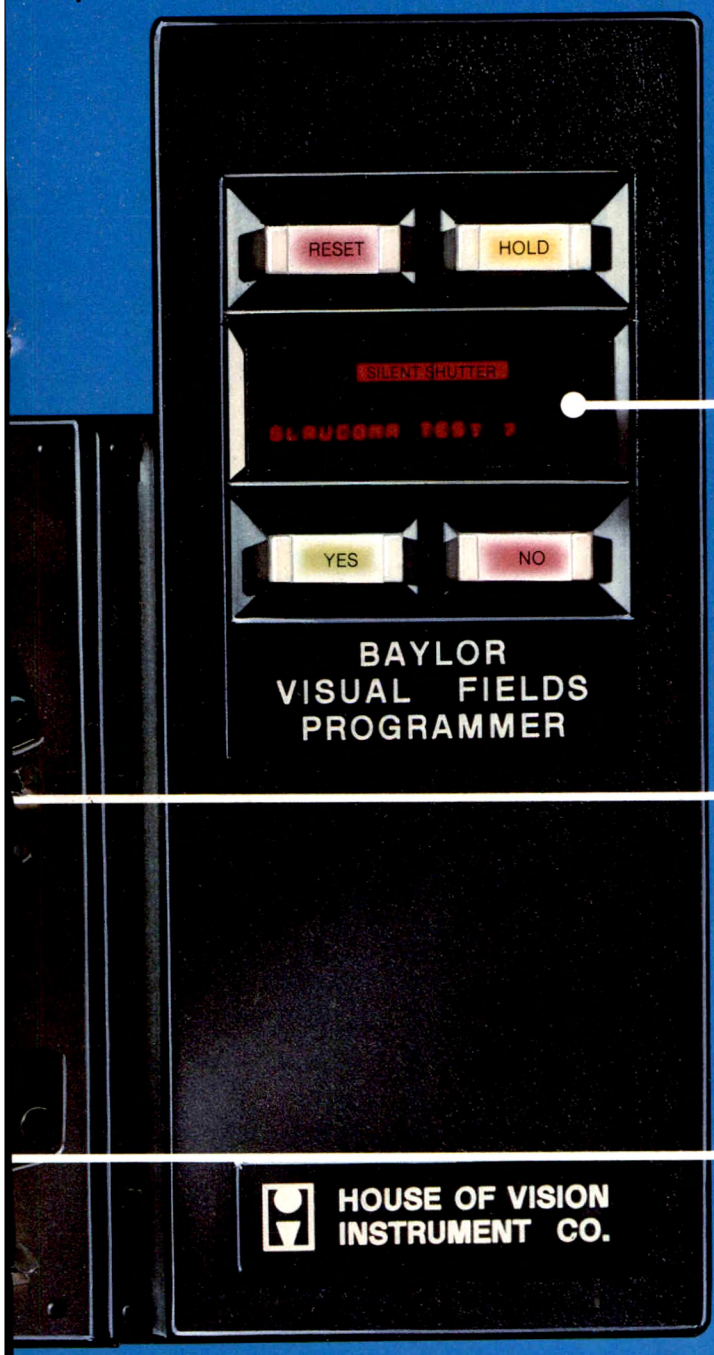
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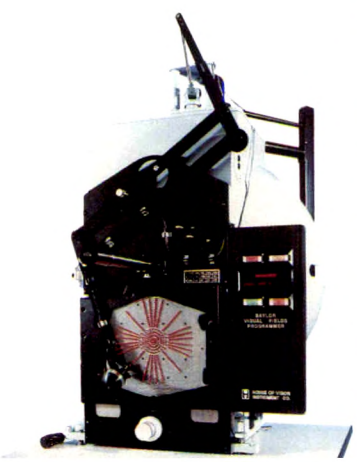
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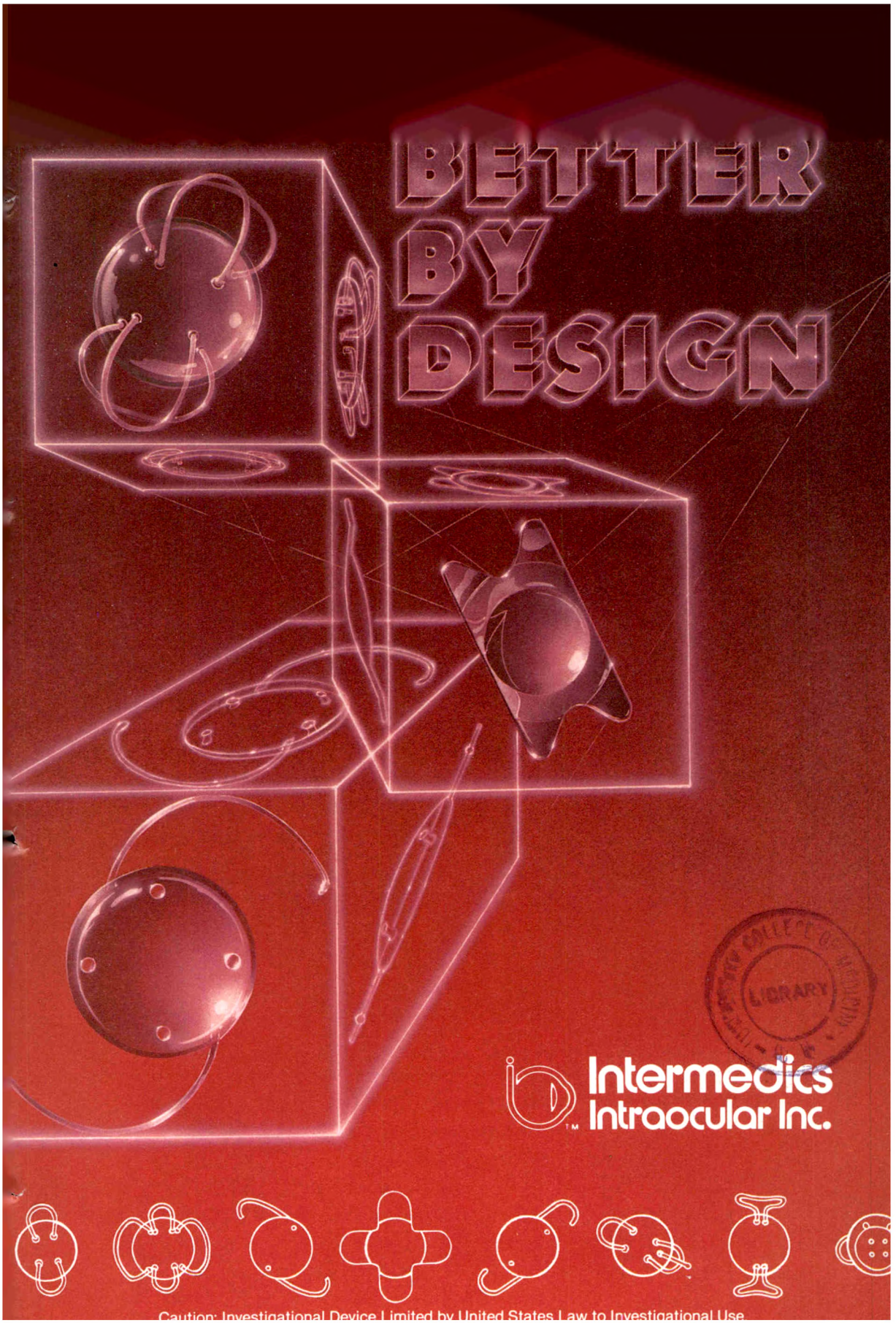
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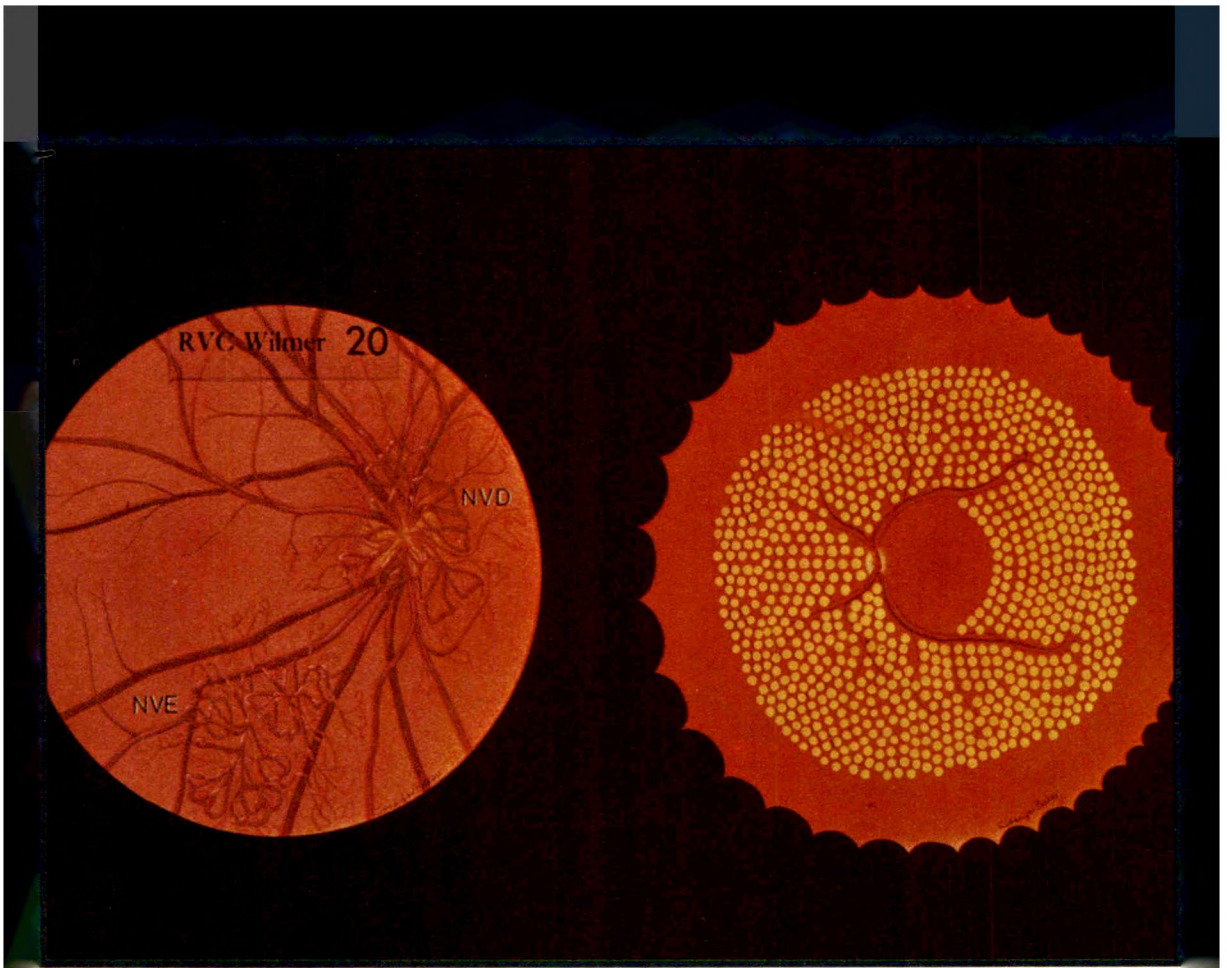
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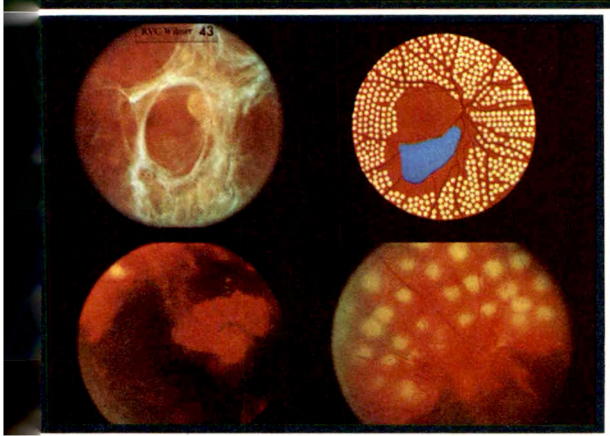
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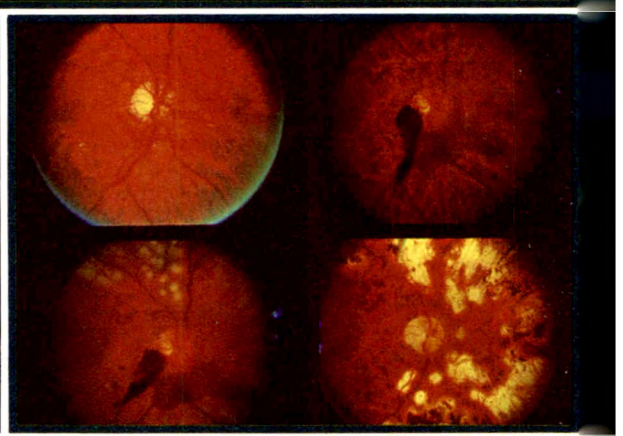
Illustrations clockwise:
Slide 20. The diabetic retinopathy study
Slide 42. Urgency of treating neovascularization
Slide 43. Contraindications to treatment

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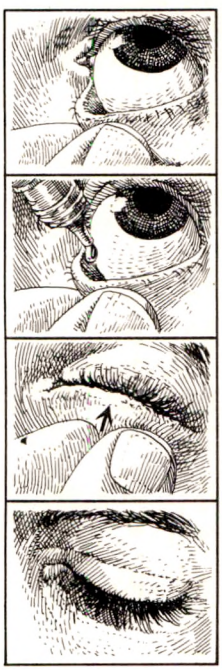
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1
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2
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3
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1. Fraunfelder FT, Hurwitz J, Marsey M: Presentation at Las Vegas Meeting of Association for Research in Vision and Ophthalmology and the American Academy of Ophthalmology and Otolaryngology, Oct 6, 1976, as reported in *Clinical Trends in Ophthalmology*, April 1977.

In a controlled study comparing a variety of common techniques, retention of eye drops on the eye after a five-minute interval went from 16% to 53% when the pouch technique was utilized in the under 20 age group. A 64% retention rate was achieved in the 50–60-year-old age group.

See adjacent page for brief prescribing information.

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INDICATIONS: For the short-term treatment of superficial external ocular infections caused by organisms susceptible to one or more of the antibiotics.

CONTRAINDICATIONS:
Contraindicated in those persons who have shown sensitivity to any of the components.

WARNINGS:
Prolonged use may result in overgrowth of nonsusceptible organisms. Ophthalmic Ointment may retard corneal healing.

PRECAUTIONS:
Culture and susceptibility testing should be performed during treatment.

Allergic cross-reactions may occur which could prevent the use of any or all of the following antibiotics for the treatment of future infections: kanamycin, paromomycin, streptomycin, and possibly gentamicin.

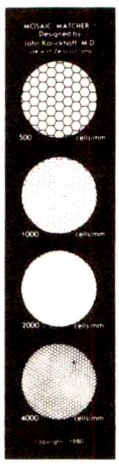
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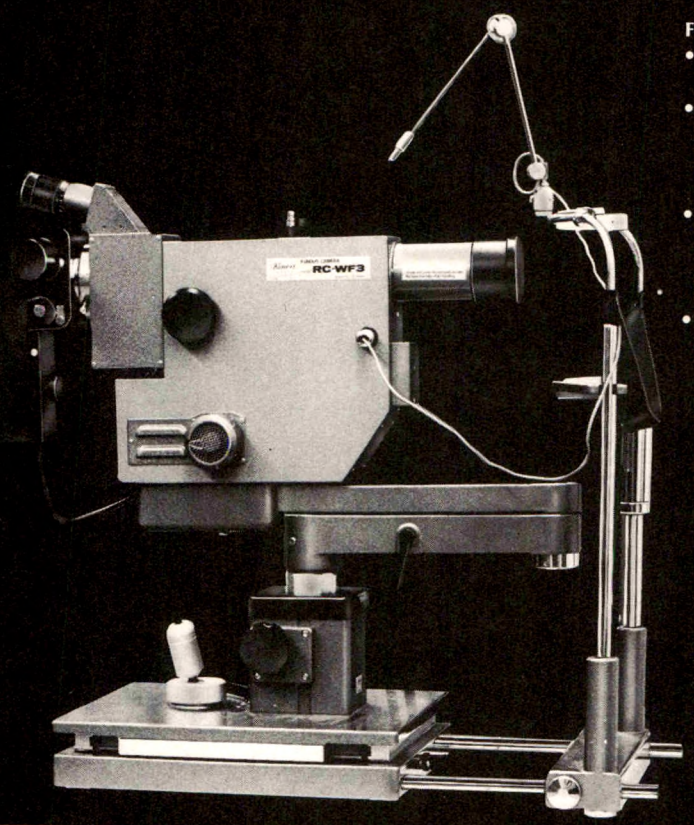
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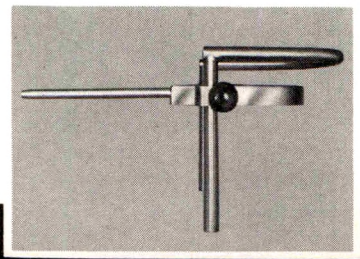
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Harry A. Quigley and W. Richard Green

An ultrastructural study of the retina in the Jansky-Bielschowsky type of neuronal ceroid-lipofuscinosis.
Hans H. Goebel, Wolfgang Zeman, and Eckhart Damaske

A specific enzyme defect in gyrate atrophy.
Muriel I. Kaiser-Kupfer, David Valle, and Luis A. Del Valle

Oculocutaneous albinism associated with Apert's syndrome.
Sheila Margolis, Irwin M. Siegel, Andrew Choy, and Goodwin M. Breinin

Ocular findings in mannosidosis.
Amir I. Arbisser, A. Linn Murphree, Charles A. Garcia, and R. Rodney Howell

Pigmentary degeneration of the retina in the Hallervorden-Spatz syndrome.
Frank W. Newell, Royce O. Johnson II, and Peter R. Huttenlocher

Scanning electron microscopy of congenital corneal leukomas (Peters' anomaly).
Frank M. Polack and Enrique L. Graue

Successful lens removal in congenital corneolenticular adhesion (Peters' anomaly).
George O. Waring III and Marshall M. Parks

Primary familial amyloidosis of the cornea.
E. Lee Stock and Richard A. Kielar

A clinical and histopathological study of François-Neetens speckled corneal dystrophy.
Don H. Nicholson, W. Richard Green, Harold E. Cross, Kenneth R. Kenyon, and Darcy Massof

Sclerocornea associated with the Smith-Lemli-Opitz syndrome.
Robert L. Harbin, Jeffrey I. Katz, Jaime L. Frias, I. Matthew Rabinowicz, and Herbert E. Kaufman

Posterior amorphous corneal dystrophy.
Emmett F. Carpel, Robert J. Sigelman, and Donald J. Doughman

The corneal endothelium and the spectrum of essential iris atrophy.
David G. Campbell, M. Bruce Shields, and Taylor R. Smith

Clinical differentiation of recessive congenital hereditary endothelial dystrophy and dominant hereditary endothelial dystrophy.
G. Frank Judisch and Irene H. Maumenee

The autosomal dominant syndrome of progressive optic atrophy and congential deafness.
Carol R. Kollarits, Marilyn L. Pinheiro, Edwin R. Swann, Daniel F. Marcus, and W. Stephen Corrie

Aniridia, cataracts, and Wilms' tumor in monozygous twins.
Edward Cotlier, Marc Rose, and Steven A. Moel

Ophthalmic features of chromosome deletion 4 p- (Wolf-Hirschhorn syndrome).
Lloyd M. Wilcox, Jr., Lionel Bercovitch, and Rufus O. Howard

Pathologic features of the eye in Down's syndrome with relationship to other chromosomal anomalies.
Joseph Ginsberg, Mary K. Bofinger, and James R. Roush

Retinoblastoma in a patient with a 13qXp translocation.
Harold E. Cross, Ronald C. Hansen, Grant Morrow III, and John R. Davis

Factors for improved genetic counseling for retinoblastoma based on a survey of 55 families.
E. A. Carlson, R. D. Letson, N. K. C. Ramsay, and R. J. Desnick

Cytogenetic investigation of cat-eye syndrome.
Richard G. Weleber, Janina Walknowska, and David Peakman

Ocular and cerebral involvement in familial lymphohistiocytosis.
Richard E. Appen, Steven W. Weber, Guillermo de Venecia, and Gabriele M. Zu Rhein

Vitreoretinal degeneration as a sign of generalized connective tissue diseases.
Irene H. Maumenee

Clinical observations in 101 consecutive patients with Duane's retraction syndrome.
Sherwin Isenberg and Martin J. Urist

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Marriott Motor Inn Hotel
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December 8-12, 1980

GUEST SPEAKER

Jack T. Holladay, M.D.
Director, Vision Unit, Hermann Eye Center, University of Texas, Health Science Center at Houston

HOSPITAL FACULTY

Juan J. Arentsen, M.D.
William E. Benson, M.D.
Vitaliano B. Bernardino, Jr., M.D.
Joseph H. Calhoun, M.D.
David H. Fischer, M.D.
Joseph C. Flanagan, M.D.
Stephen B. Lichtenstein, M.D.
Joseph I. Markoff, M.D.
Michael A. Naidoff, M.D.
Ralph S. Sando, M.D.
Peter J. Savino, M.D.
Jerry A. Shields, M.D.

Peter J. Savino, M.D., Course Director

SUBJECTS

Anatomy and Embryology
Anterior Segment Surgery
Cornea and External Diseases
Glaucoma
Intraocular Inflammation and Immunology
Neuro-Ophthalmology
Ocular Pathology
Ocular Tumors
Optics and Refraction-Contact Lenses
Orbit, Eyelid, Lacrimal System
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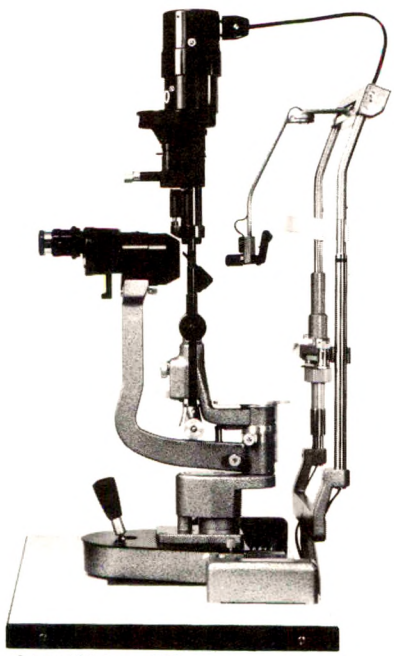
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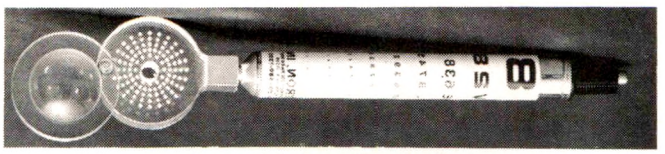
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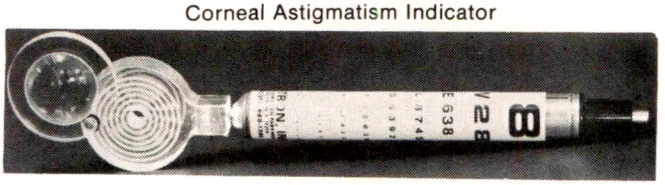
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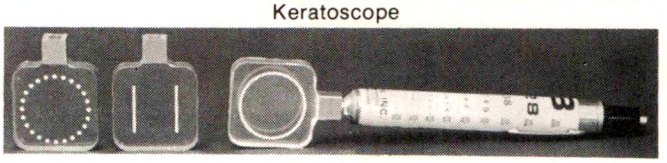
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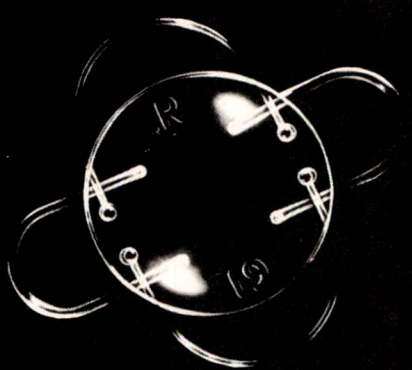


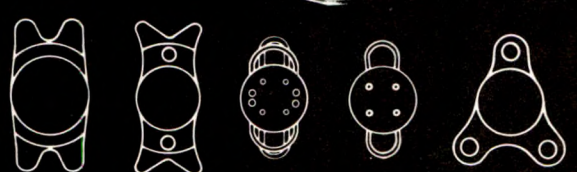
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In an open, long-term maintenance study* of the effect of continuous therapy (up to 40 months) of chronic open-angle glaucoma with TIMOPTIC, 96% of the patients exhibited IOP values of ≤ 21 mm Hg at each measurement during treatment with TIMOPTIC...and the drug was generally well tolerated. (See graphs and data on facing page.)

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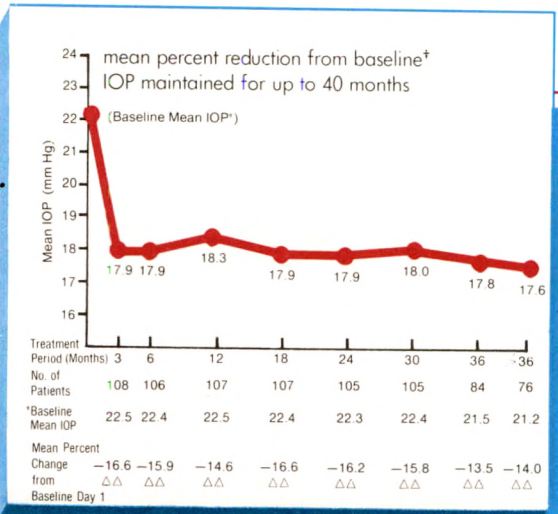
Patients on TIMOPTIC generally do not experience the following therapy-limiting side effects: night blindness/blurred vision/dimming of vision.**

Patients on TIMOPTIC have significantly fewer and less severe side effects than those on pilocarpine or epinephrine, i.e., a much lower incidence of ocular discomfort—such as less burning, smarting, itching, browache, and headache.**

TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product. As with other topically applied ophthalmic drugs, this drug may be absorbed systemically. As with the use of other antiglaucoma drugs, diminished responsiveness to TIMOPTIC after prolonged therapy has been reported in some patients.

*Data on file, Merck Sharp & Dohme Research Laboratories. Additional information available on request.

**Based on controlled multiclinic studies. Data on file, Merck Sharp & Dohme Research Laboratories. Additional information available on request.

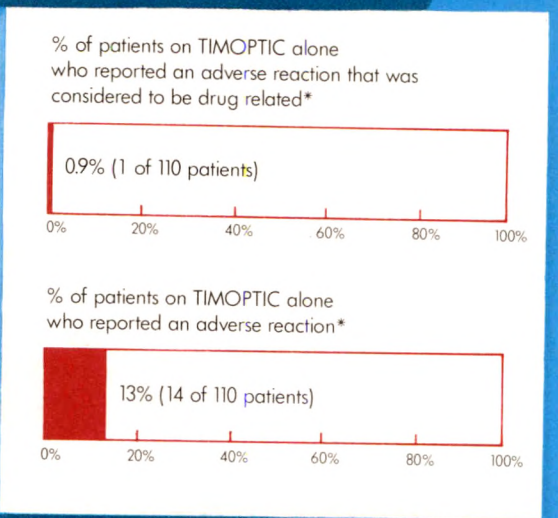


long-term efficacy of Timoptic® (Timolol Maleate) MSD

There was virtually no loss of therapeutic effect in patients treated for up to 40 months with TIMOPTIC alone, i.e., the IOP of almost all patients was lowered to 21 mm Hg or below throughout the study.*

†Of the 110 patients on TIMOPTIC alone, 91 were on previous antiglaucoma treatment and had a treated baseline mean IOP of 21.4 mm Hg. The 19 patients with no previous antiglaucoma treatment had an untreated baseline mean IOP of 27.3 mm Hg. Thus, the mean IOP at baseline for the combined group is 22.5 mm Hg.

ΔΔSignificant percent change from baseline, $p < 0.01$.



long-term tolerability of Timoptic® (Timolol Maleate) MSD

During the 40-month study,* 14 of 110 patients (13%) on TIMOPTIC alone reported an adverse reaction, but only one of these, an allergic reaction, was considered to be drug related.

Visual disturbances including refractive changes (due to withdrawal of miotic therapy in some cases) have been infrequently associated with therapy with TIMOPTIC. Rarely, aphakic cystoid macular edema has been reported, but a causal relationship to therapy with TIMOPTIC has not been established.

*Data on file, Merck Sharp & Dohme Research Laboratories. Additional information available on request.

For a brief summary of prescribing information, please see following page.

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- aphakic glaucoma
- some patients with secondary glaucoma
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- some patients who respond inadequately to multiple antiglaucoma drug therapy

How to start patients on TIMOPTIC:
Usual starting dosage: one drop 0.25% TIMOPTIC in each eye twice a day.

How to transfer from current therapy to TIMOPTIC:
1. On the first day: continue with the agent(s) already being used and add one drop 0.25% TIMOPTIC in each eye b.i.d.

2. On the second day: discontinue the previously used agent(s) and continue with TIMOPTIC in each eye b.i.d.

If clinical response is not adequate:
Dosage may be increased (from the 0.25% solution) by changing to one drop 0.5% TIMOPTIC twice a day in each eye. Dosages above one drop of 0.5% TIMOPTIC twice a day generally have not been shown to produce further reduction of IOP. If the intraocular pressure is maintained at satisfactory levels, the dosage schedule may be changed to one drop once a day in each eye.

Because of diurnal variations in intraocular pressure, satisfactory response to the once-a-day dose is best determined by measuring the intraocular pressure at different times during the day.

In patients with a history of severe cardiac disease, pulse rates should be checked.

CONTRAINDICATIONS: TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product.
WARNINGS: As with other topically applied ophthalmic drugs, this drug may be absorbed systemically.
PRECAUTIONS: Should be used with caution in patients with known contraindications to systemic use of beta-adrenergic receptor blocking agents. These include bronchospastic disease; sinus bradycardia and greater than first-degree block; cardiogenic shock; cardiac failure; concomitant use with adrenergic-augmenting psychotropic drugs. In patients with a history of severe cardiac disease, pulse rates should be checked. Patients who are already receiving a beta-adrenergic blocking agent orally and who are given TIMOPTIC should be observed for a potential additive effect either on the intraocular pressure or on the known systemic effects of beta blockade.

Sufficient clinical data have not been obtained to show safety or efficacy in narrow-angle or angle-closure glaucoma.

As with the use of other antiglaucoma drugs, diminished responsiveness to TIMOPTIC after prolonged therapy has been reported in some patients. However, in one long-term study in which 96 patients have been followed for at least 3 years, no significant difference in mean intraocular pressure has been observed after initial stabilization.

Pregnancy: TIMOPTIC has not been studied in human pregnancy. The use of TIMOPTIC in pregnant women requires that the anticipated benefit be weighed against possible hazards.

Use in Children: Since clinical studies in children have not been done, TIMOPTIC is not recommended for use in children at this time.

Animal Studies: No adverse ocular effects were observed in rabbits and dogs administered TIMOPTIC topically in studies lasting one and two years respectively.

Lifetime studies with timolol have been completed in rats at oral doses of 25, 100, and 300 mg/kg/day and in mice at oral doses of 5, 50, and 500 mg/kg/day. In male and female rats and male mice at all dose levels and in female mice at dose levels of 5 and 50 mg/kg/day, timolol demonstrated no carcinogenic effect. There was a slight increase in the incidence of mammary adenocarcinomas in female mice that received 500 mg/kg/day (about 500 times the maximum recommended human oral dose on a mg/kg basis). Timolol caused dose-related elevations of serum prolactin in female mice at doses of 100 mg/kg or more, but only very slight transient elevations were found in male mice at doses of 500 mg/kg. Since numerous studies have demonstrated that drugs which cause elevations of serum prolactin are associated with mammary tumors in rodents, the mammary tumors in the female mice in the highest dosage group of this study were considered to have resulted from an increased serum prolactin. In humans, no such association between serum prolactin and mammary carcinoma has been established. Furthermore, in adult human female subjects who received oral dosages of up to 60 mg of timolol, the maximum recommended human oral dosage, there were no clinically meaningful changes in serum prolactin.
ADVERSE REACTIONS: TIMOPTIC Ophthalmic Solution is usually well tolerated. The following adverse reactions have been reported either in clinical trials of up to 3 years duration prior to release in 1978 or since the drug has been marketed.

Signs and symptoms of ocular irritation, including conjunctivitis, blepharitis, and keratitis, have been reported occasionally.

Hypersensitivity reactions, including localized and generalized rash, and urticaria have been reported rarely.

Visual disturbances including refractive changes (due to withdrawal of miotic therapy in some cases) have been infrequently associated with therapy with TIMOPTIC. Rarely, aphakic cystoid macular edema has been reported, but a causal relationship to therapy with TIMOPTIC has not been established.

Aggravation or precipitation of certain cardiovascular and pulmonary disorders has been reported, presumably related to effects of systemic beta blockade (see PRECAUTIONS). These include bradyarrhythmia, hypotension, syncope, and bronchospasm (predominantly in patients with pre-existing bronchospastic disease). In clinical trials, slight reduction of the resting heart rate in some patients (mean reduction 2.9 beats/minute, standard deviation 10.2) has been observed.

The following adverse effects have been reported rarely, and a causal relationship to therapy with TIMOPTIC has not been established: headache, anorexia, dyspepsia, nausea, dizziness, CNS effects (fatigue, confusion, depression, somnolence, and anxiety), palpitation, and hypertension.
HOW SUPPLIED: TIMOPTIC Ophthalmic Solution, 0.25% and TIMOPTIC Ophthalmic Solution, 0.5%. Both are available in 5-ml and 10-ml plastic OCUMETER® Ophthalmic Dispensers with a controlled drop tip.
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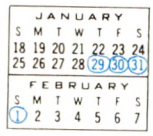
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PRACTICE SEMINAR
(9:00 a.m. - 12:00 noon Thursday)

Scientific Program begins at 2:00 p.m. Thursday

Thursday Afternoon (January 29)

Symposium: EXTENDED WEAR OF CONTACT LENSES IN APHAKIA ..... Jack Hartstein, M.D., *Moderator*
Patient Selection ..... Joseph A. Baldone, M.D.
Cooper Lenses ..... Richard G. Lembach, M.D.
Hydrocurve Lenses ..... Perry S. Binder, M.D.
Sauflon Lenses ..... Anthony B. Nesburn, M.D.
Silicone Lenses ..... Robert T. Blackhurst, M.D.
CSI Lenses ..... H. Dwight Cavanagh, M.D.
Complications of Extended Wear Lenses ..... G. Peter Halberg, M.D.
Panel Discussion

THE USE OF EXTENDED WEAR LENSES IN MYOPIA ..... Louis A. Wilson, M.D.
Symposium: THE USE OF EXTENDED WEAR LENSES FOR THERAPEUTIC PURPOSES .....
..... Richard H. Keates, M.D., *Moderator*
Cooper Lenses ..... Richard G. Lembach, M.D.
Bausch & Lomb Lenses ..... John Brodrick, M.D.
Sauflon Lenses ..... Bruce Bodner, M.D.
Softsight Therapeutic Lenses ..... Henry Gelender, M.D.
PRACTICAL WORKSHOPS ..... *Topics to be announced*

Friday Morning (January 30)

Symposium: TORIC SOFT LENSES ..... Barry A. Maltzman, M.D., *Moderator*
Lens Availability and Design ..... Fitting Techniques and Wear Expectations (Dura-Soft—Hydrocurve)
Fitting Techniques and Wearing Expectations (Hydromore–Miracon–Hydron)...Pitfalls to Avoid in Fitting Toric Lenses
Panel Discussion - Questions and Answers. ***Participants:*** Herschell H. Boyd, M.D., Paul R. Honan, Jr., M.D., Bernard J. Slatt, M.D., Harold A. Stein, M.D.
Symposium: HYGIENIC CARE OF SOFT LENSES ..... Ellis Gruber, M.D., *Moderator*
The Role of Saline and Enzymatic Cleaners in the Care of Soft Lenses ..... Mark B. Abelson, M.D.
The Role of Surfactant Cleaners in the Care of Soft Lenses ..... R. Linsy Farris, M.D.
Hot or Cold Care Systems? ..... Ellis Gruber, M.D.
OPHTHALMIC DEVICES: A Review of Three Years of FDA Involvement ..... Max Talbott, Ph.D.
Symposium: CONTACT LENSES IN INFANTS AND CHILDREN ..... Harold P. Koller, M.D., *Moderator*
Indications and Available Materials for Contact Lenses in Infants and Children ..... Judith E. Gurland, M.D.
Pathophysiology of Contact Lens Therapy in Infants and Children ..... I. Matthew Rabinowicz, M.D.
Contact Lens Fitting Techniques Unique to the Pediatric Population ..... Harold P. Koller, M.D.
Indications and Complications of Intraocular Lenses vs. Contact Lenses in Infants and Children .....
..... Jerry W. Maida, M.D.
Results of Extended and Daily Wear Contact Lenses in Infants and Children ..... Harold P. Koller, M.D.
DISTINGUISHED VISITING LECTURER ..... **Thomas F. Spring, M.D.**

Spouse Luncheon at Don the Beachcomber Compliments of CLAO

Friday Afternoon (January 30)

CONTACT LENS COURSES - Section I ..... R. Linsy Farris, M.D., *Director, Course Program*
1. Current Concepts in the Correction of Myopia—Extended Wear Lens, Orthokeratology and Refractive Corneal Surgery ..... Perry S. Binder, M.D.
2. Management of Corneal & Contact Lens Problems ..... G. Peter Halberg, M.D., Robert A. D'Amico, M.D.

3. How to Select the Best Soft Lens for Your Patient in 1981 ..... Ellis Gruber, M.D.
4. Astigmatism Correction with Hard & Soft Contact Lenses ..... Paul R. Honan, M.D., Herschell H. Boyd, M.D.
5. Specialized Techniques of Lens Fitting ..... Joseph W. Soper
6. Hard Lens Modification—Basic Lens Evaluation and Modification Techniques .....Paul R. Honan, Jr., M.D., Marvin A. Garland, M.D.
7. Fitting of CAB and Silicone Lenses for Daily and Extended Wear .. Jay I. Lippman, M.D., Daniel Sigband, M.D.
8. Contact Lens Fitting in Infants & Children ..... Harold P. Koller, M.D.
9. Introduction to Refractive Keratoplasty ..... Miles H. Friedlander, M.D., Herbert E. Kaufman, M.D.
10. Contact Lens—Lacrimal System Interaction ..... David Lamberts, M.D., Frank J. Holly, Ph.D.
11. Extended Wear of Contact Lenses ..... Rao N. Gullapalli, M.D.
12. Contact Lens Equipment and Solutions ..... Ronald H. Akashi, M.D.

INTRAOCULAR LENS COURSES—Section I ..... Herve M. Byron, M.D., *Director, Course Program*

13. Intracapsular Extractions with Implants—I ..... Stephen Obstbaum, M.D., Marvin Kwitko, M.D.
14. Extracapsular Extractions with Implants—I ..... William Harris, M.D., Robert F. Azar, M.D.
15. Techniques for the Advance Implant Surgeon—I ..... Richard P. Kratz, M.D., Charles Kelman, M.D.
16. Special Instrumentation & Therapeutic Agents ..... Buol K. Heslin, M.D., David J. McIntyre, M.D.

CONTACT LENS COURSES—Section II ..... R. Linsy Farris, M.D., *Director, Course Program*

1. Extended Contact Lens Wear ..... Anthony B. Nesburn, M.D.
2. Update on Therapeutic Contact Lenses ..... Edward L. Shaw, M.D.
3. Keratoconus ..... Frank B. Hoefle, M.D.
4. Soft Lens Techniques ..... Joseph W. Soper
5. Bifocal & Multifocal Contact Lenses for the Presbyope and Aphake ..... Frank J. Weinstock, M.D., Abraham Schlossman, M.D.
6. The Beginning Training & Utilization of a Contact Lens Technician ..... Robert J. Crossen, M.D.
7. Starting from Scratch—Contact Lens Fitting ..... Paul R. Honan, M.D., Herschell H. Boyd, M.D.
8. Hard Lens Modification—Advanced Techniques ..... Marvin A. Garland, M.D., Paul R. Honan, M.D.
9. Soft Toric Lenses ..... Barry A. Maltzman, M.D.
10. Fitting Contact Lenses by Piggy Back Technique ..... Joseph A. Baldone, M.D.
11. Contact Lens Fitting in Difficult Cases ..... Donald J. Doughman, M.D.
12. Ocular Allergy Problems in Contact Lens Wearers..... Mark B. Abelson, M.D., Ira J. Udell, M.D.

INTRAOCULAR LENS COURSES—Section II ..... Herve M. Byron, M.D., *Director, Course Program*

13. Intracapsular Extraction with Implants—II ..... Stephen Obstbaum, M.D., Marvin Kwitko, M.D.
14. Extracapsular Extraction with Implants—II ..... William Harris, M.D., Robert F. Azar, M.D.
15. Techniques for the Advanced Implant Surgeon—II ..... Richard Kratz, M.D., Charles Kelman, M.D.
16. Keratorefractive Surgery ..... James V. Aquavella, M.D., Dennis D. Shepard, M.D.

Wine and Cheese Tasting Party Compliments of Barnes-Hind Pharmaceuticals, Inc.

Saturday Morning (January 31)

Symposium: INTERACTION OF THE CORNEAL EPITHELIUM, TEAR FILM, AND THE CONTACT LENS ..... James V. Aquavella, M.D., *Moderator*

Stimulation and Tear Flow ..... Jules L. Baum, M.D.
The Contact Lens Surface ..... Rao N. Gullapalli, M.D.
Immunological Aspects ..... Mark B. Abelson, M.D.
Tear Film ph ..... Michael A. Lemp, M.D.
Tear Film Tonicity ..... R. Linsy Farris, M.D.
Summary and Discussion ..... James V. Aquavella, M.D.

Symposium: PROTECTION OF ENDOTHELIUM DURING IOL SURGERY .. Herbert E. Kaufman, M.D., *Moderator*

OVERVIEW—Problem of Cell Loss During Surgery ..... Herbert E. Kaufman, M.D.
Quantitation of Cell Loss and Its Prevention by Altering Surgical Technique ..... William M. Bourne, M.D.
The Use of Viscose Agents to Minimize Surgical Cell Loss ..... David Miller, M.D.
Lens Coatings and Other Non-Viscose Type Approaches to the Prevention of Cell Loss . Herbert E. Kaufman, M.D.

ISSUES FACING OPHTHALMOLOGY ..... George Edward Garcia, M.D., *President American Association of Ophthalmology*

CONRAD BERENS LECTURER ..... **John A. Dyer, M.D.**
OPHTHALMIC TECHNICIANS COURSE ..... Topics to be announced

Saturday Afternoon (January 31)

THEODORE E. OBRIG MEMORIAL LECTURER ..... To Be Announced
Symposium: WHAT'S NEW AND INTERESTING IN LENS IMPLANTATION ..... Harold A. Stein, M.D., *Moderator*

Patient Selection—Extended Wear of IOL? ..... Harold A. Stein, M.D.
Instrumentation in Surgery ..... Aziz J. Anis, M.D., F.A.C.S.
Newer Intraocular Lens Designs ..... Ronald W. Barnet, M.D.
Sutures for Cataracts, Implant Surgery and Astigmatism Control ..... J. Elliott Blaydes, Jr., M.D.
Statistics on Implant Results ..... James V. Aquavella, M.D.
Some Mechanical Considerations ..... C. William Simcoe, M.D.
Preventing Complications with IOL's ..... Stanley C. Becker, M.D.
Managing Complications ..... Richard P. Kratz, M.D.

Symposium: IS EXTRACAPSULAR CATARACT SURGERY WORTH IT? .... C. William Simcoe, M.D., *Moderator*
Three panelists (Richard P. Kratz, M.D., Richard H. Keates, M.D., and K. Buol Heslin, M.D.) will each give a 10 minute presentation of their personal experiences regarding: Prognosis for a Successful Repair of Retinal Detachment; Cystoid Macular Edema; Quality of Vision. A Panel Discussion and Questions from the Audience will follow.

CATARACT SURGICAL TECHNIQUES (Movies) ..... Robert F. Azar, M.D., *Moderator*
Recent Advances in Cataract Surgery ..... Leeds E. Katzen, M.D.
Cataract Surgery in Infants and Juveniles ..... J. Elliott Blaydes, M.D.
Extracapsular Technique (Manual) ..... Charles W. Simcoe, M.D.
Phacoemulsification Technique ..... Robert M. Sinskey, M.D.
Extracapuslar Technique (Mechanical) ..... William S. Harris, M.D.
Intracapsular Technique ..... Marvin L. Kwitko, M.D.

International Party Sponsored by Alcon/BP

Sunday Morning (February 1)

SURGICAL CORRECTION OF REFRACTIVE ERRORS
Jack Hartstein, M.D., *Chairman*

Symposium: SURGICAL CORRECTION OF ASTIGMATISM ..... Richard P. Kratz, M.D., *Moderator*
Prevention: Cataract Wound Closure ..... Clifford Terry, M.D.
Comparison of Keratometers ..... Dennis Shepard, M.D.
Correction: Reconstruction of Previous Incision .....Thomas Cravy, M.D.
Corneal Tuck ..... Robert F. Azar, M.D.

Symposium: SURGICAL CORRECTION OF MYOPIA—PRO's AND CON's ..... Walter J. Stark, M.D., *Moderator*
History of Surgical Correction of Myopia and Early Clinical Results ..... Leeds Katzen, M.D.
Surgical Correction of Myopia—Studies in Laboratory Animals and Human Cadaver Eyes ..... Ronald Smith, M.D.
Complications of Radial Keratotomy ..... J. James Rowsey, M.D.
Update of Clinical Experience with Surgical Correction of Myopia ..... Leo D. Bores, M.D.
A Prospective, Multicenter, Clinical Trial of Radial Keratotomy for Myopia ..... George Waring, M.D.
Summary and Conclusions ..... Walter J. Stark, M.D.

Symposium: SURGICAL CORRECTION OF APHAKIA ..... Perry S. Binder, M.D., *Moderator*
Keratophakia and Hypermetropic Keratomileusis ..... Casimir A. Swinger, M.D.
Hypermetropic Keratomileusis ..... Jorge Krumeich, M.D.
Keratophakia & Hyperopia Keratomileusis Using Precarved Frozen Material ..... Miles H. Friedlander, M.D.
Epikeratophakia ..... Herbert E. Kaufman, M.D.
Panel Discussion—Summary and Conclusions

Contact & Intraocular Lens Presentations—Thursday afternoon, Friday morning and Saturday morning

PRACTICE SEMINAR
Thursday, January 29, 1981 8:50 a.m. - 12:45 p.m.
Herschell H. Boyd, M.D., *Chairman*

Symposium: Time to Buy a Computer? Phillip C. Hessburg, M.D., *Moderator*
Participants: Richard H. Keates, M.D., Phillip C. Hessburg, M.D. *Questions from Audience*

Symposium: Office Management of Extended Wear Lenses Herve M. Byron, M.D., *Moderator*

Optical Dispensing by the Ophthalmologist? ..... R. Hugh Minor, M.D.
Office Surgery for Cataracts ..... David P. Morrison, M.D.
Office Surgery for Cataracts ..... Douglas E. Williamson, M.D.
Merits of a Subjective Refractor ..... Robert F. Azar, M.D.
Merits of a Subjective Refractor ..... Herschell H. Boyd, M.D.
Oil and Gas as a Diversion ..... Charles R. Moore, M.D.
Stop Your P.S. Corporation! ..... Paul R. Honan, Jr., M.D.
Security for the 80's ..... Herschell H. Boyd, M.D.

Questions from Audience

See next page for Ophthalmic Technicians Program, Special Notes and Registration Form

OPHTHALMIC TECHNICIAN PROGRAM
Saturday, January 31, 1981

Update of Contact Lenses
for Ophthalmic Assistants, Contact Lens Technicians and Residents

Director: Harold A. Stein, M.D. **Program Coordinator:** Penny Cook, F.O.C.L.A.
Faculty: L. Dean Clements, Doris Gaston, C.O.T., Ellis Gruber, M.D., Keith Harrison, F.O.C.L.A., Maija Kaufman, R.N., C.O.T., John Morgan, M.D., Bernard J. Slatt, M.D., Kenneth Swanson, C.O.T.

Instructional Objectives:
This course is designed for the resident, ophthalmic assistant and contact lens technician who has some experience in contact lens fitting. It is designed to review existing material available in the contact lens field and bring new material concerning current items of interest in the contact lens world.

Topics: Physiology Related to Contact . . . New Thrusts in Cornea and Tear Research . . . Clinical Selection of Patients . . . Practical Tips on Dispensing Contact Lenses . . . Office Cleaning of Soft Lenses . . . Staining Patterns with Hard Lenses . . . Selecting the Correct Lens . . . Toric Soft Lenses . . . Systems for Fitting Hard Lenses . . . Silicone Lenses . . . Newer Lens Materials and Designs . . . Gas Permeable Lenses . . . Extended Wear Lenses . . . Therapeutic and Coloured Lenses . . . Problems with Soft and Hard Contacts

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An optional Practice Management Seminar will be conducted on Thursday, January 29, 1981 immediately preceding the scientific session. There is no registration fee for the Practice Management Seminar; the registration fee for the Scientific Session is the total fee for all sessions.

Registration for the Scientific Session will take place on Wednesday, January 28 from 12:00 noon to 5:00 p.m. and Thursday, January 29 from 7:30 a.m. to 5:30 p.m. Use the registration form below to pre-register and save time at the meeting.

Special One Day Registration—Anyone who wishes to attend the meeting for only one day may do so for the nominal registration fee of $50.00 which includes participation in that day's sessions and the evening function.

Refund Policy. Full refund if cancellation is received by January 1, 1981. A $25.00 service charge per person for cancellations from January 2, 1981 to January 28, 1981. No refunds for cancellations received after January 29, 1981.

Spouse Luncheon (Friday)—Compliments of the Contact Lens Association of Ophthalmologists, Inc.

A special room rate has been arranged with the Sahara Hotel. For room reservations only, write to the Sahara Hotel, Las Vegas, Nevada 89114.

PRE-REGISTRATION FORM—CLAO MID-WINTER NATIONAL MEETING
January 29 - February 1, 1981 Sahara Hotel Las Vegas, Nevada

Name _____ M.D. _____ Other _____

Address _____

City, State, Zip Code _____ Telephone _____

☐ Physician Member of CLAO—US$100.00 ☐ Physician Non-Member—US$250.00

☐ Resident Physician in Ophthalmology—US$50.00

☐ Technician* employed by Ophthalmologist (Physician Member of CLAO). Must be identified by letter by the employing ophthalmologist—US$75.00

☐ Technician* employed by Ophthalmologist (Non-Member of CLAO). Must be identified by letter by the employing ophthalmologist—US$175.00

☐ ALL OTHERS—US$275.00 ☐ ONE DAY REGISTRATION—US$50.00 Please specify day: _____

Please make check payable to: CLAO—Mid Winter Meeting and forward to R. Linsy Farris, M.D.
Treasurer—CLAO
2620 Jena Street
New Orleans, LA 70115

*Technicians. This fee includes the entire meeting (3-1/2 days), in addition to the Ophthalmic Assistants and Technicians Course.

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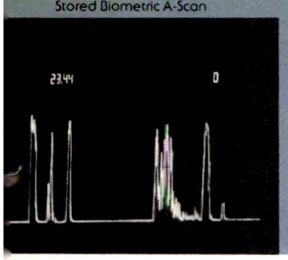
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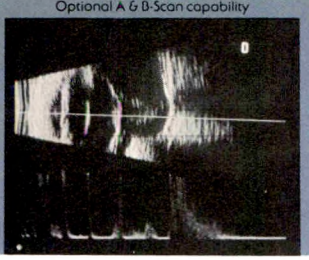


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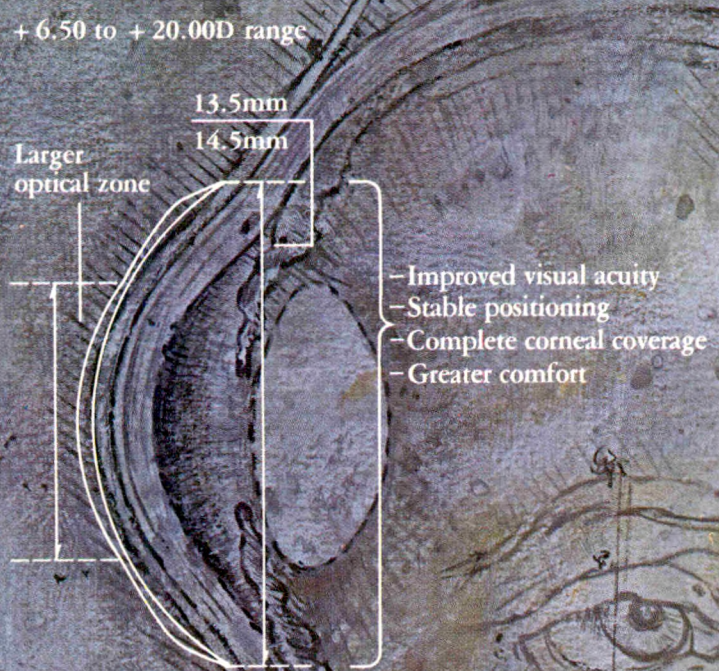
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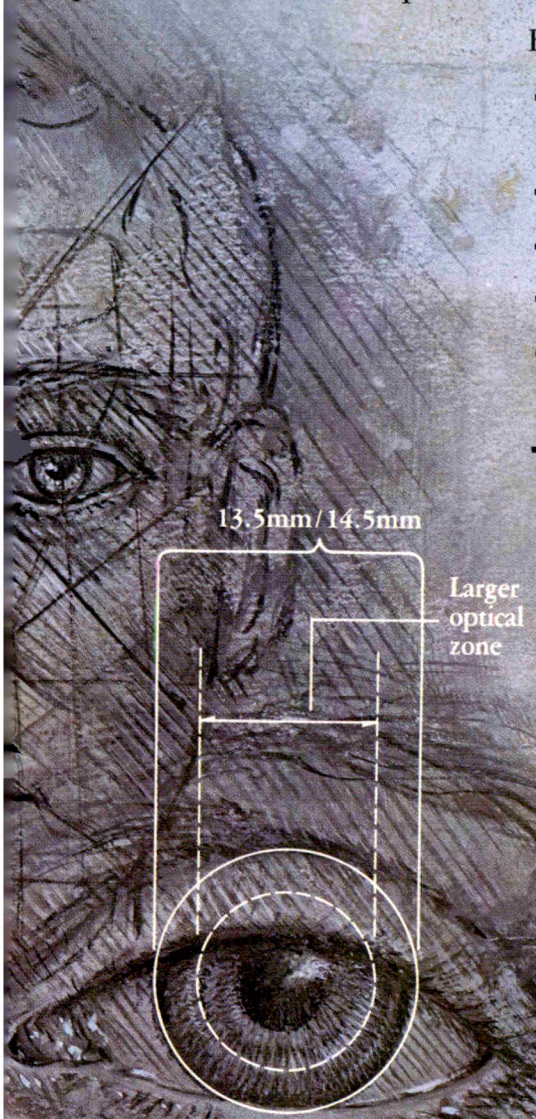
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See next page for brief summary of prescribing information.

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BAUSCH & LOMB SOFLENS® (polymacon) Contact Lenses

BRIEF SUMMARY (For full prescribing information see package insert.)
VISION CORRECTION USE

INDICATIONS: SOFLENS Contact Lenses are indicated for vision correction use in persons with non-diseased eyes and in aphakia. **CONTRAINDICATIONS:** SOFLENS Contact Lenses are contraindicated in the presence of any active untreated infection or abnormality of the anterior segment of the eye. **WARNINGS: Abrasions and Infections**—If a lens becomes less comfortable than it was when first placed on the wearer's cornea, the lens should be removed immediately and the wearer's eye and the lens examined for the possible presence of a foreign body. If any eye abrasion, ulceration, irritation or infection is present or any abnormal eye condition is observed concurrent with lens wear, the lens should be removed immediately and a physician consulted. **Aphakic Patients:** Aphakic patients should not be fitted with SOFLENS Contact Lenses during the postoperative period until, in the opinion of the surgeon, the eye has healed completely. **Lens Care and Handling:** Persons who require only vision correction and who would not, or could not adhere to the recommended daily care of SOFLENS Contact Lenses or who are unable to place and remove the lenses should not be provided with them. Failure to follow handling and cleaning instructions could lead to serious eye infections which might result in corneal ulcers. Malfunction and rusting of the metal interior of the BAUSCH & LOMB™ Disinfecting Unit I (ASEPTOR®) as well as discoloration and cracking of the lens case has been reported after varying periods of use. If such occurs, appropriate replacement is indicated to avoid interference with the disinfection procedure. Keep the BAUSCH & LOMB™ Disinfecting Unit I (ASEPTOR®) and II (ASEPTRON™) clean and dry at all times. **Medicants and Eye Drops:** When the lenses are used by persons requiring only vision correction no ophthalmic solutions or medicants should be used. No conventional (hard) contact lens solutions and eye drops, except BAUSCH & LOMB™ Lens Lubricant should be used by SOFLENS Contact Lens wearers prior to placement, or while the lens is in place, on the eye. Also, no solutions, including conventional (hard) contact lens solutions should be used. Only daily cleaner, lens lubricant, saline and the solution made from the SOFLENS™ Enzymatic Contact Lens Cleaning Tablets are to be used on a SOFLENS Contact Lens when the lens is off the eye. **Wearing Restrictions:** SOFLENS Contact Lenses when used only for vision correction should be removed before sleeping or swimming and in the presence of noxious and irritating vapors. Insufficient lacrimal secretions or hypoesthesia may be restricting factors to the wearing of soft contact lenses. **PRECAUTIONS: Storage**—SOFLENS Contact Lenses must be stored ONLY in saline solution or BAUSCH & LOMB™ Sterile Disinfecting Solution. If left exposed to air, the lenses will dehydrate, become brittle, and break readily. If a lens dehydrates, it should be resoaked in saline solution or BAUSCH & LOMB™ Sterile Disinfecting Solution until it returns to its soft, pliable state which may take as long as forty minutes. Saline solution must be used DAILY for cleaning, storing, and disinfection of lenses. The carrying case must be emptied and refilled with fresh saline solution just before disinfecting the lenses. **Lens Handling:** Hands must be washed, rinsed thoroughly, and dried with a lint-free towel before handling the lenses. Cosmetics, lotions, soaps, and creams must not come in contact with the lenses since eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until the hair spray has settled. A SOFLENS Contact Lens may be damaged by nicking or tearing if care is not exercised during placement on or removal from the eye, replacing or removing it from the carrying case or in the cleaning process. **Disinfecting:** Saline solution must be used DAILY for cleaning, storing, and disinfection of lenses. After removal from the eye, the SOFLENS Contact Lens must be rubbed gently with Daily Cleaner then rinsed with saline solution to remove mucus and film from the lens surface. The BAUSCH & LOMB™ Lens Carrying Case (LENSGARD™) must be emptied and refilled ⅔ full with fresh saline solution just before disinfecting the lenses and must always be tightly closed to prevent leakage of saline from the case and subsequent dehydration of the lenses. In order to remove protein deposits which may form on the lenses, wearers should use the SOFLENS™ Enzymatic Contact Lens Cleaning Tablets according to the directions for use which accompany the tablets. To help remove tear residues and mucus as well as other deposits that tend to accumulate on the lens surface, patients should use Daily Cleaner according to the directions for use which accompany the product. Disinfecting with a BAUSCH & LOMB™ Disinfecting Unit I (ASEPTOR®) or II (ASEPTRON™) is necessary to kill microorganisms. If a BAUSCH & LOMB™ Disinfecting Unit I (ASEPTOR®) or II (ASEPTRON™) is not available, lenses may be disinfected by immersing your lenses in BAUSCH & LOMB™ Saline Solution in the carrying case and dropping the sealed case into a pan of already boiling water. Remove the pan from heat after ten minutes and allow the water to cool before removing your lenses. When this boiling method of disinfection is used, the lenses can be damaged if the boiling water is allowed to completely evaporate. Chemical disinfection with BAUSCH & LOMB™ Sterile Disinfecting Solution has been tested microbiologically and shown to be an effective disinfection procedure for the SOFLENS hydrophilic contact lens. If chemical disinfection is used the SOFLENS hydrophilic contact lens must be cleaned and rinsed daily with BAUSCH & LOMB™ Sterile Daily Cleaner and BAUSCH & LOMB™ Sterile Disinfecting Solution. The lens carrying case must be emptied and refilled with fresh BAUSCH & LOMB™ Sterile Disinfecting Solution prior to disinfecting the lens. Fresh BAUSCH & LOMB™ Sterile Disinfecting Solution must be used daily for storing and disinfecting the lens. **WARNING:** BAUSCH & LOMB™ STERILE DISINFECTING SOLUTION SHOULD NOT BE USED WITH HEAT. **Fluorescein:** Never use fluorescein while the patient is wearing the lenses because the lenses will become discolored. Whenever fluorescein is used, flush the eyes with sterile normal saline solution and wait at least one hour before replacing the lenses. Earlier replacement may cause the lenses to absorb residual fluorescein. **ADVERSE REACTIONS:** Serious corneal damage may result from wearing a SOFLENS Contact Lens which has been soaked in a conventional contact lens solution. Eye irritation may occur within a short time after putting on a hypertonic lens. Removal of the lens will relieve the irritation. A lens adheres very rarely to an eye as a result of sleeping with the lens on, or wearing a hypotonic lens. If a lens adheres for any reason, apply fresh saline and wait until the lens moves freely before removing it. Clinical studies indicate that corneal edema, as manifested by symptoms such as rainbows, halos around lights, or foggy vision, may occur in less than 5% of SOFLENS Contact Lens wearers. If these symptoms occur, the lenses should be removed and professional consultation obtained. Excessive tearing, unusual eye secretions, and photophobia are not normal. If these symptoms occur, the patient should be examined to determine their cause. Neovascularization of the cornea has been observed in some aphakic patients fitted with the SOFLENS Contact Lens, which may require discontinuation of the lens. Medical consultation should be obtained in such an instance.

4/80

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SOFLENS DIVISION
Rochester, New York 14602

THIRD ANNUAL WINTER SEMINAR of the MIAMI OPHTHALMOLOGICAL SOCIETY
January 11-18, 1981
Wildwood Inn, Snowmass at Aspen
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30 Hours of Category I CME credit

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Henry Hirschman, M.D.
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THE POSTGRADUATE INSTITUTE OF THE NEW YORK EYE AND EAR INFIRMARY ANNOUNCES THE SIXTEENTH ANNUAL REVIEW COURSE "BASIC SUBJECTS IN OPHTHALMOLOGY"

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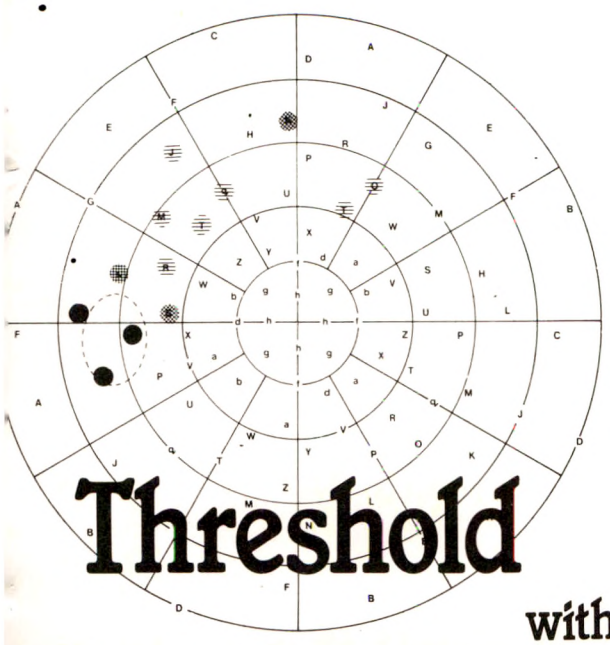
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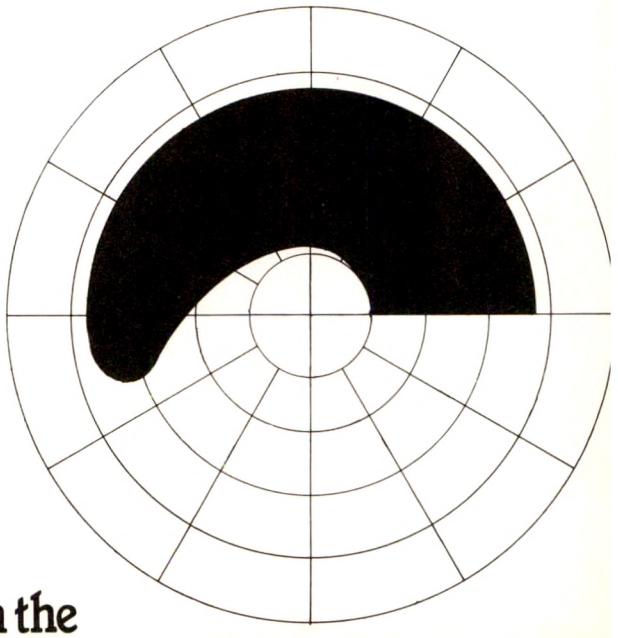
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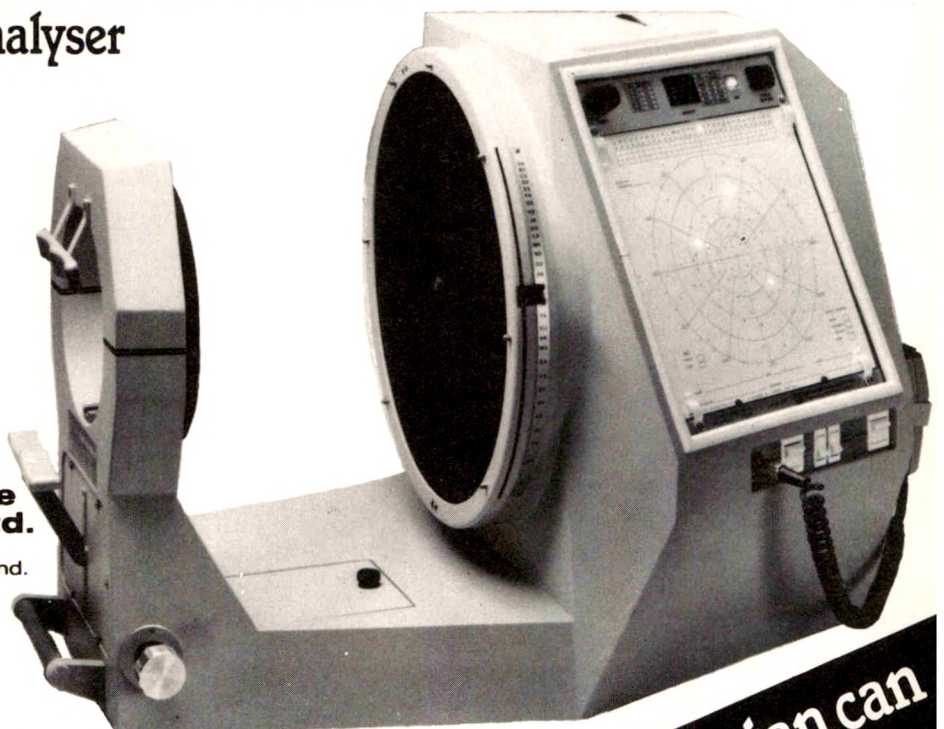


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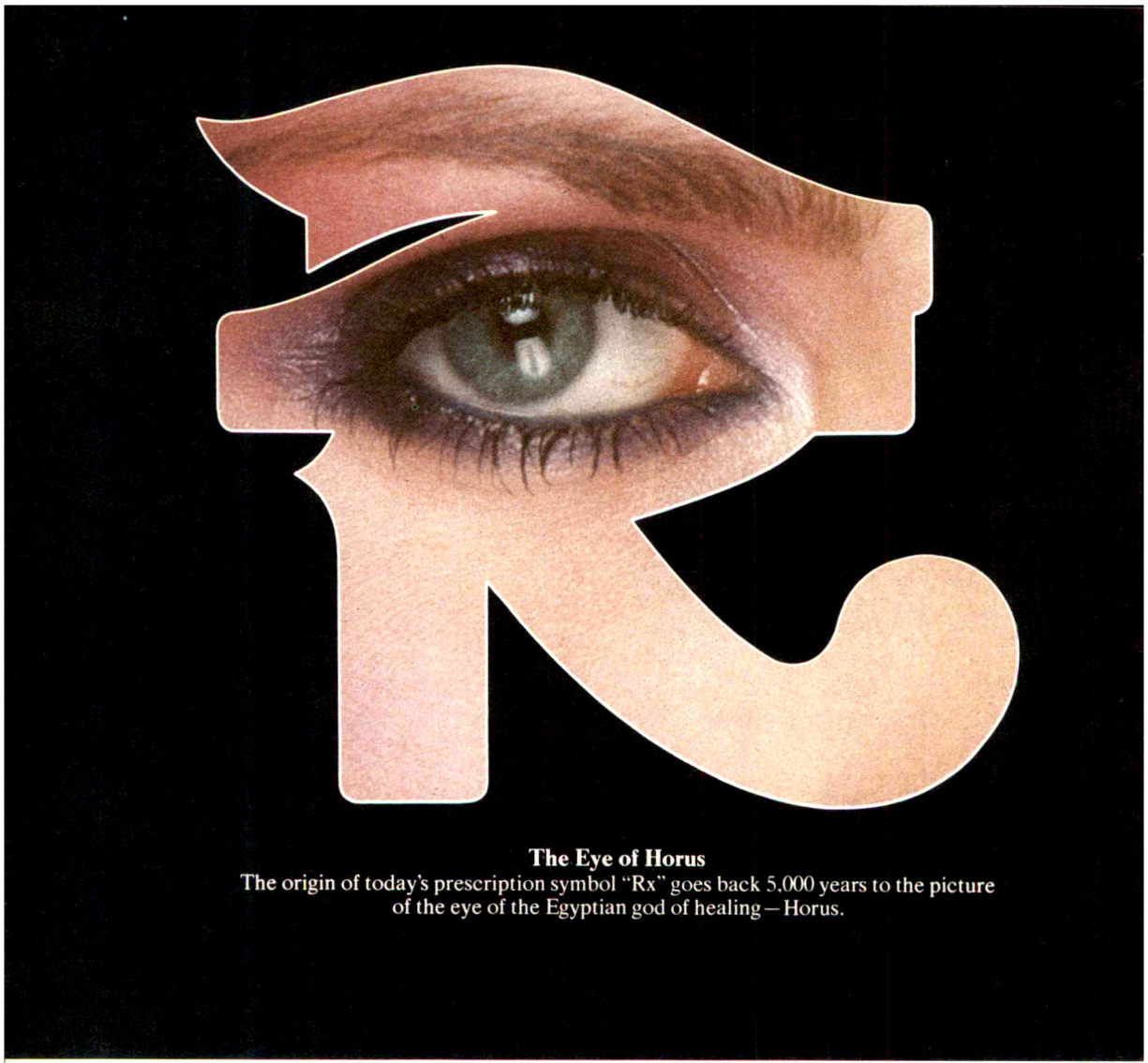
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The Eye of Horus
The origin of today's prescription symbol "Rx" goes back 5,000 years to the picture of the eye of the Egyptian god of healing—Horus.

DESCRIPTION: VIROPTIC® is the brand name for trifluridine (also known as trifluorothymidine, F_3TdR,F_3T), an antiviral drug for topical treatment of epithelial keratitis caused by Herpes simplex virus. The chemical name of trifluridine is 5-trifluoromethyl-2′-deoxyuridine.

VIROPTIC sterile opthalmic solution contains 1% trifluridine in an aqueous solution with acetic acid and sodium acetate (buffers), sodium chloride, and thimerosal 0.001% (added as a preservative).

CLINICAL PHARMACOLOGY: Trifluridine is a fluorinated pyrimidine nucleoside with *in vitro* and *in vivo* activity against Herpes simplex virus, types 1 and 2 and vacciniavirus. Some strains of Adenovirus are also inhibited *in vitro*.

Intraocular penetration of trifluridine occurs after topical instillation of VIROPTIC into human eyes. Decreased corneal integrity or stromal or uveal inflammation may enhance the penetration of trifluridine into the aqueous humor. Unlike the results of ocular penetration of trifluridine *in vitro*, 5-carboxy-2′-deoxyuridine was not found in detectable concentrations within the aqueous humor of the human eye.

INDICATIONS AND USAGE: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% is indicated for the treatment of primary keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus, types 1 and 2. VIROPTIC is also effective in the treatment of epithelial keratitis that has not responded clinically to the topical administration of idoxuridine or when ocular toxicity or hypersensitivity to idoxuridine has occurred. In a smaller number of patients found to be resistant to topical vidarabine, VIROPTIC was also effective.

The clinical efficacy of VIROPTIC in the treatment of stromal keratitis and uveitis due to Herpes simplex or ophthalmic infections caused by vacciniavirus and Adenovirus has not been established by well-controlled clinical trials. VIROPTIC has not been shown to be effective in the prophylaxis of Herpes simplex virus keratoconjunctivitis and epithelial keratitis by well-controlled clinical trials. VIROPTIC is not effective against bacterial, fungal or chlamydial infections of the cornea or nonviral trophic lesions.

During controlled multicenter clinical trials, 92 of 97 (95%) patients (78 of 81 with dendritic and 14 of 16 with geographic ulcers) responded to VIROPTIC therapy as evidenced by complete corneal re-epithelialization within the 14 day therapy period. In these controlled studies, 56 of 75 (75%) patients (49 of 58 with dendritic and 7 of 17 with geographic ulcers) responded to idoxuridine therapy. The mean time to corneal re-epithelialization for dendritic ulcers (6 days) and geographic ulcers (7 days) was similar for both therapies. In other clinical studies, VIROPTIC was evaluated in the treatment of Herpes simplex virus keratitis in patients who were unresponsive or intolerant to the topical administration of idoxuridine or vidarabine. VIROPTIC was effective in 138 of 150 (92%) patients (109 of 114 with dendritic and 29 of 36 with geographic ulcers) as evidenced by corneal re-epithelialization. The mean time to corneal re-epithelialization was 6 days for patients with dendritic ulcers and 12 days for patients with geographic ulcers.

CONTRAINDICATIONS: VIROPTIC brand Trifluridine Ophthalmic Solution, 1%, is contraindicated for patients who develop hypersensitivity reactions or chemical intolerance to trifluridine.

WARNINGS: The recommended dosage and frequency of administration should not be exceeded (see Dosage and Administration).

PRECAUTIONS:

General: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% should be prescribed only for patients who have a clinical diagnosis of herpetic keratitis.

VIROPTIC may cause mild local irritation of the conjunctiva and cornea when instilled but these effects are usually transient.

Although documented *in vitro* viral resistance to trifluridine has not been reported following multiple exposure to VIROPTIC, the possibility exists of viral resistance development.

For Topical Treatment of
Epithelial Keratitis Caused by Herpes Simplex Virus

VIROPTIC®

(Trifluridine)

OPHTHALMIC SOLUTION, 1%

STERILE

95% of Patients Responded*

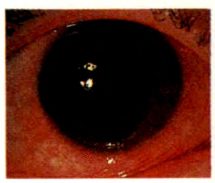
During controlled clinical trials 95% of patients (92 of 97) responded to VIROPTIC therapy. Dendritic and geographic ulcers were re-epithelialized in a mean time of 6 and 7 days, respectively.

Significantly More Convenient

- Less frequent applications
- No middle of the night dosage
- None of the blurring and inconvenience of ointments

5 to 9 Drops Daily

One drop every 2 hours while awake until herpetic lesion has completely re-epithelialized, then one drop every 4 hours for 7 days post-re-epithelialization.



0 days



5 days after VIROPTIC therapy

Effective in 138 of 150 patients* Unresponsive or Intolerant to Idoxuridine or Vidarabine

In studies of patients unresponsive or intolerant to idoxuridine or vidarabine, VIROPTIC demonstrated 92% effectiveness. Dendritic and geographic ulcers were re-epithelialized in a mean time of 6 and 12 days, respectively.

*Data on file, Medical Division, Burroughs Wellcome Co.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Mutagenic Potential. Trifluridine has been shown to exert mutagenic, DNA damaging and cell transforming activities in various standard *in vitro* test systems, and clastogenic activity in *Vicia faba* cells.

Although the significance of these test results is not clear or fully understood, there exists the possibility that mutagenic agents may cause genetic damage in humans.

Oncogenic Potential. The oncogenic potential of trifluridine is unknown at this time. The oncogenic potential of trifluridine in rodents is being evaluated.

Pregnancy: The drug should not be prescribed for pregnant women unless the potential benefits outweigh the potential risks.

Nursing Mothers: It is unlikely that trifluridine is excreted in human milk after ophthalmic instillation of VIROPTIC because of the relatively small dosage (≤ 5.0 mg/day), its dilution in body fluids and its extremely short half-life (approximately 12 minutes). The drug should not be prescribed for nursing mothers unless the potential benefits outweigh the potential risks.

ADVERSE REACTIONS: The most frequent adverse reactions reported during controlled clinical trials were mild, transient burning or stinging upon instillation (4.6%) and palpebral edema (2.8%). Other adverse reactions in decreasing order of reported frequency were superficial punctate keratopathy, epithelial keratopathy, hypersensitivity reaction, stromal edema, irritation, keratitis sicca, hyperemia, and increased intraocular pressure.

OVERDOSAGE: Overdosage by ocular instillation is unlikely because any excess solution be quickly expelled from the conjunctival sac.

Acute overdosage by accidental oral ingestion of VIROPTIC has not occurred. However, should such ingestion occur, the 75 mg dosage of trifluridine in a 7.5 ml bottle of VIROPTIC is not likely to produce adverse effects. Single intravenous doses of 15-30 mg/kg/day in children and adults with neoplastic disease produce reversible bone marrow depression as the only potentially serious toxic effect and only after 3-5 courses of therapy. The acute oral LD_{50} in the mouse and rat was 4379 mg/kg or higher.

DOSAGE AND ADMINISTRATION: Instill one drop of VIROPTIC Ophthalmic Solution, 1% onto the cornea of the affected eye every two hours while awake for a maximum daily dosage of nine drops until the corneal ulcer has completely re-epithelialized. Following re-epithelialization, treatment for an additional seven days of one drop every four hours while awake for a minimum daily dosage of five drops is recommended.

If there are no signs of improvement after seven days of therapy, or complete re-epithelialization has not occurred after 14 days of therapy, other forms of therapy should be considered. Continuous administration of VIROPTIC for periods exceeding 21 days should be avoided because of potential ocular toxicity.

HOW SUPPLIED: VIROPTIC Ophthalmic Solution, 1% is supplied as a sterile ophthalmic solution in a plastic Drop-Dose® dispenser bottle of 7.5 ml. Store under refrigeration 2° to 8° C (36° to 46° F).

The First Antiviral Product From Burroughs Wellcome Co.
Research Triangle Park North Carolina 27709

Wellcome

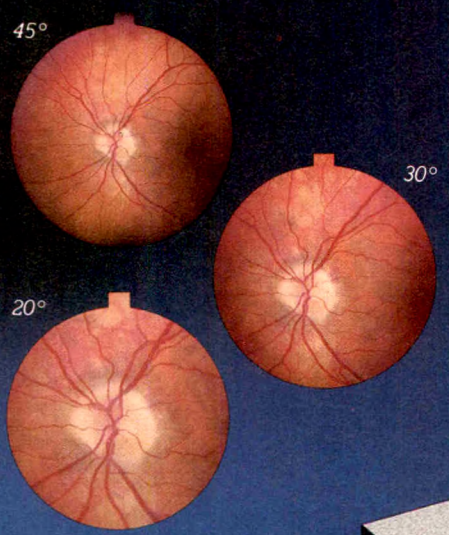
Variable Angle at the Flip of a Switch

The Versatile Topcon TRC-W Retinal Camera

Once again Topcon moves forward the art of fundus photography. With a variable angle camera that lets you instantly switch between three different settings —45,° 30,° and 20° —for maximum operating flexibility.

The TRC-W produces superior fluorescein angiograms of the fundus *or* iris. At the specific magnifications that meet your needs, with correct exposure control maintained throughout the magnification range. Two standard camera bodies are supplied for color documentation or fluorescein. Or choose the optional Polaroid back.

You'll find the TRC-W's price equally accommodating to your needs. Available with 2 or 3 frame per second power supplies; standard or tilt models.





TOPCON

A New World of Precision Optics

OXFORD

Epidemiology and Statistics for the Ophthalmologist

Alfred Sommer, Wilmer Institute, The Johns Hopkins University. A straightforward presentation of epidemiologic and statistical techniques relevant to conducting, interpreting, and assimilating the most common types of clinical research, this text is illustrated with many examples covering such topics as the sensitivity and specificity of glaucoma tests, prevalence of SMCD, and the incidence of visual field loss with different levels of ocular hypertension.

CONTENTS: I. Epidemiology. Rates: Their Meaning and Use; Clinical Studies: Techniques, Common Sense, and a Few More Definitions; The Statistical Interface; Putting It All Together. II. Statistics. Simple Concepts and Cookbook Formulas; Tests of Statistical Significance; Choosing a Sample Size; Confidence Limits; Some Parting Advice.

1980 96 pp.; illus. paper $11.95

World Blindness and its Prevention

Edited by **The International Agency for the Prevention of Blindness** under the direction of **Sir John Wilson.** Most blindness, particularly in developing countries, is curable or avoidable. This book outlines strategies for multidisciplinary efforts, national and regional planning, and the mobilization of resources to prevent and cure the six major types of blinding diseases, which are described.

1980 104 pp. $17.95

A Handbook of Ophthalmology for Developing Countries

Second Edition

Geoffrey G. Bisley, St. John Ophthalmic Hospital, Jerusalem. In addition to scientific and clinical updating, the new edition of this practical handbook features more material on the prevention of eye diseases and new material on mobile eye units and on trachoma. The *British Journal of Ophthalmology* called the first edition "an admirable book. . . almost unique in covering the special requirements of such a practical but limited ophthalmic service."

December 1980 184 pp.; 60 illus. paper $12.95

Introduction to Ophthalmology

John Parr, University of Otago Medical School, Australia. "A medical student or non-ophthalmologist seeking a brief but coherent discussion of ophthalmology can do no better than Parr's *Introduction.*"—*New England Journal of Medicine.* "This new textbook of ophthalmology is particularly welcome . . . . the text is clear and readable, and the copious diagrams are a superb embellishment."—*British Medical Journal.*

1979 224 pp.; 239 illus paper $11.95

Essentials of Ophthalmic Optics

E. W. Allen, Oxford Eye Hospital. Beginning with the relevant theory of light and continuing via the principles of visual optics to the fitting of eyeglasses and contact lenses, this book correlates the technical and professional aspects of ophthalmic optics.

1979 144 pp.; illus. paper $5.95

Optic Neuritis and its Differential Diagnosis

G. D. Perkin and **F. Clifford Rose,** both of Charing Cross Hospital, London. "The best review of a large series of patients with optic neuritis in the current literature."—*Journal of Neurology, Neurosurgery and Psychiatry.* "The whole book is well presented, with excellent summaries at the close of each chapter. It achieves a dual function, both as an outstanding review of the subject, and as a comprehensive reference source."—*Lancet*

1979 304 pp.; 116 illus. $60.00

Prices and publication dates are subject to change.

Oxford University Press
200 Madison Avenue New York, New York 10016



Cortisporin® Ophthalmic Suspension
(Sterile)

For your eyes only!

Cortisporin® Ophthalmic Ointment
(Sterile)



☐ ***Because of your specialized knowledge and training,*** we believe that only you should prescribe the Cortisporin® ophthalmic products—and that's why they are promoted only to ophthalmologists.

☐ ***For your patients*** the Cortisporin ophthalmic products provide a wide therapeutic spectrum. The antibacterials act together against susceptible bacterial pathogens commonly involved in the ophthalmic infections accessible to topical therapy.* The hydrocortisone component gently and rapidly relieves signs and symptoms of inflammation and inhibits corneal neovascularization.

☐ ***Economical, too,*** with the two sterile forms providing convenient day and night coverage at lower cost than most other major brands of similar activity.

Cortisporin® Ophthalmic Suspension Sterile

(Polymyxin B–Neomycin–Hydrocortisone)

Each cc. contains: Aerosporin® brand Polymyxin B Sulfate, 10,000 Units; neomycin sulfate (equivalent to 3.5 mg. neomycin base), 5 mg.; hydrocortisone, 10 mg. (1%); thimerosal (preservative), 0.001%.
The vehicle contains the inactive ingredients cetyl alcohol, glyceryl monostearate, liquid petrolatum, polyoxyl 40 stearate, propylene glycol and purified water.

Cortisporin® Ophthalmic Ointment Sterile

(Polymyxin B–Bacitracin–Neomycin–Hydrocortisone)

Each gram contains: Aerosporin® brand Polymyxin B Sulfate 5,000 Units; zinc bacitracin 400 Units; neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base); hydrocortisone 10 mg (1%); special white petrolatum qs.

***INDICATIONS:** Based on a review of these drugs by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

"Possibly" effective: For the treatment of nonpurulent bacterial infections of the eye due to organisms sensitive to the antibiotic ingredients of the drugs and when the anti-inflammatory action of the hydrocortisone is indicated as in nonpurulent bacterial, allergic, vernal and phlyctenular conjunctivitis; nonpurulent blepharitis; interstitial, sclerosing, postoperative or acne rosacea keratitis; superficial chemical and thermal burns of the cornea.

Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS: These products are contraindicated in acute purulent conjunctivitis and blepharitis; tuberculous, fungal or viral lesions of the skin or eye, including herpes simplex, dendritic keratitis, vaccinia or varicella; and conditions involving the posterior segment of the eye. They are also contraindicated in those individuals who have shown hypersensitivity to any of their components.

PRECAUTIONS: Extended ophthalmic use of topical steroid therapy may result in glaucoma with possible damage of the optic nerves, posterior subcapsular cataract formation, or may aid in the establishment of secondary ocular infections from fungi or viruses liberated from ocular tissues. It is advisable that intraocular pressure be checked frequently. In those diseases causing thinning of the cornea, perforation has been known to occur with the use of topical steroids. As with any antibiotic preparation, prolonged use may result in the overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Articles in the current medical literature indicate an increase in the prevalence of persons allergic to neomycin. The possibility of such a reaction should be borne in mind.

Complete literature available on request from Professional Services Dept. PML.

ONE HUNDRED YEARS WELLCOME

Burroughs Wellcome Co.
Research Triangle Park
North Carolina 27709

Grieshaber



MPC Membrane Peeler Cutter

A significant contribution to ophthalmic surgery.

New Grieshaber MPC Automated Microscissors, from 20-gauge tip to dual-function foot control, are designed to do one job with distinction: Membrane peeling and cutting. The design eliminates the tension movement inevitable in hand-controlled scissors.

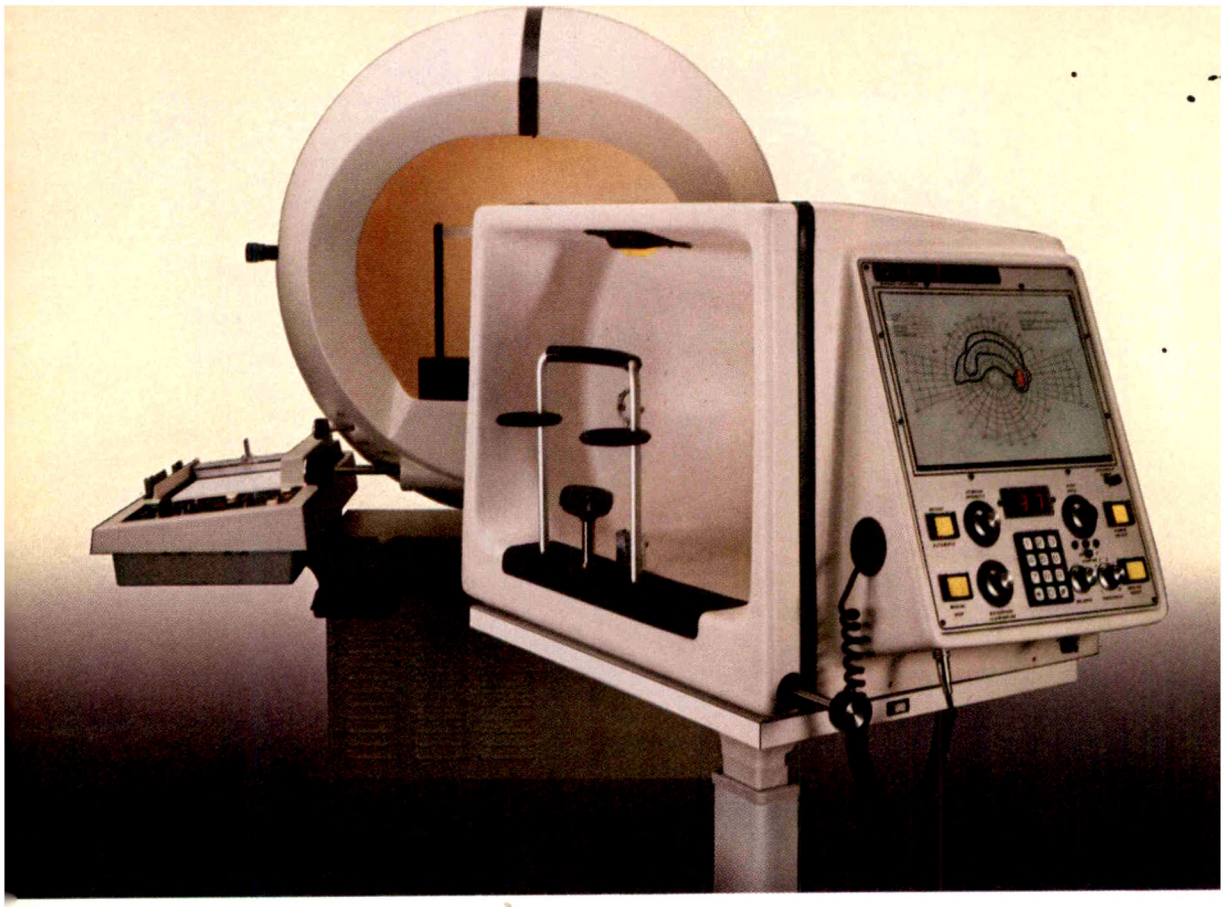
As in all Grieshaber instruments, the proof of quality is in the cutting. The fixed distal blade is specially shaped to peel or position membraneous tissue, while the proximal blade moves to make the cut when the foot control is pressed. Single stroke or variable speed continuous cutting can be selected on the remote control.

Developed by Robert Machemer, M.D., and tested extensively by vitreous surgeons, MPC Automated Microscissors are now available directly from Grieshaber in the U.S. Write or call for our detailed brochure and ordering information.

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No vision care specialist should be without the Coherent Ocuplot Automatic Visual Field Screener. Ease of operation, unsurpassed reliability, and state-of-the-art electronics, make early detection of eye disorders an integral part of your practice. You'll be able to administer this crucial test to more patients—quickly and inexpensively.

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- ☐ POSITIVE DISPLAY OF DEFECTS
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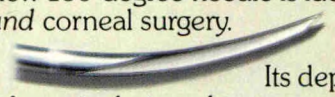
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DAVIS+GECK

THE OPHTHALMIC NEEDLE YOU'LL PREFER TOMORROW YOU'LL FIND ON OUR SUTURES TODAY.

The leading edge in ophthalmic surgery begins with our 160-degree needle...

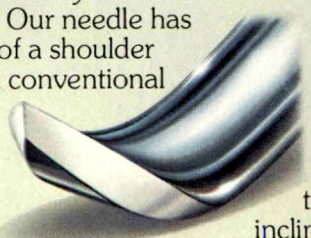
In 6 or 8-wire diameter, the Davis + Geck new 160-degree needle is ideal for cataract *and* corneal surgery.



Its depth of pass is designed to encourage precise approximation and minimize posterior gape in delicate ophthalmic closures.

For ease of handling, it's unsurpassed. The needle almost seems to draw your hand along behind it. And its point is precisely angled, honed and polished by hand.

Our needle has less of a shoulder than conventional



needles, so tissue is less inclined to tent during passes. It's made of a special blend of steel alloys, to resist bending and breaking. And it won't glare under the lights, like electro-polished needles can.

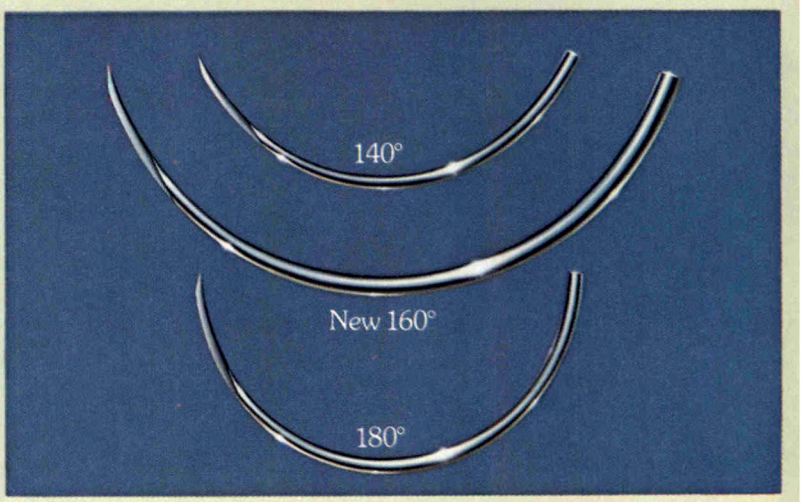
The new 160-degree micro- surgical needle is one of a full line of Davis + Geck ophthalmic needles that come in a variety of diameters and arc sizes.

...and ends with our sutures.

For procedures requiring nonabsorbable sutures, Davis + Geck needles are available on Dermalon® monofilament nylon suture and on silk.

When you need an absorbable suture, use a Davis + Geck needle on Dexon® "S" polyglycolic acid suture.

Whatever ophthalmic procedures you perform, you'll get precise, predictable results from your sutures when you close with Davis + Geck.



DG
DAVIS+GECK

The infected eye–

If you trace it to one of these common problems...



*Acute bacterial conjunctivitis** Marked by itching or burning, hyperemia, lid edema and lacrimation or purulent discharge. Second only to refractive errors in frequency among ocular complaints.



*Bacterial corneal ulcer** Pain, photophobia, lacrimation and blepharospasm are usual presenting symptoms. Prompt institution of therapy is vital to avoid possible visual difficulty.

*When due to susceptible microorganisms.

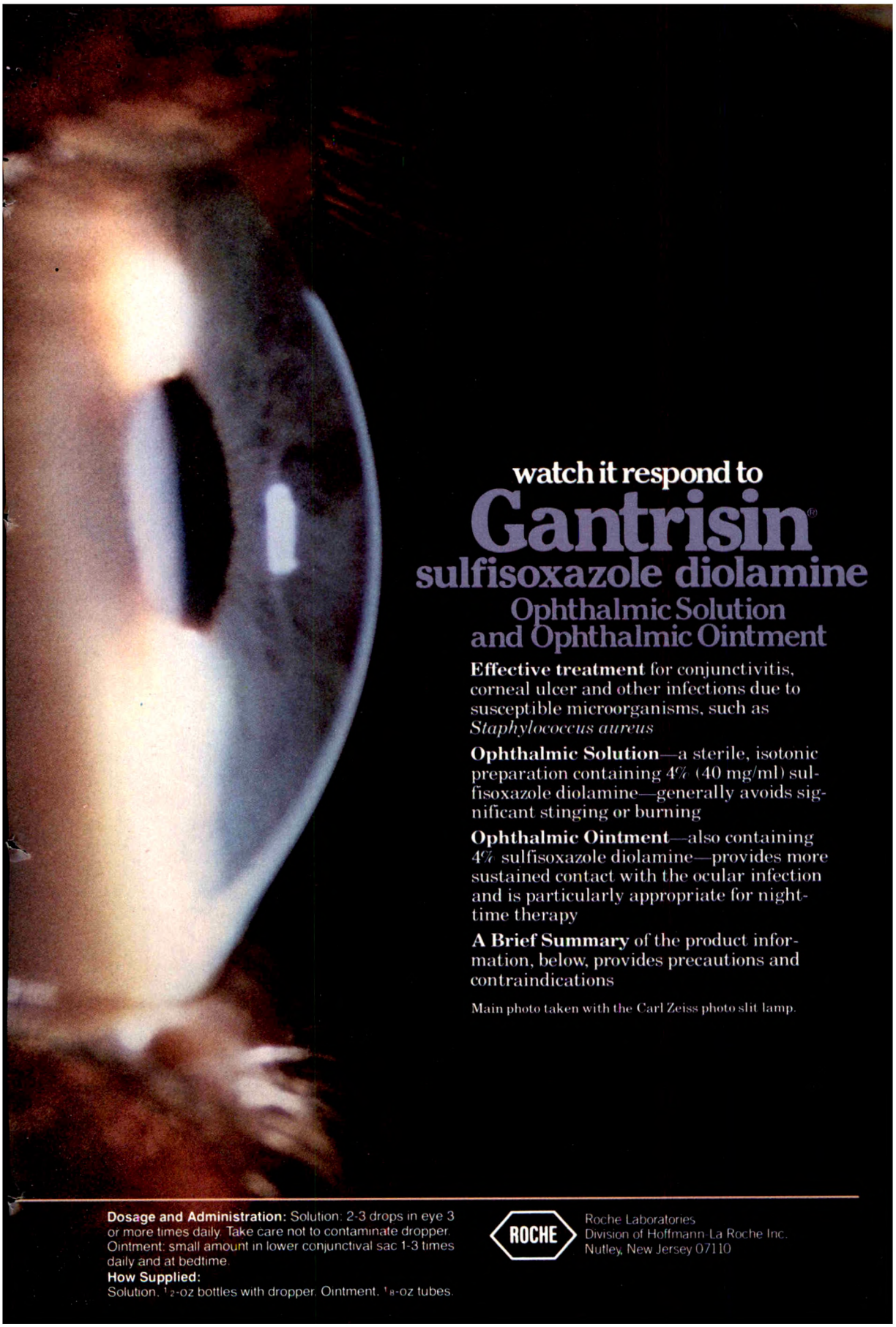
Gantrisin® **sulfisoxazole diolamine/Roche**
Ophthalmic Solution, Ophthalmic Ointment

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Conjunctivitis, corneal ulcer, other superficial ocular infections due to susceptible microorganisms; as adjunct in systemic sulfonamide therapy of trachoma.

Contraindications: Hypersensitivity.

Precautions: Incompatible with silver preparations; inactivated by para-aminobenzoic acid in purulent exudates; ma increase growth of nonsusceptible organisms, including fungi. Ointment may retard corneal healing. Discontinue use



watch it respond to

Gantrisin®

sulfisoxazole diolamine

Ophthalmic Solution and Ophthalmic Ointment

Effective treatment for conjunctivitis, corneal ulcer and other infections due to susceptible microorganisms, such as *Staphylococcus aureus*

Ophthalmic Solution—a sterile, isotonic preparation containing 4% (40 mg/ml) sulfisoxazole diolamine—generally avoids significant stinging or burning

Ophthalmic Ointment—also containing 4% sulfisoxazole diolamine—provides more sustained contact with the ocular infection and is particularly appropriate for nighttime therapy

A Brief Summary of the product information, below, provides precautions and contraindications

Main photo taken with the Carl Zeiss photo slit lamp.

Dosage and Administration: Solution: 2-3 drops in eye 3 or more times daily. Take care not to contaminate dropper. Ointment: small amount in lower conjunctival sac 1-3 times daily and at bedtime.

How Supplied:
Solution, ½-oz bottles with dropper. Ointment, ⅛-oz tubes.

ROCHE

Roche Laboratories
Division of Hoffmann-La Roche Inc.
Nutley, New Jersey 07110

IT CAN MAKE YOUR

It's the new office computer from Acuity Systems.

And it's specifically designed to take a successful solo or group eye care practice like yours, and make it even more productive.

Because it speeds up paperwork in your front office, accelerates your cash flow, and gives you a more complete financial picture of your practice. It also lets you spot problems before they occur, so you can manage your practice better. And it shows you how to make your working day more efficient, so you can spend more time seeing patients.

What's more, it does all this without adding staff. And without adding space.

Think of your next patient as 15 pieces of paperwork.

You may not realize it, but every patient who enters your office creates as many as 15 separate pieces of paperwork.

And no matter how small your practice is, handling those ledger cards, bills, files, and insurance forms (to name only a few) takes thousands of staff hours a year, as well as your own time. Time you could be putting to better use.

But the Acuity Systems office computer drastically cuts the time spent on paperwork. Because it keeps complete patient accounts, types up insurance forms, and schedules appointments. It also produces itemized bills and daily bank deposit slips. And it performs a variety of other time-consuming daily record-keeping chores quickly and accurately.

It also functions as a word processor. In this mode, it can search your records for patients who need to be recalled, and then type labels for reminder postcards. Plus it can generate personalized form letters for insurance inquiries, referral replies, and other repetitive correspondence.

What's more, the Acuity Systems office computer stores all its files electronically on magnetic disks. This allows your staff instant access to records, speeding answers to patient inquiries, and improving patient relations.

You get a better cash flow, plus vital financial data.

Because Acuity Systems' office computer does everything with incredible speed, patients can receive bills on the spot, and insurance claims can be processed the same day.

And, it also keeps track of aging accounts for you. At the touch of a button, it can produce a list of past-due patients your staff can utilize for an on-going collection program.

As a result, collectibles are greatly reduced. So you'll have a steadier, more reliable cash flow that will allow you to manage your funds better.

In addition, the Acuity Systems office computer automatically prepares a variety of important financial reports, including trial balance, profit and loss statements, and balance sheets. These reduce your need for costly accounting services. And they give you a day-to-day understanding of your financial position you may never have had before.



The Acuity Systems' office computer, part of the Intelligent Office™ system of practice management. Shown are the display terminal with keyboard, high-speed printer, and disk drive. The disk drive can also be placed in a closet, allowing the terminal and printer to be placed on a desk top.

It puts your finger on the pulse of your practice.

In addition to performing all your daily bookkeeping and billing functions, Acuity Systems' office computer also compiles a variety of practice management reports, including:

- practice income by doctor, procedure, day, or any of a variety of other variables.
- revenue analysis, allowing you to monitor your sources of income.
- inventory control (for practitioners who dispense).

With these reports, you'll be able to monitor your own workload, or that of other members of your practice. You'll be able to see where your income comes from, and where you may be able to reduce your overhead. And you'll have the information you need to manage your practice effectively.

What's more, since the computer is completely contained in your office, only you have access to this confidential patient and practice information.

It speaks plain English, so it's easy to operate.

You might think that a computer system this sophisticated would

© 1980 Acuity Systems Incorporated.

F RACTICE PERFECT.

need its own programmer. But the Acuity Systems office computer doesn't, because it's preprogrammed. In fact, it's so easy to use that your secretary can be trained to run it in just a few hours. There's no special computer language to learn, because it actually prompts your secretary with simple, step-by-step instructions. Which means it's almost impossible to make a mistake.



Designed for eye care professionals, by the people who know eye care best.

As an eye care professional, both the way you run your practice and the procedures you perform are very different from the way other practitioners operate.

Yet all too many of the new "doctor's computers" offer a single program which attempts to meet the needs of all practitioners, regardless of specialty.

The Acuity Systems program on the other hand, is the result of thousands of hours spent studying efficient eye care practices, as well as four years of research and development in the United States and Canada.

What all this means is that, whether yours is a solo or a multi-doctor practice, the Acuity Systems office computer has the features and functions you need for eye care, programmed in the way that's most convenient to use them.

With Acuity Systems behind it, you can be sure it'll work.

And that's a feeling of security you won't get elsewhere.

Because most other "computer companies" sell you the computer and the program, and then kiss you goodbye. They don't help you through the transition period, when the computer is new. While some of them do train your employees, they make no provision for training new employees later on. None of them follow up to make sure the system's working right. And few of them actually service both the hardware and software.

But at Acuity Systems, we give you all the services the other companies don't. Like complete planning for the transition to computerization by our Practice Management Analyst. Complete training, plus follow-up and new employee training by our Customer Service representatives. And 24-hour service of any problem by our own nationwide service network.

Why do we do all this for you? Because in the past 10 years, we've acquired an excellent reputation from our other products, the Auto-Refractor® and the Auto-Lensmeter™. And making sure our new office computer works perfectly in every practice is the only way we're going to keep that reputation.

Perfect the way you practice.

Send for our free booklet, "The Doctor's Guide to Efficient Practice Management". Better still, call us toll-free: **800-336-0359.***

Tell me more.

☐ Please send me your free booklet "The Doctor's Guide to Efficient Practice Management."
☐ Please call to arrange a time for a systems analyst to visit my office to determine its feasibility for such a computer/word processor system.
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☐ M.D.
☐ O.D.

Name _____

Street _____

City _____ State _____ Zip _____

Your Practice Specialty _____

Telephone ( ) _____

We would like to tailor a package of information best suited to your interest, so please provide us with this information to help us best respond:

1. I ☐ have ☐ have not previously investigated a computer system for my office needs.
2. I ☐ have ☐ have not previously investigated a word processing system for my office.
3. My patient billing currently ☐ is ☐ is not computerized.
4. I have a front-office staff of _____ persons.
5. In a typical day I see approximately _____ patients.
6. About _____ % of my patients require some type of 3rd-party form to be completed by me.
7. I currently am re-paid by _____ types of 3rd party insurance or government plans.
8. My interest in such a system is ☐ immediate ☐ 6 to 12 months ☐ long-term.

Mail to: Acuity Systems, Inc. AJOPH110
11413 Isaac Newton Sq., Reston, VA 22090

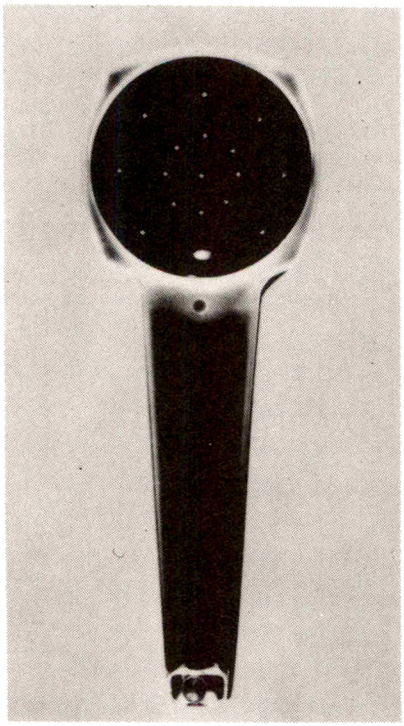
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Advanced Instrumentation and Systems for Health Care

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Medical Instrumentation Division,
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*In Virginia, call collect: (703) 471-4700.



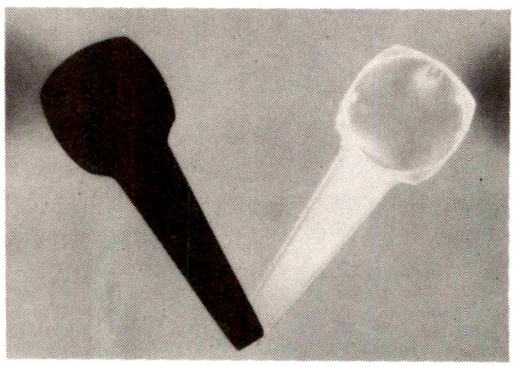
A PLUS FOR OPHTHALMOLOGY WITH VELOTRON'S PINHOLE PLUS

Lightweight (21 grams), Economical and Pocketable, it allows for rapid estimate of visual acuity of post-operative cataract patients for both distance (20 feet) and near (14 inches).

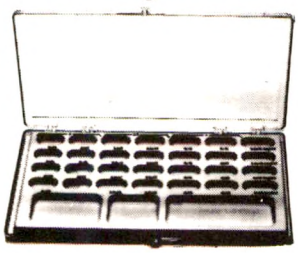
Easily carried in either pocket or purse, patients with sub-normal reading vision find the PINHOLE PLUS an extremely valuable aid.

For further information, please contact:

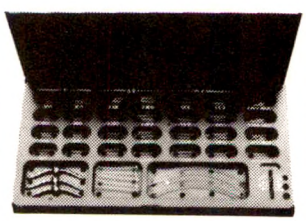
VELOTRON, INC.
6517 Ft. Hamilton Pkwy.
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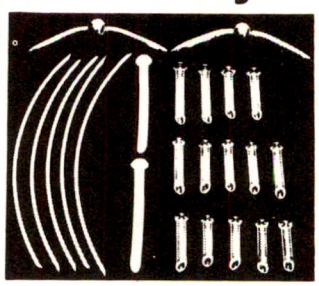
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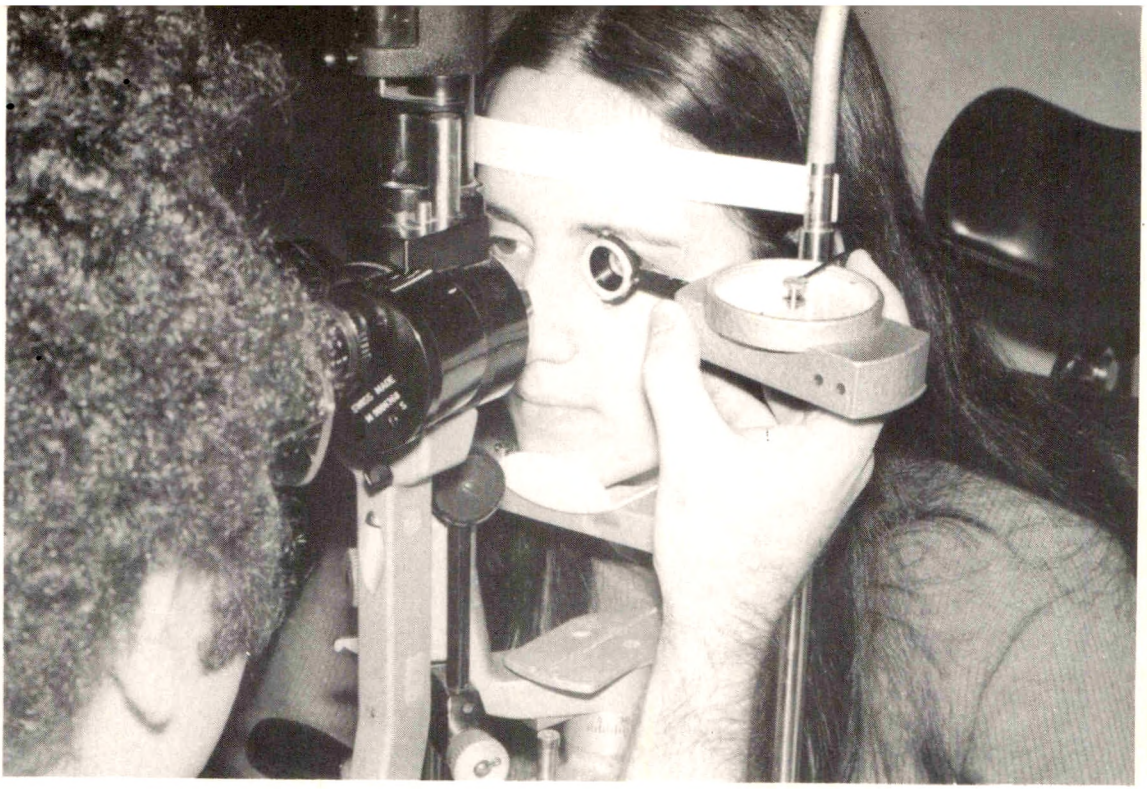
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* L.T. Jones M.D., Dacryocystorhinostomy, American Journal of Ophthalmology, Volume 59, No. 5, May, 1965.
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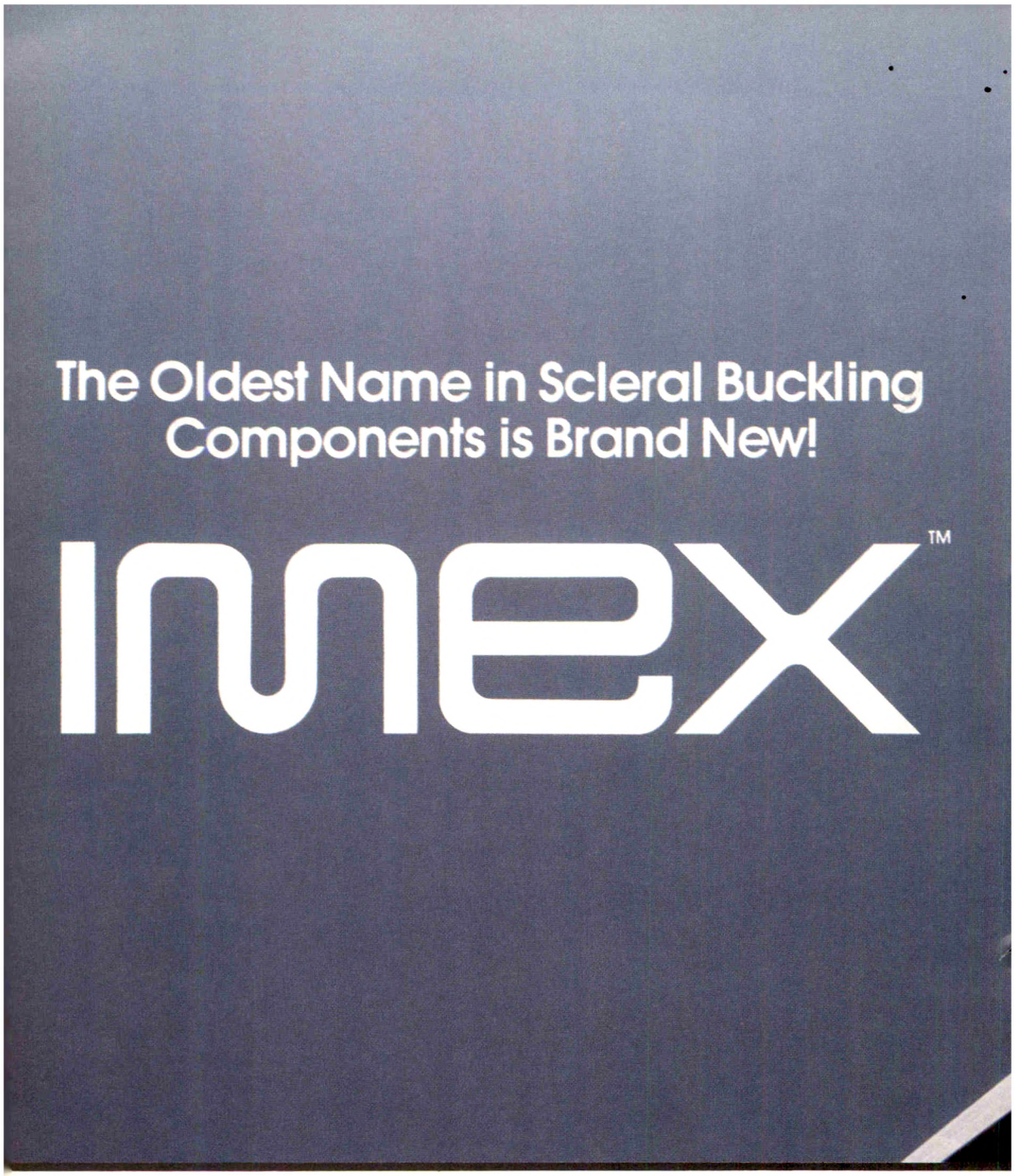
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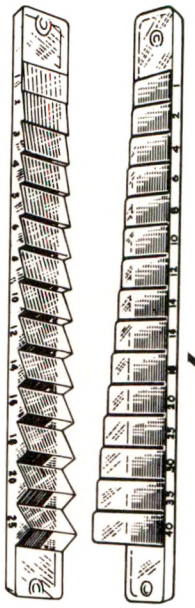
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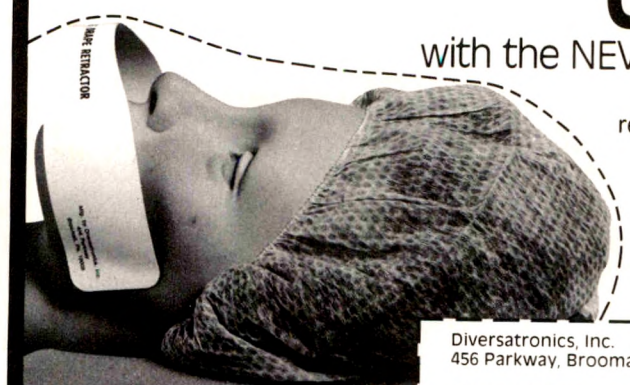
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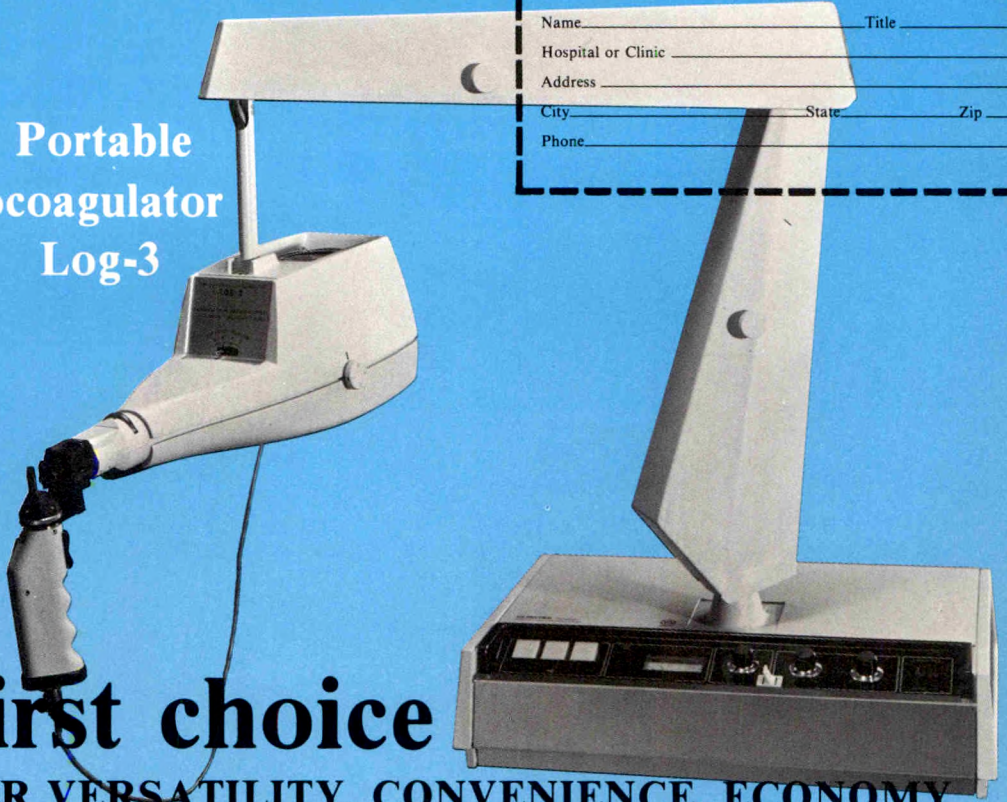
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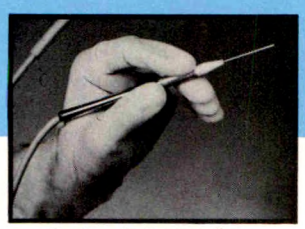
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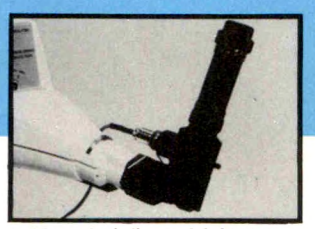
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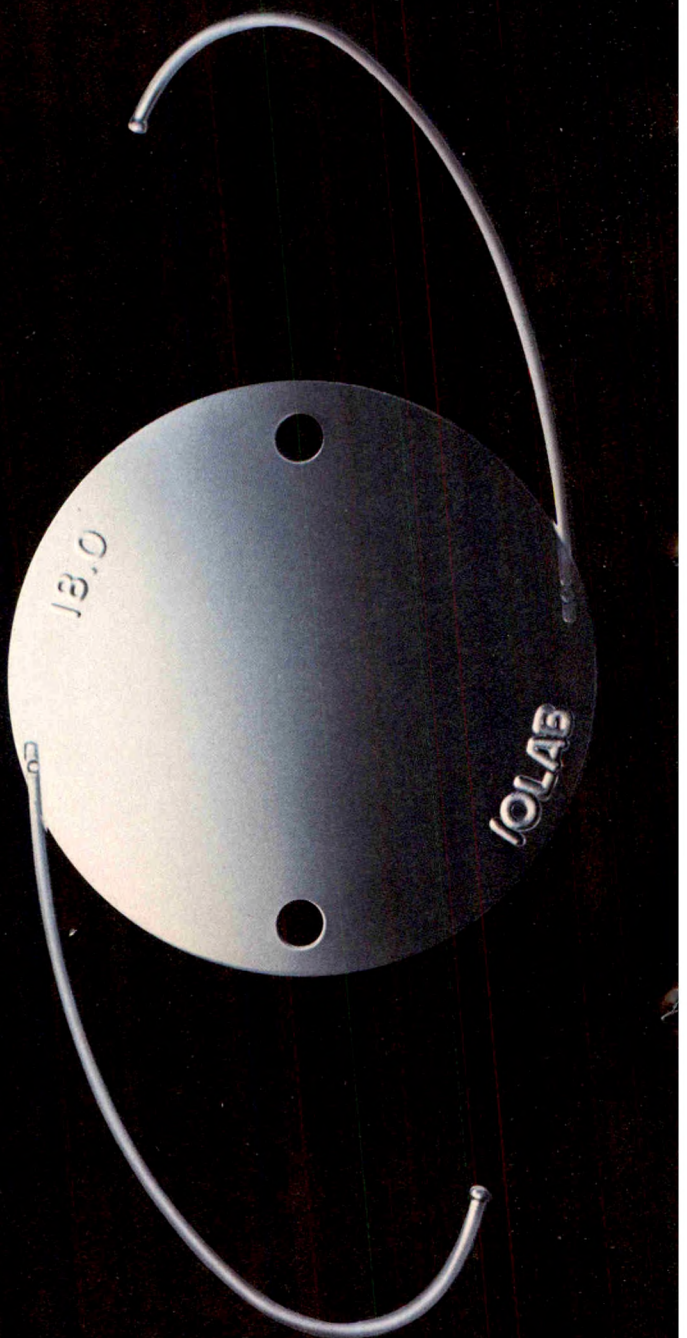
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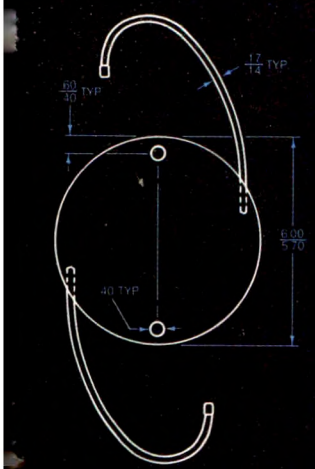
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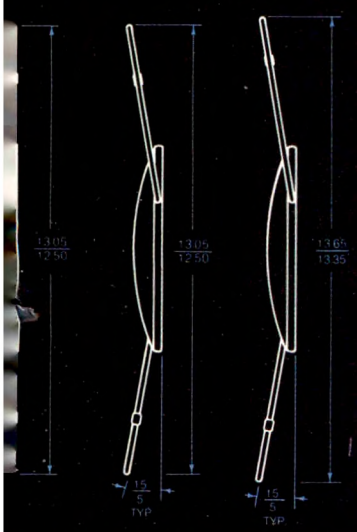
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del 103J * Model 103M Model 103L

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Model 103M
same specifications as Model 103J except loops are angled 10° toward the convex side
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**"Intraocular Degradation of Non-absorbable Sutures." Richard L. Kronenthal, Ph.D., Somerville, New Jersey, Ethicon, Inc., Vol. 3, No. 3 & 4, July, October, 1977, American Intra-Ocular Implant Society Journal.

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AMERICAN JOURNAL OF OPHTHALMOLOGY™

VOLUME 90 NUMBER 5 NOVEMBER, 1980

ATROPHIC MACULOPATHY ASSOCIATED WITH HEREDITARY ATAXIA

K. U. DUINKERKE-EEROLA, M.D., J. R. M. CRUYSBERG, M.D., AND A. F. DEUTMAN, M.D.

Nijmegen, The Netherlands

The association of hereditary ataxia with optic atrophy and pigmentary retinopathy is well known. There have been, however, only a few reports on hereditary ataxia with macular degeneration. We studied two siblings with circumscribed atrophic lesions in the macula of each eye. Retinal function studies disclosed no selective disturbance of the cone or rod system. Both patients suffered from a progressive neurologic disease with ataxia, probably caused by olivopontocerebellar degeneration.

SUBJECTS AND METHODS

Two affected siblings (Fig. 1) underwent routine ophthalmologic evaluations, fluorescein angiography, fundus photography, color vision testing, visual field examination, scotopic and photopic electroretinography (ERG), electro-oculography (EOG), and testing of the visual-evoked potentials. The techniques used have been described elsewhere.[1-4] Three other siblings and the parents underwent routine ophthalmologic examinations.

CASE REPORTS

Case 1—A 43-year-old woman was referred to us in August 1978 because of severely reduced visual acuity in both eyes. She had first noted diminishing visual acuity 16 years earlier. At that time she had consulted a neurologist because she was beginning to develop ataxia and slurred speech. A recent neurologic examination had confirmed the findings of the earlier examinations: ataxia, dysarthria, nystagmus, hyperreflexia, and slight mental deterioration. In addition to cerebellar symptoms, the patient also had pyramidal and extrapyramidal disturbances. The cerebrospinal fluid was normal. Computed axial tomography showed cerebellar atrophy. Pontine atrophy could not be seen clearly because of pneumatized osseous structures. The clinical pattern was consistent with olivopontocerebellar degeneration. The symptoms had progressed and, by the time of our examination, the patient was able to move about only in a wheelchair. She did not take any medications.

The patient was the fourth of 11 children (Fig. 1). One brother showed a similar neurologic pattern and had poor visual acuity.

Visual acuity in both of her eyes was counting fingers at 1 meter. There was emmetropia but no strabismus; ocular motility was normal. The pupils were equal and reacted normally to light. Slit-lamp examination disclosed no unusual findings. The ophthalmoscopic examination disclosed bilateral macular lesions with horizontal ovoid zones of atrophy (Fig. 2). The optic disks were normal. The visual fields, recorded by Goldmann's kinetic method, were generally depressed and nasally contracted. However, the patient was not able to cooperate perfectly.

Fluorescein studies disclosed a central pigment dystrophy with secondary atrophy of the lamina choriocapillaris (Fig. 3). There was a horizontal oval area of atrophic pigment epithelium extending to the temporal border of the optic disk. In the middle of the atrophic area, there was a small round area showing secondary loss of the lamina choriocapillaris. There was no evidence of leakage. The ERG showed normal photopic and scotopic responses. The EOG was normal in both eyes (light peak:dark trough ratio was R.E.: 1.85 and L.E.: 1.92). Dark adaptation was grossly normal. The visual-evoked responses were not recordable.

Because of her poor visual acuity, color vision testing was limited to identifying large colored surfaces.

Case 2—A 32-year-old man, the brother of Patient

From the Institute of Ophthalmology, University of Nijmegen, Nijmegen, The Netherlands.

Reprint requests to K. U. Duinkerke-Eerola, M.D., University of Nijmegen, Institute of Ophthalmology, Philips van Leydenlaan 15, Nijmegen, The Netherlands.

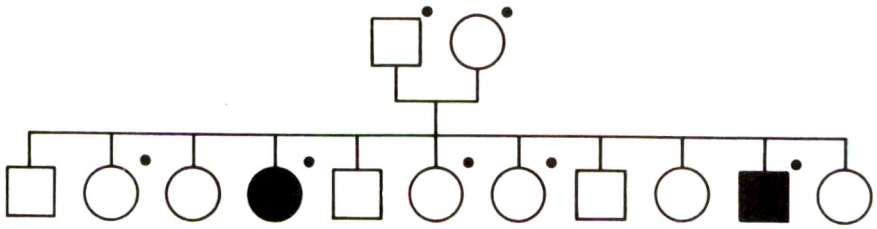


Fig. 1 (Duinkerke-Eerola, Cruysberg, and Deutman). Pedigree showing the two affected persons with bull's-eye maculopathy and hereditary ataxia. Open squares, unaffected males; open circles, unaffected females; dots, examined; solid circle, Patient 1; solid square, Patient 2.

1, was referred to us in January 1979 because of gradual loss of visual acuity. The patient had been examined by a neurologist because he had had slowly progressive gait disturbances and slurred speech since he was 8 years old.

Clinical examination showed ataxia, dysarthria, dysdiadochokinesia, dysmetria, nystagmus, hyperreflexia, and slight mental deterioration. We found no extrapyramidal disturbances. The electroencephalogram and the electromyogram were normal. Laboratory evaluation provided no unusual findings. The cerebrospinal fluid was normal. The pneumoencephalogram disclosed a conspicuously small cerebellum and frontal atrophy. The fourth ventricle was dilated, but the diameter of the pons cerebelli was normal. Although the clinical signs and symptoms were mostly cerebellar, this disease was presumably similar to that of his sister.

Visual acuity was R.E.: 6/60 (20/200) and L.E.: 6/30 (20/100). There was no ametropia. There was esotropia with limited abduction of the left eye. Slit-lamp examination disclosed no unusual findings. The ophthalmoscopic examination showed early macular pigment alterations and an incomplete ring-like zone of atrophy. The optic disks and the retinal peripheries were normal. Fluorescein studies supported these findings (Fig. 4). Temporal to both maculas there were horseshoe-shaped atrophied areas in the pigment epithelium. No other abnormalities were visible by fluorescein angiography. The photopic and scotopic ERG responses were at the lower limit of normal. Dark adaptation was grossly normal. The EOG was normal in both eyes (light peak:dark trough ratio was R.E.: 2.05 and L.E.: 2.50). Visual-evoked potentials were bilaterally diminished. The pseudo-isochromatic color tests (AOH-R-R plates) were normal. The New Color Test Box 6/4 also gave normal results. The desaturated D-15 test of Lanthony showed only minor errors. The result of the Farnsworth-Munsell 100-hue test was normal in the right eye, but there was a higher error score without prominent axis in the left eye. Visual fields, recorded by Goldmann's kinetic method, disclosed centrally diminished sensitivity of both

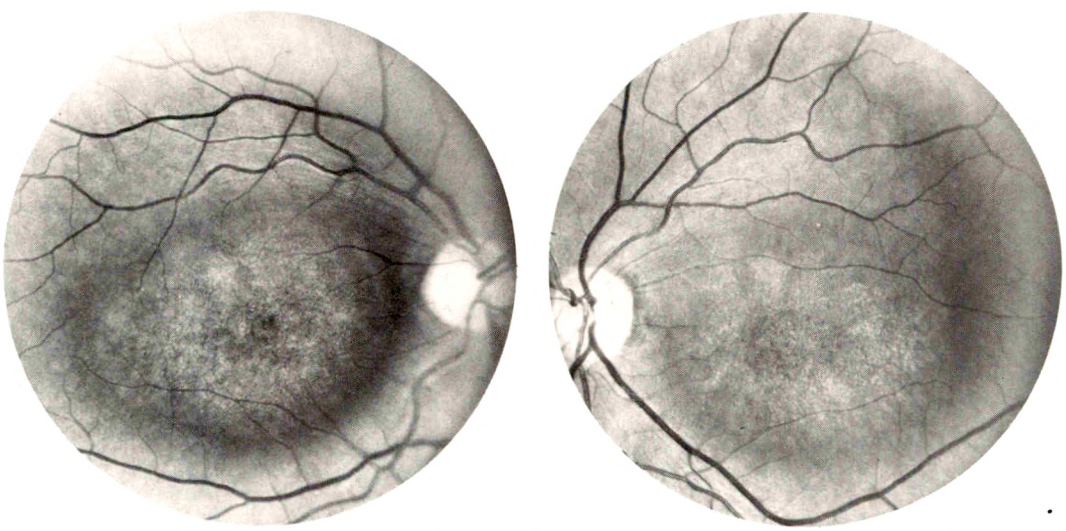


Fig. 2 (Duinkerke-Eerola, Cruysberg and Deutman). Case 1. Fluorescein angiograms of the right eye (left) and the left eye (right) show advanced bull's-eye lesions in the maculas.



Fig. 3 (Duinkerke-Eerola, Cruysberg, and Deutman). Case 1. Fluorescein angiograms of the right eye (left) and the left eye (right) show horizontal oval areas of atrophic pigment epithelium extending to the temporal border of the optic disk. There was no evidence of leakage.

eyes. The peripheral isopters were normal. There was significantly decreased sensitivity, both centrally and pericentrally, measured by static perimetry with the Tübinger perimeter.

Discussion

Two siblings had hereditary ataxia with diminished visual acuity and an atrophic maculopathy in different stages. The ERGs showed no selective cone dysfunction and there was no photophobia. The color vision of Patient 1 was difficult to test because of her poor visual acuity. The color vision of Patient 2 was slightly disturbed. Neither had an acquired type I red-green defect.

The parents of these patients were not,

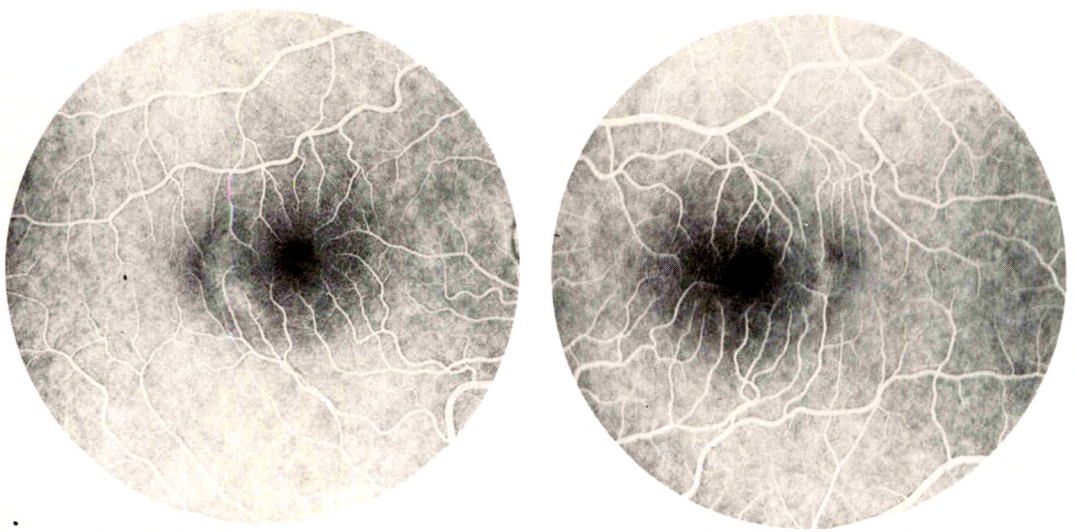


Fig. 4 (Duinkerke-Eerola, Cruysberg, and Deutman). Case 2. Fluorescein angiograms of the right eye (left) and the left eye (right) show horseshoe-shaped areas of atrophy of the pigment epithelium temporal to both maculas.

to our knowledge, consanguineous. Except for an old, unilateral retinal vascular lesion in the mother, their eyes were normal. Ophthalmologic examinations of three siblings failed to show any abnormalities. The other siblings refused to be examined. One brother and one sister of the father had died of a progressive neurologic disease about which we unfortunately could not get detailed information. The aunt and uncle had had no visual complaints, but their disease might have been similar to that of our two patients.

The ophthalmologic history of the family disclosed no specific features. The macular lesions suggested an early bull's-eye maculopathy in Case 2 and a late form of bull's-eye maculopathy with a horizontal ovoid zone of complete atrophy in Case 1. Bull's-eye lesions of the macula have been found in cone dystrophy, chloroquine intoxication, ceroid lipofuscinosis (Spielmeyer-Vogt-Batten-Mayou), benign concentric annular macular dystrophy, and in Stargardt's disease.[5-8]

Our patients had no history of chloroquine ingestion. Because of the slight disturbances in the ERG and color vision, the ocular findings in our patients have a certain resemblance to benign concentric annular macular dystrophy.[6] The visual acuities of our patients, however, were significantly lower. Because of the age of the patients and the ERG findings, we excluded the diagnosis of neuronal ceroid lipofuscinosis.[8]

Bull's-eye macular lesions are frequently found in cone dystrophy. The color vision and the photopic ERG are usually severely affected in cone dystrophy. The cone part of the dark-adaptation curve is elevated but the rod part is usually normal. Results of an EOG are generally normal in the beginning, but below normal in advanced cases.[5]

Our patients showed an atrophic macular pattern without selective cone dysfunction and, therefore, probably did not have any cone dystrophy syndrome. It was difficult to place these patients into a well-defined neuro-ophthalmologic category.

McKusick[9] distinguished five types of olivopontocerebellar atrophy: Type 1 (Menzel), autosomal dominant; Type 2 (Fickler-Winkler), autosomal recessive; Type 3, retinal degeneration, autosomal dominant; Type 4 (Schut-Haymaker), autosomal dominant; and Type 5, dementia and extrapyramidal symptoms, autosomal dominant. Although we were not able to confirm autosomal-dominant inheritance, we believed our patients had Type 3, with retinal degeneration.

We were unable to find any description of bull's-eye maculopathy in published reports of cone dysfunction associated with hereditary ataxia.[10-13] Froment, Bonnet, and Colrat[14] described four affected persons in three successive generations. They referred to the neurologic lesion as spinocerebellar degeneration. The retinopathy was peripheral in the first generation, macular in the second, and macular and circumpapillary in the third.

Havener[15] described a combination of cerebellar and macular degeneration in a father whose daughter was also neurologically affected. At autopsy the father appeared to have olivopontocerebellar degeneration. The daughter showed no ophthalmologic abnormalities, whereas the father had bilateral macular degeneration and temporal pallor of the optic disks.

Björk, Lindblom, and Wadensten[10] reported macular degeneration associated with cone dysfunction in patients with autosomal-dominant Marie's ataxia. Their patients also had rod involvement, which they thought was secondary to cone dysfunction. These patients later developed achromatopsia.

In 1959 Woodworth, Beckett, and Netsky[16] described a family with progressive

ataxia and olivopontocerebellar degeneration, Leber's optic nerve atrophy, and Friedreich's ataxia. Ophthalmoscopic examination showed spotty pigmentations in the fundus with optic nerve atrophy. Sections of the retina showed loss of ganglion, bipolar, and receptor cells and glial adhesions of the retina to the choroid. The authors did not report any macular lesion. This degenerative disorder appeared in four men in two generations and the pattern of inheritance may have been irregular dominant. Jampel, Okazaki, and Bernstein[17] described eight patients with spinocerebellar ataxia, external ophthalmoplegia, and pigmentary mottling beginning in the macula and progressing to the periphery (in four sibships in three generations). The ERGs of these patients showed a general impairment of the retinal function.

Foster and Ingram[18] described six affected persons in four generations of one family with ataxia and retinal degeneration. The ophthalmologic findings consisted of marked clumping of pigment peripherally and in the region of the macula and attenuated vessels. The pattern of inheritance appeared to be autosomal dominant.

Carpenter and Schumacher[19] described a family in which a father and three of his five children were affected by a progressive neurologic illness. Ophthalmoscopy of the father showed perivenous sheathing and optic nerve atrophy. Histopathologic examination of the father disclosed olivopontocerebellar degeneration. Ophthalmologic examinations of the children showed pigmentary mottling throughout the retinas. No other examinations were performed.

Halsey, Scott, and Farmer[20] found degenerative changes in the retinas and cerebellums of 11 persons in three generations of a family. Blindness and ataxia were the clinical features. The fundus changes were mainly macular. Consanguinity and the skipped generations suggested recessive inheritance although dominant transmission could not be excluded because of a high illegitimacy rate in this family.

Weiner and associates[21] described a family in which 27 members in five generations were affected by histopathologically confirmed olivopontocerebellar degeneration. The ophthalmologic findings consisted of pigmentary changes in the macula, progressing further to the periphery. No EOGs were performed. Sections of the eyes showed a total loss of the receptors and an almost complete absence of ganglion cells. The bipolar cells and the cells of the outer nuclear layer were greatly reduced in number.

Ryan and associates[22] suggested that the initial pathologic process of the patients described by Weiner and colleagues was probably localized in the retinal pigment epithelium with secondary changes in the sensory retina and the lamina choriocapillaris.

Ryan and Smith[23] described three persons in one family (the propositus and his sister and her daughter) with olivopontocerebellar degeneration. They reported a progressive macular dystrophy with migration of pigment in addition to loss of the lamina choriocapillaris and the pigment epithelium in the affected area. Results of ERGs and the EOGs were aspecifically below normal. The process progressed to the peripheral retina. The inheritance suggested an autosomal-dominant pattern.

Verougstraete and Toussaint[11] described a family with autosomal-dominant spinocerebellar heredoataxia, macular pigment migration, and optic nerve atrophy. The ERGs disclosed below-normal function with specific dysfunction of the cone system. Color vision testing showed an acquired red-green dyschromatopsia.

The combination of a photopically disturbed ERG and macular pigment migra-

tion was also noted by Ohrn-Degueldre and associates[12] in a family with peroneal muscular atrophy (Charcot-Marie-Tooth disease).

Stanescu-Segal and Michiels[13] studied six patients with different forms of heredoataxia. Despite minor or no ophthalmologic changes, the ERGs showed a predominantly impaired cone function. The authors thought that this association had been missed in the past. However, our patients showed no selective cone dysfunction.

De Jong and associates[24] examined 45 persons in one family with olivopontocerebellar degeneration. Nineteen patients showed pigmentary mottling in the maculas. Three of the patients showed peripheral pigmentary mottling. Autopsy of one patient showed the cones, the rods, and the outer nuclear layer of the maculas to be absent. There was also a subfoveal blister containing protein-rich fluid in the left eye. The ERGs, EOGs, and the visual-evoked potentials provided no unusual findings.

Some of the earlier reports found a photopically disturbed ERG with a macular lesion, which often gradually progressed to the retinal periphery. Our patients had a lesion confined to the macula without selective cone dysfunction. Color vision testing did not show the typical acquired defects found in central cone dystrophies.[25] One of our patients had the atrophic macular lesion characteristic of long-standing bull's-eye maculopathy, whereas the other had the ring-like depigmentation in the macular area characteristic of early bull's-eye maculopathy. We did not find any description of distinct bull's-eye maculopathy in the earlier reports where the macular lesion was called pigmentary mottling. Two siblings being affected suggested an autosomal-recessive inheritance pattern but the disease may have skipped a generation in the father. One brother and one sister of the patient's father apparently had a progressive neurologic disorder, suggesting an irregular autosomal-dominant inheritance pattern.

Summary

Two siblings had atrophic maculopathy associated with hereditary ataxia clinically defined as an olivopontocerebellar degeneration. The macular changes were suggestive of an early bull's-eye maculopathy in one case and of a late atrophic stage of bull's-eye maculopathy in the other. This suggests that concentric annular (bull's-eye) macular dystrophy may occur as part of olivopontocerebellomacular degeneration. Exogenous causes were excluded. The inheritance pattern was autosomal recessive or irregular autosomal dominant. The relationship of these two degenerative disorders is not yet clear. This combination of findings has been, to our knowledge, only rarely described.

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RETINAL PIGMENT EPITHELIAL DETACHMENTS IN POSTERIOR SCLERITIS

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Posterior scleritis is a rare unilateral condition that generally affects women in the fourth through sixth decades of life.[1,2] Benson and associates[2] recently reported fluorescein angiographic findings in posterior scleritis with exudative retinal detachment. They described multiple, pinpoint areas of leakage in the region of chronic exudative retinal detachment. We studied a 12-year-old girl with posterior scleritis with the typical symptoms and multiple retinal pigment epithelial detachments. To our knowledge, this girl is the youngest patient with posterior scleritis ever described in published reports.

CASE REPORT

A previously healthy 12-year old girl had had a headache in the area of the right eye for five days. Additionally, the patient experienced generalized malaise.

Ocular examination showed a visual acuity of R.E.: 6/9 (20/30) and L.E.: 6/7 (20/25). Positive findings were confined to the right eye. The globe was tender and 5 mm proptotic. The eyelids were mildly edematous and mild blepharoptosis was also present. The eye had limitation of duction movements in all directions. Lacrimation and photophobia were present. The conjunctiva was mildly chemotic and moderately injected. The episclera was also injected. Results of the remainder of the slit-lamp examination were normal. Many blister-like elevations of the retinal pigment epithelium and overlying retina were present throughout the macula (Fig. 1, left). The left eye was completely normal.

B-scan ultrasonography disclosed a flattened posterior globe, scleral and choroidal thickening, and retrobulbar edema (Fig. 2). Fluorescein angiography demonstrated many retinal pigment epithelial detachments in the macula (Fig. 1, right).

Results of a complete blood cell count, chest X-ray, serum lysozyme, serum calcium, and serum protein electrophoresis, quantitative immunoglobulin test, and qualitative immunoelectrophoresis were normal. The fluorescent treponemal antibody-absorption and antinuclear antibody tests were nonreactive. The erythrocyte sedimentation rate was increased at 41 mm/hr and the total complement level was also slightly elevated. The quantitative T-cell level was slightly decreased and quantitative B-cells were slightly increased. A PPD skin test was nonreactive.

The patient was hospitalized but no underlying systemic disease was found. Visual acuity worsened to 6/60 (20/200) and the patient began taking 80 mg of prednisone per day. The pain and proptosis dramatically improved. The erythrocyte sedimentation rate declined to 24 mm/hr.

Since these manifestations of posterior scleritis were unusual, we performed a computed axial tomographic scan. This disclosed thickening of the posterior sclera of the right eye (Fig. 3).

Since the patient had some abnormal results on laboratory tests for immunity, we performed a conjunctival biopsy. It showed no abnormal deposition of immunoglobulins or complement and no viral inclusion bodies or lymphocytic infiltration. After six days of corticosteroid treatment, the retinal pigment epithelial detachments had resolved and visual acuity had improved to 6/15 (20/50) (Fig. 4).

DISCUSSION

Posterior scleritis is a rare condition accounting for approximately 2% of all scleritis cases.[1] It is usually associated with anterior scleritis and older reports frequently referred to it as diffuse scleritis with posterior involvement.[3] As tenonitis is often present pathologically, the term sclerotenonitis has also been used for this condition.[4]

Our patient with posterior scleritis had typical symptoms and responded in the expected manner to corticosteroid treatment. The unusual features of the case were the young age of the patient, the presence of the pigment epithelial detachments, and the absence of posterior uveitis. Since choroiditis is frequently seen histologically in scleritis,[5-7] this adjacent inflammation may explain the pigment epithelial detachments. Perhaps the short period of time between the

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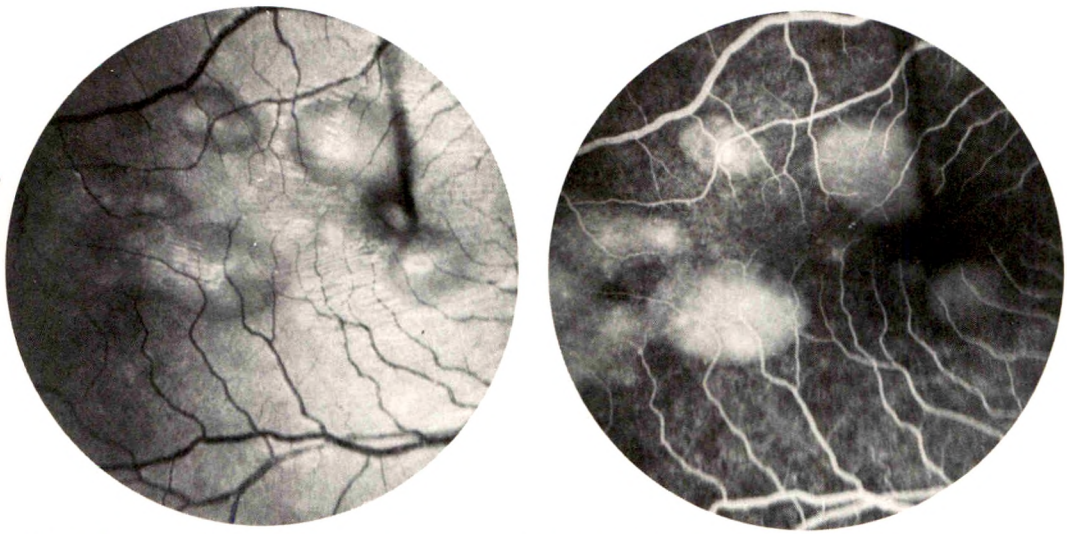


Fig. 1 (Berger and Reeser). Left, Many pigment epithelial detachments in macula. Right, Fluorescein angiogram of the same area.

onset of symptoms and treatment prevented the development of posterior uveitis and a secondary exudative retinal detachment. In addition to choroiditis, previous reports on the posterior complications of scleritis have mentioned retinal detachment,[1-10] macular edema,[3] retinal striae,[10] choroidal striae,[2,10] and optic disk edema.[3,4] More recently, investigators have emphasized the combination of retinal detachment, choroidal elevation, and posterior uveitis simulating a choroidal melanoma.[5-7] The role of fluorescein angiography and ultrasonography in the diagnosis of posterior scleritis was recently reported by Benson and associates.[2] However, all of their patients had chronic disease, frequently with long-standing exudative retinal detachments.

Since visual acuity is not dramatically affected in the acute phase of posterior scleritis, a careful ophthalmoscopic exam-

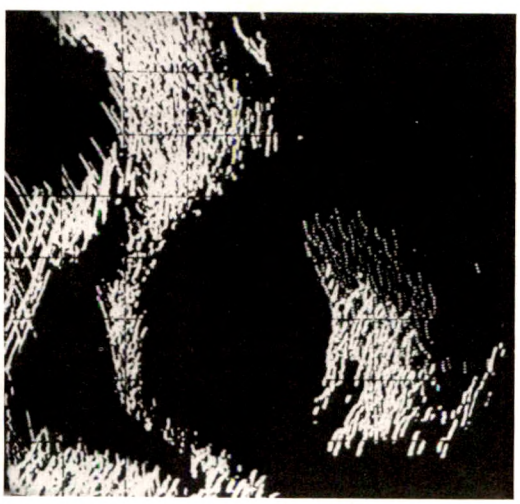


Fig. 2 (Berger and Reeser). B-scan ultrasonography demonstrating choroidal and scleral thickening and retrobulbar edema.



Fig. 3 (Berger and Reeser). Computed axial tomographic scan showing thickening of posterior sclera.

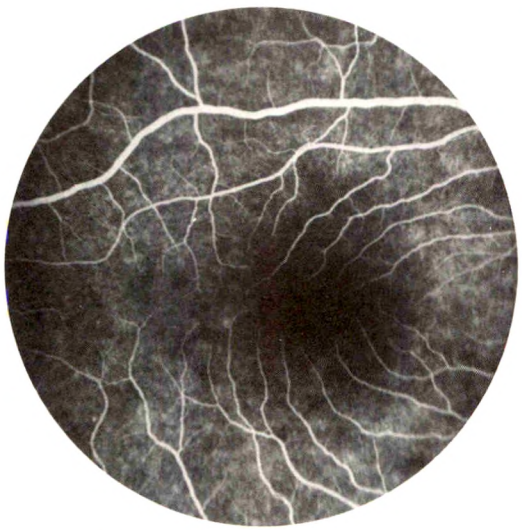


Fig. 4 (Berger and Reeser). Fluorescein angiogram showing resolution of pigment epithelial detachments with corticosteroid therapy.

ination may not be performed, especially if marked photophobia is present. If fluorescein angiography were to be done routinely during the acute phase of posterior scleritis, perhaps pigment epithelial detachments would be a more common finding.

The computed axial tomographic scan is a sensitive test for the diagnosis of posterior scleritis. Thickening of the sclera is easily detected, and this becomes more prominent with contrast infusion. Bernardino and associates[11] reported that orbital pseudotumor also causes thickening of the scleral-uveal rim, but only with contrast infusion. The two diseases share many of the same symptoms, including thickening of the extraocular muscles.[4] However, it is usually possible to distinguish between the two since computed axial tomography or ultrasonographic examination usually shows an orbital mass lesion in orbital pseudotumor. With infiltrating orbital pseudotumor, a mass lesion may not be shown, and the two diseases must be distinguished on the basis of the absence of anterior scleritis, exudative retinal detachment, and sexual predilection in pseudotumor.

Scleritis has been associated with rheumatoid arthritis, periarteritis nodosum, Wegener's granulomatosis, and relapsing polychondritis.[1,8] Although our patient had no evidence of these diseases, her youth puts her at risk for developing them in the future.

Summary

A 12-year-old girl developed posterior scleritis and many detachments of the retinal pigment epithelium. Computed axial tomographic findings, reported in posterior scleritis for the first time, disclosed thickening of the posterior sclera. Computed axial tomography appears to be a sensitive test for the diagnosis of posterior scleritis.

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PIGMENT EPITHELIAL DISEASES WITH ABNORMAL CHOROIDAL PERFUSION

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Gass[1] described a condition in which there were many bilateral subretinal lesions associated with rapid loss of central vision, followed by spontaneous resolution, leaving discrete pigment epithelial scars. Visual recovery was often incomplete. He called this condition acute posterior multifocal placoid pigment epitheliopathy. The fluorescein angiographic appearance of the pigment epithelial lesions was dark during the initial dye transit but became hyperfluorescent later in the study. He considered two possible explanations for this phenomenon: either that the choroid did not fill in the areas of swelling or that the swollen pigment epithelium obscured the choroidal fluorescence[1]; in either case the later hyperfluorescence could have been caused by leakage of fluorescein into the diseased pigment epithelial cells. It was not possible to distinguish between these alternatives with any confidence, but he favored primary pigment epithelial disease, possibly caused by infection, as the origin of the disorder. A number of authors supported the theory that a virus was the causative agent.[2-6]

Van Buskirk, Lessell, and Friedman[7] were the first to report an ocular condition identical to acute posterior multifocal placoid pigment epitheliopathy occurring in a patient with manifest systemic vascular disease. This association, combined with an abnormal pattern of choroidal filling on fluorescein angiography, suggested to them that the ocular disease was caused by choroidal vascular obstruction. This view was supported by many authors who believed that the lesions represented focal infarcts of the retinal pigment epithelium caused by intrinsic choroidal vascular disease.[4,8-12]

The most persuasive evidence for abnormal choroidal perfusion was Deutman and Lion's[11] demonstration of patent choroidal vessels underlying the placoid lesion on fluorescein angiography, indicating that obscuration of the choroidal fluorescence could not fully account for the dark appearance during the dye transit. They suggested that the obstruction was at the level of the precapillary arterioles. However, some masking undoubtedly occurs since large choroidal blood vessels cannot be seen in the depths of all lesions.

A few reports have demonstrated that the condition is not as well defined as it originally appeared. Changes similar to those described originally in acute posterior multifocal placoid pigment epitheliopathy[1] have been observed in eyes with limited serous retinal detachment[9] and in eyes with optic disk swelling.[10] Wright, Bird, and Hamilton[13] described a spectrum of disease from acute posterior multifocal placoid pigment epitheliopathy to Harada's disease and illustrated the difficulties in identifying specific clinical entities within their group of patients on the basis of fundus appearances and the clinical course of the disease. These patients had multifocal retinal pigment epithelial swelling in common and many showed choroidal filling patterns which the authors believed indicated abnormal choroidal perfusion.

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Headaches,[1,2,6,14,15] cerebrospinal fluid changes,[6,10,15,16] neurologic deficit,[6,15] and obstruction of cerebral blood vessels[6,15] have been described often enough to indicate that cerebrovascular inflammation commonly accompanies the ocular disorder.

We studied patients whose disorder was characterized by acute pigment epithelial swelling and in whom we found abnormal patterns of choroidal filling (similar to those originally described by Van Buskirk, Lessell, and Friedman[7]) at some stage during the disease.

Subjects and methods

We selected 16 patients who had acute pigment epithelial lesions and who showed delayed choroidal arrival of dye or prolonged choroidal filling during fluorescein angiography. We excluded patients with identical fundus appearances but who showed no evidence of abnormal slow filling or who were examined more than seven days after their first symptoms appeared.

Results

The pattern of fundus changes allowed us to divide the cases into three broad groups (Table).

Group 1—Five patients had bilateral, multifocal, yellow-white placoid lesions at the level of the retinal pigment epithelium with associated visual loss. On recovery there was discrete atrophy of the affected pigment epithelium and normal visual acuity except in one eye in which the lesion involved the fovea (Case 1). There was no detachment of the sensory retina or abnormality of the optic nerve head. These findings conformed with the original description of acute posterior multifocal placoid pigment epitheliopathy. Many discrete areas of each posterior pole showed early hypofluorescence on angiography, some of which stained in the later stages while others blended in with the background fluorescence (Fig. 1).

Group 2—In three patients the principal lesion was a unilateral, well-defined plaque of swelling at the level of the retinal pigment epithelium; in all three this was associated with profound and permanent central visual loss and was followed by atrophy of the pigment epithelium. There was no evidence of detachment of the sensory retina in any case. In the acute phase we could not detect choroidal fluorescence through the affected retinal pigment epithelium and we saw late staining of the lesion in only one patient. Additionally, there was evidence of a widespread abnormality of the choroidal filling pattern. On recovery large choroidal vessels were visible through the atrophic pigment epithelial scar.

When first seen, Patient 6 had a lesion at the left macula. On angiography choroidal fluorescence was not apparent beneath the pigment epithelial lesion, but there was some staining in the late films. One week later the affected area was no larger and patchy areas of fluorescence were visible as the retinal pigment epithelium became atrophic; two weeks later this atrophy was more pronounced. Two years after the initial symptoms large choroidal vessels were demonstrated in the area of the atrophy.

When first examined, Patient 7 had an area of swelling of the retinal pigment epithelium with central atrophy in the region of the superior temporal vessel arcade in the left eye. Fluorescein angiography showed no choroidal fluorescence in the area of swelling and transmission defects in the atrophic patches; there was no late staining of the swollen retinal pigment epithelium. During a period of three weeks, this lesion extended to involve the entire posterior pole; initially there was swelling and later atrophy; there was one small additional

TABLE

CLINICAL SUMMARY OF 16 CASES

Case No., Sex, Age (yrs)	Visual Acuity				Affected Eye	Duration of Retinal Detachment	Disk Leakage				Notes
	1st Examination		Recovery				1st Examination		Recovery		
	R. E.	L. E.	R. E.	L. E.			R.E.	L.E.	R. E.	L. E.	
Group 1											
1, F, 17	6/12 (20/40)	C.F.	6/5 (20/16)	3/36 (10/120)	Both	—	No	No	No	No	Angioedema
2, M, 15	6/18 (20/60)	6/6 (20/20)	6/6 (20/20)	6/9 (20/30)	Both	—	No	No	No	No	Headache
3, F, 32	6/18 (20/60)	6/9 (20/30)	6/9 (20/30)	6/6 (20/20)	Both	—	No	No	No	No	—
4, F, 38	6/60 (20/200)	6/36 (20/120)	6/18 (20/60	6/24 (20/80)	Both	—	No	No	No	No	Final visual acuity impaired by cataracts
5, M, 24	6/5 (20/16)	6/9 (20/30)	6/5 (20/16)	6/9 (20/30)	Both	—	No	No	No	No	Headache
Group 2											
6, M, 24	6/6 (20/20)	C.F.	6/6 (20/20)	6/60 (20/200)	L.E.	—	No	No	No	No	—
7, M, 51	6/5 (20/16)	6/60 (20/200)	6/5 (20/16)	H.M.	L.E.	—	No	No	No	No	—
8, M, 43	6/5 (20/16)	C.F.	6/5 (20/16)	6/36 (20/120)	L.E.	—	No	No	No	No	—
Group 3											
9, M, 34	6/18 (20/60)	6/9 (20/30)	6/6 (20/20)	6/6 (20/20)	Both	14 days	Yes	Yes	Yes	Yes	—
10, M, 34	C.F.	4/60 (13/200)	6/9 (20/30)	6/5 (20/16)	Both	12 days	Yes	Yes	No	No	Headache
11, M, 24	6/36 (20/120)	6/9 (20/30)	6/9 (20/30)	6/9 (20/30)	R.E.	14 days	Yes	Yes	Yes	Yes	Headache
12, F, 21	6/60 (20/200)	6/60 (20/200)	6/60 (20/200)	6/6 (20/20)	L.E.	10 days	No	Yes	No	No	Severe myopia in R. E.
13, F, 46	6/36 (20/120)	6/9 (20/30)	6/6 (20/20)	6/9 (20/30)	R.E.	21 days	Yes	No	Yes	No	—
14, F, 21	6/5 (20/16)	6/24 (20/80)	6/5 (20/16)	6/5 (20/16)	Both	14 days	No	No	No	No	—
15, F, 28	6/18 (20/60)	6/12 (20/40)	6/9 (20/30)	6/6 (20/20)	Both	21 days	Yes	Yes	Yes	Yes	—
16, M, 63	6/6 (20/20)	6/24 (20/80)	6/6 (20/20)	6/9 (20/30)	L.E.	14 days	Yes	Yes	Yes	Yes	—

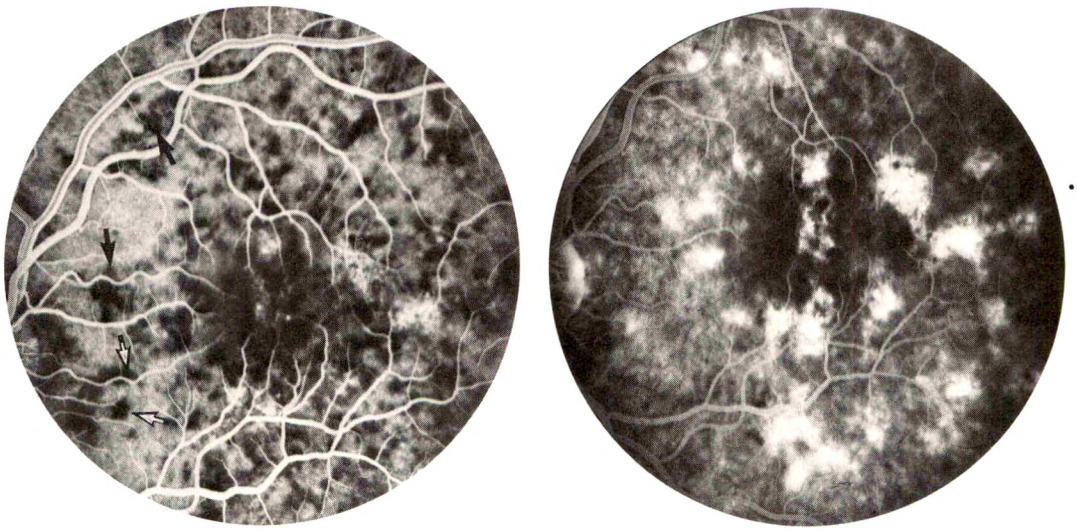


Fig. 1 (Young, Bird, and Sehmi). Case 2. Diffuse areas of choroidal hypofluorescence (left), some of which stain (solid arrows) and others merge with the background fluorescence (open arrows) in the late stages of the study (right).

atrophic area which remained unchanged throughout. Visual acuity in this eye was reduced to hand movements and there was an afferent pupillary defect. In the final angiographic study, four weeks after the first examination, fluorescence was transmitted through the atrophic retinal pigment epithelium and stained the optic disk and part of the wall of the inferotemporal vein. There has been no progression of the disease.

Patient 8 was first seen at another hospital because of an area of central retinal pigment epithelial swelling in the left eye which was documented by photography. When we examined him one week later, this lesion had enlarged. Angiography showed absence of central choroidal fluorescence at the site of the swelling except for one small island. We interpreted this as a lobule of perfused choriocapillaris within an area of infarction caused by microvascular occlusion; the patch of swelling did not stain in the late films. Elsewhere in the posterior pole of this eye, there were many small areas of delayed choriocapillaris perfusion where the retinal pigment epithelium appeared normal (Fig. 2). He began taking systemically administered corticosteroids; two weeks later the lesion had become atrophic and there was no further increase in its size. On recovery there was no longer any demonstrable abnormality of choroidal filling except in the central area.

Group 3—The eight patients in this group had serous detachments of the sensory retina in one (three cases) or both (five cases) eyes which resolved spontaneously within three weeks. Although the underlying retinal pigment epithelial changes, angiographic findings, and appearances of the optic disks showed a greater degree of polymorphism than in the previous two groups, all the patients had abnormal choroidal perfusion, suggesting that they were part of the same spectrum of disease.

In three patients (Cases 10, 11, and 16), the sensory retinal detachment was widespread and bullous, simulating Harada's disease (Fig. 3). However, only in Case 10 was visual loss as extensive as

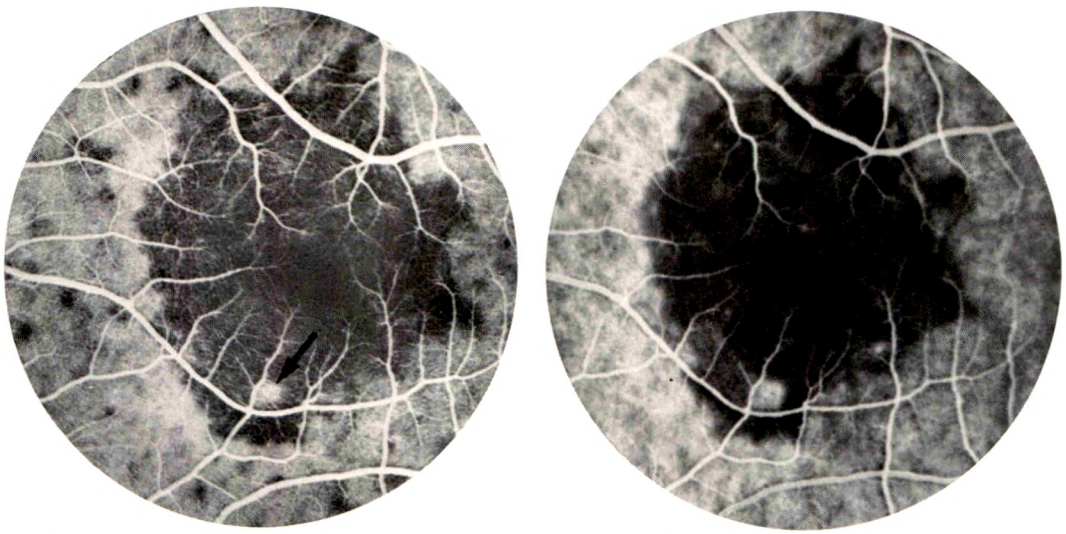


Fig. 2 (Young, Bird, and Sehmi). Case 8. Left, Central choroidal nonperfusion with sparing of one small lobule (arrow). Right, The surrounding choroid fills slowly.

expected in this condition. One patient (Case 9) had a deep detachment in one eye and a more shallow, localized detachment in the other. The other four patients had shallow retinal detachments; the visual acuity in four of the six eyes with shallow detachment was less than 6/12 (20/40) (Figs. 4 and 5).

On fluorescein angiography, there was delayed dye entry and prolonged transit in the choroid in all patients with retinal detachments; in six eyes (Cases 9 to 13 and 15) these findings appeared after the retinal phase had begun. Dye leaked into the subretinal space from large spots (Cases 9, 10, 14, and 16) (Fig. 3), small spots (Cases 12 and 15) (Fig. 4), and both large and small spots (Cases 11 and 13) (Fig. 5).

Of the four patients with unilateral retinal detachments, there was delayed perfusion of the choroid in the fellow eye

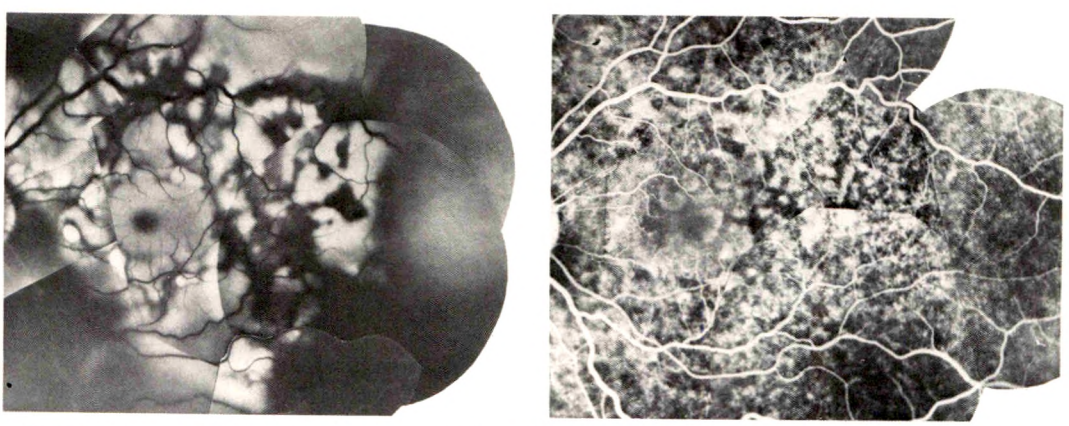


Fig. 3 (Young, Bird, and Sehmi). Case 10. Left, Bullous retinal detachment. Right, Widespread abnormalities of the retinal pigment epithelium remain after recovery.

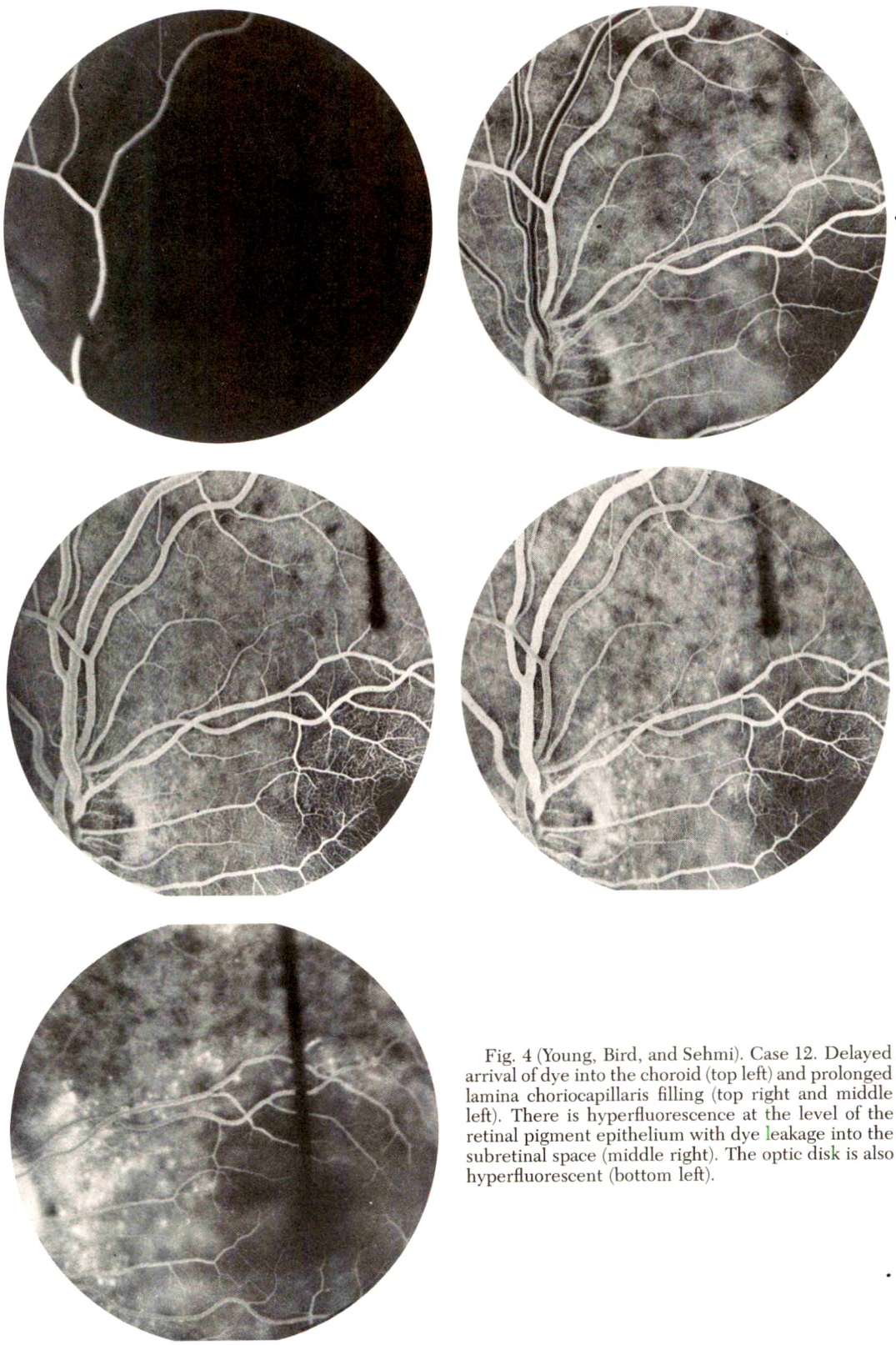


Fig. 4 (Young, Bird, and Sehmi). Case 12. Delayed arrival of dye into the choroid (top left) and prolonged lamina choriocapillaris filling (top right and middle left). There is hyperfluorescence at the level of the retinal pigment epithelium with dye leakage into the subretinal space (middle right). The optic disk is also hyperfluorescent (bottom left).

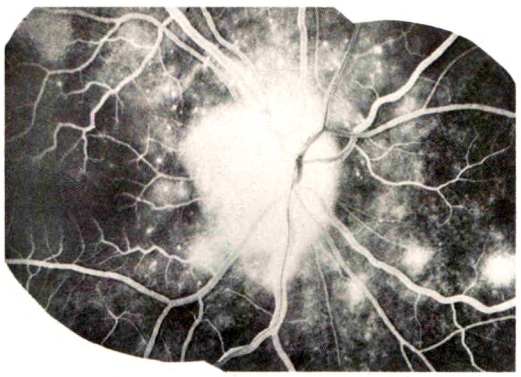


Fig. 5 (Young, Bird, and Sehmi). Case 13. Dye leakage through the retinal pigment epithelium in small and large spots with hyperfluorescence of the optic disk.

in two (Cases 11 and 16) (Fig. 6); in Case 13 there were no early pictures of the fellow eye and in Case 12 the fellow eye was severely myopic, making comparison impossible. In the late films there was hyperfluorescence of the optic disk in six patients (Cases 9 to 11, 13, 15, and 16), including both eyes of the patients with unilateral detachments (Figs. 4 to 6).

In all cases the retina flattened spontaneously within three weeks with full recovery of visual function. All patients except Patient 16 had diffuse mottling of the retinal pigment epithelium, visible in some only on fluorescein angiography (Fig. 3); Patient 16 had well-defined scars at the level of the retinal pigment epithelium resembling the changes seen in Group 1 (Fig. 7). There was prolonged choroidal filling in many patients during the recovery period. Hyperfluorescence of the optic disk was still a feature in four patients (Cases 9, 13, 15, and 16) after resolution of the detachment.

Discussion

The variety of fundus changes described may be related to different pathogenic mechanisms, but we believe that the diseases have in common obstruction of the choroidal blood flow that produces diffuse ischemia and focal infarction of the retinal pigment epithelium, as originally proposed by Van Buskirk, Lessell, and Friedman[7] in acute posterior multifocal placoid pigment epitheliopathy. The concept of focal choroidal infarction would have been difficult to defend at a time when anatomical studies implied that the choriocapillaris was a continuous network of capillaries supplied by many arterioles.[17,18] However, Dollery and associates[19] showed that the choriocapillaris in pigs with increased intraocular pressure filled not as a continuous layer, but as a series of dots which later enlarged to become uniform; they concluded that this filling pattern indicated the presence of individual choriocapillaris units. Hayreh[20] demonstrated the lobular pattern of choriocapillaris filling in the posterior pole of normal animals and other injection studies[21-23] have supported the conclusion that lobules of choriocapillaris in the submacular choroid are supplied by short, nonanastomosing precapillary arterioles without functional anastomoses between adjacent capillary units in the normal eye. This lobular arrangement is less well defined in the anterior choroid.

Further information about the choroidal circulation has been derived from experimental occlusion of vessels at different levels.[22,24,25] Hayreh and Baines[24] found that occlusion of the lateral short posterior ciliary arteries in the orbit resulted in nonperfusion of the temporal half of the choroid with a watershed at the optic disk. The disturbance of blood supply was sufficient to produce infarcts of the pigment epithelium and outer retina which appeared as large oval patches in the posterior fundus or as triangular areas more peripherally; these areas stained with fluorescein late in the study and became atrophic during a period of two to three weeks.[8] Within two weeks of the occlusion, the temporal choroid filled but the filling was delayed;

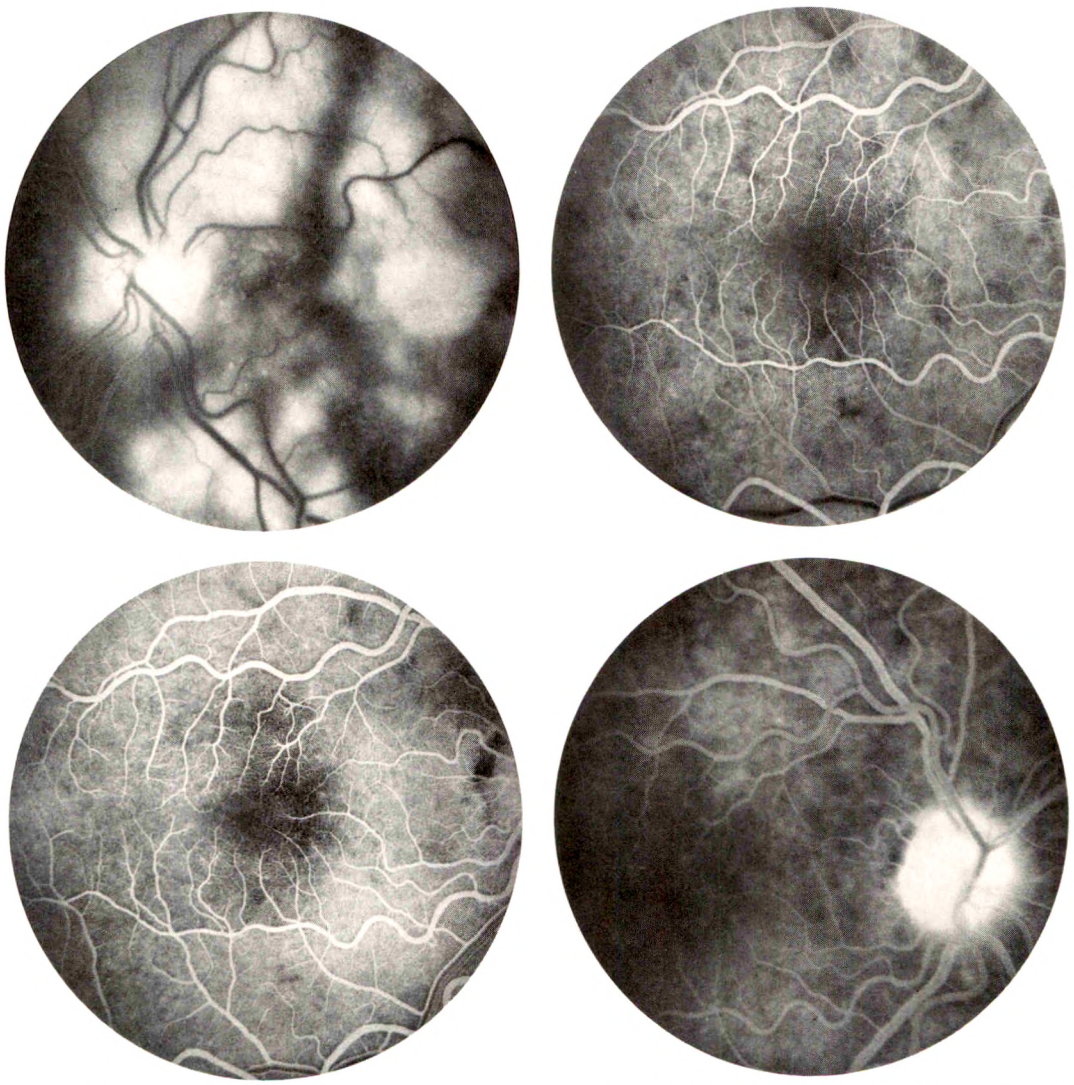


Fig. 6 (Young, Bird, and Sehmi). Case 16. Bullous retinal detachment and optic disk hyperfluorescence in the left eye (top left). The right eye appears normal but fluorescein angiography demonstrated delayed filling of the lamina choriocapillaris (top right and bottom left) with late hyperfluorescence of the optic disk (bottom right).

there was still some delay after three months. Their work demonstrated the presence of potential anastomotic channels which rapidly became functional after an acute arterial occlusion. Ernest, Stern, and Archer[22] have shown that this may occur more rapidly if the venous drainage of the choroid is also compromised.

Stern and Ernest[25] also identified changes in the retinal pigment epithelium and overlying retina after embolic occlusion of vessels within the choroid. Injection of embolic microspheres into the lateral short posterior ciliary arteries resulted in delayed choroidal filling with spotty leakage through the retinal pigment epithelium. Histologic studies

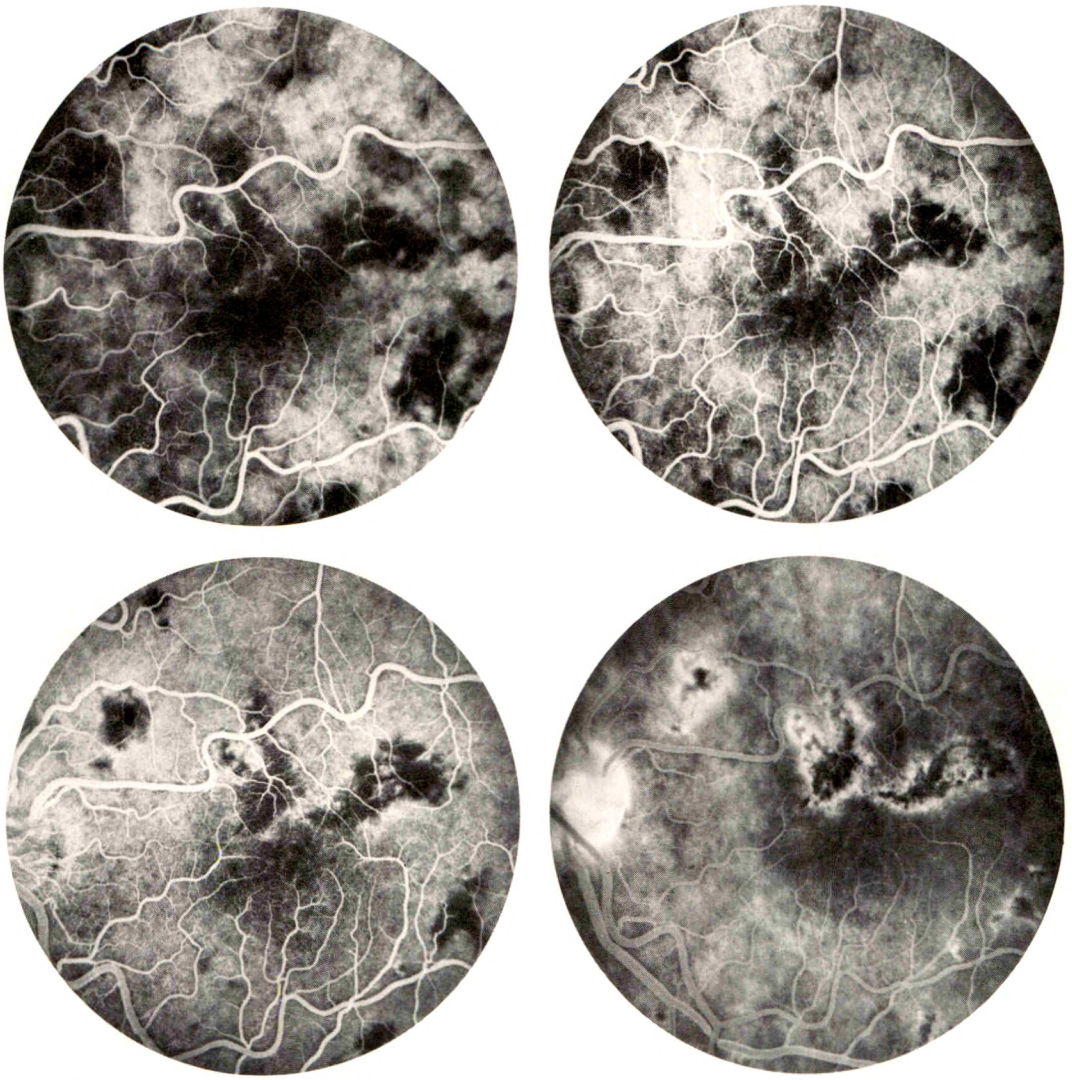


Fig. 7 (Young, Bird, and Sehmi). Case 16. Two weeks later, the retina is flat but delayed choroidal filling is still present in the left eye (top left, top right, and bottom left). Focal pigment epithelial changes result in discrete scars one month later (bottom right).

showed focal necrosis of the pigment epithelium and a concentration of microspheres at the posterior pole. Within four days, the whole choroid filled rapidly and simultaneously and by two months the only detectable angiographic abnormality was hyperfluorescence through the small atrophic pigment epithelial defects. Retinal detachment accompanied these changes in the acute phase in animals with experimentally induced hypertension. The pattern of pigment epithelial infarction contrasted with that seen after occlusion of the short posterior ciliary arteries in that it was multifocal and was seen only at the macula, despite the widespread distribution of microspheres.

These experimental observations have their clinical counterparts. Amalric[26,27] and Foulds, Lee, and Taylor[28] described

triangular lesions of the retinal pigment epithelium in the peripheral fundus and ascribed them to occlusion of a short ciliary artery. Small multifocal infarcts similar to those reported by Stern and Ernest[25] are found in accelerated hypertension,[29-31] giant cell arteritis,[28] Goodpasture's syndrome,[32] disseminated intravascular coagulopathy,[33] and, rarely, sickle cell disease,[34] all of which are known or presumed to be caused by occlusion of the choroidal precapillary arterioles. In these conditions there is diffuse choroidal vascular disease which produces widespread arteriolar obstruction but only focal infarcts.

These observations have suggested that the primary disorder in acute posterior multifocal placoid pigment epitheliopathy exists at the level of the precapillary arteriole[4,8-12] but, in view of the diffuse abnormality of choroidal flow, the possibility of arterial and venous occlusion cannot be discounted.

In Group 1, the size of the swollen areas may be equivalent to a choriocapillaris lobule and it is possible that these lesions are related to a single nonperfused lobule as previously postulated by Hayreh and Baines,[8] although angiography showed diffusely abnormal choroidal flow.

In Group 2, the presence of a single confluent plaque of infarction could be ascribed to occlusion of a specific submacular vessel. However, the presence of such a vessel is disputed[17,20,23,35,36] and the enlargement of the lesion in two cases, the diffuse abnormality of choroidal perfusion beyond the borders of the presumed infarct, and the hyperfluorescence of the optic disk demonstrated the widespread nature of the disorder.

The disease in Group 3 differed from those in the other groups in many respects. The presence of serous detachments was the most obvious distinguishing feature, but many patients in Group 3 had optic disk hyperfluorescence and focal scarring of the pigment epithelium was seen in only one case. The disorder in these patients was similar to Harada's disease clinically and angiographically both early in the disease and on recovery, but the progression differed markedly. In Group 3 the apparent delay of dye entry may have been caused by masking of the choroidal fluorescence by subretinal fluid in those patients with deep retinal detachments, but it was also seen in eyes with shallow detachments and abnormal choroidal perfusion was still present when the detachment had resolved. Furthermore, in two patients with unilateral disease, there was abnormal choroidal flow in the apparently normal fellow eye.

The evidence indicates that patients in Group 3 had decreased choroidal perfusion, but the lack of focal scarring suggests that the manifest disease is a product of ischemic dysfunction rather than frank infarction of the retinal pigment epithelium. It is also clear that patients with unilateral fundus changes had bilateral choroidal perfusion deficits presumably insufficient to interfere with pigment epithelial function in the fellow eye. Only one patient had features of both Groups 1 and 3 (Case 16); at first examination there was bullous retinal detachment but within one week the fundus changes were indistinguishable from those of acute posterior multifocal placoid pigment epithiopathy. On recovery the patient had well-defined retinal pigment epithelial scars in the posterior pole. The explanation for the difference in response between Groups 1 and 2 on the one hand and Group 3 on the other is not apparent.

Despite the variety of fundus appearances, there appears to be sufficient evidence in our patients and in previous reports to indicate that the manifest disorders are produced by intrinsic choroidal vascular disease. The polymor-

phism may be caused by variations in the rapidity, completeness, and site and extent of the obstruction, which in turn may be determined by the nature of the basic disorder causing the obstruction.

Summary

Sixteen patients, seen during the first week of their disease, had swelling of the retinal pigment epithelium and angiographic evidence of a widespread abnormality of choroidal filling. The patients fell into three groups: In Group 1 there were multifocal pigment epithelial lesions identical to those of acute posterior multifocal placoid pigment epitheliopathy. In Group 2 the pigment epithelial lesions were confluent. The retina was not detached in either of these groups. In Group 3, multifocal lesions of the pigment epithelium were associated with retinal detachment. We postulated that all patients had diffuse choroidal hypoperfusion and focal pigment epithelial infarcts which, in turn, may have been caused by a variety of basic disorders. We detected no relevant systemic abnormalities in any of the patients.

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SHORT POSTERIOR CILIARY ARTERY INSUFFICIENCY WITH HYPERTHERMIA (UHTHOFF'S SYMPTOM)

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Uhthoff's symptom,[1] the reduction of visual acuity with exercise or hyperthermia, is associated with multiple sclerosis.[2,3] Nelson, Jeffreys, and McDowell[4] stated that this symptom occasionally occurs in association with other neurologic diseases such as intrasellar and parasellar tumor, Friedreich's ataxia, and insufficiency of the posterior cerebral arteries. However, in the last-mentioned case, they did not describe how the diagnosis of posterior cerebral artery insufficiency was established.

To our knowledge, Uhthoff's symptom has not been previously reported in cases of documented vascular disease. We treated two patients with Uhthoff's symptom who had arterial rather than neurologic disease. Although one had giant-cell arteritis and the other total carotid occlusion, they each had compromised short posterior ciliary artery circulation.

CASE REPORTS

Case 1—On Feb. 2, 1979, a 42-year-old man experienced an episode of decreased visual acuity and pain in his left eye as he left a health spa where he had been vigorously exercising. Later that day, he consulted an ophthalmologist. The patient's visual acuity in the left eye was 6/6 (20/20); color vision was poor in the left eye and there was an afferent pupillary defect, but his central visual fields were full to the 2/1,000 white isopter. When he was reexamined by the same ophthalmologist two days later, visual acuity and color vision in the left eye were essentially normal, but the afferent pupillary defect persisted.

Because his ophthalmologist thought the patient had Uhthoff's symptom, and thus multiple sclerosis, the patient was referred to the neuro-ophthalmology service here on Feb. 7. We learned that he had had five episodes of inferior visual field loss in the left eye over the preceding two years; he recalled that these episodes had occurred when he turned his head to the left.

His visual acuity was 6/6 (20/20) in each eye; there was an afferent pupillary defect in the left eye. The central and peripheral visual fields were normal. Intraocular pressure was R.E.: 18 mm Hg and L.E.: 13 mm Hg. The cup:disk ratio was 0.2 in the right eye and the retinal veins were normal; the left optic disk was flat and the retinal veins were dilated.

Neurologic findings were unremarkable. The carotid arteries had good, equal pulses without bruits.

Three abnormalities were apparent on fluorescein angiography of the left eye: generally delayed choroidal circulation, shown by the filling of the retinal arterioles before the choroid; patchy choroidal filling defects; and inadequate perfusion of the temporal aspect of the optic disk (Figs. 1 and 2).

Other laboratory results included normal values

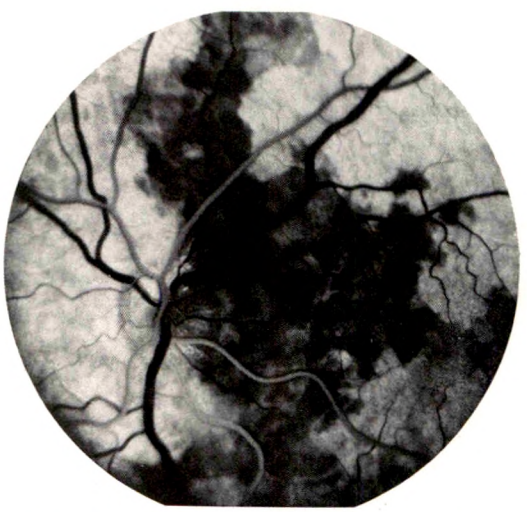


Fig. 1 (Raymond and associates). Case 1, left eye. Fluorescein angiogram shows filling defects in choroid and temporal portion of left optic disk.

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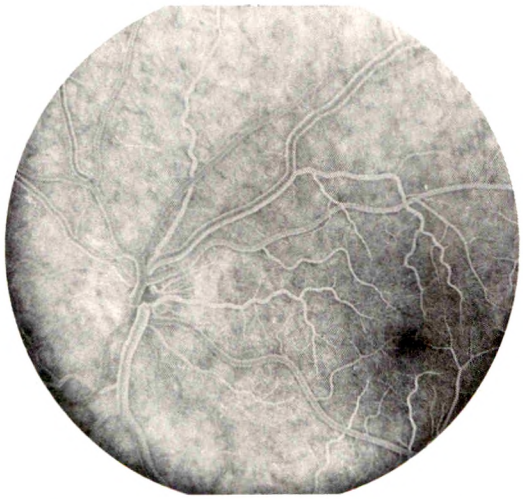


Fig. 2 (Raymond and associates). Case 1, left eye. Fluorescein angiogram at 46 seconds demonstrates that the filling defects have disappeared. A sluggish retinal circulation still shows laminar filling of the veins.

for cholesterol, triglycerides, erythrocyte sedimentation rate, VDRL, and FTA-ABS tests. A glucose tolerance test showed a slightly increased value.

Cerebral arteriography showed total occlusion of the left internal carotid artery beginning about 2 cm above the carotid bifurcation; it was again patent above the ophthalmic artery. The external carotid and middle meningeal arteries filled well. The left ophthalmic artery was supplied by muscular orbital branches and lacrimal branches; it thus filled in a retrograde fashion and, in turn, supplied the intracranial portion of the internal carotid artery distal to the occlusion (Fig. 3). The patient began taking an anticoagulant. Despite this, visual symptoms persisted.

In July 1979, a left superficial temporal-middle cerebral artery branch anastomosis[5] was performed. Postoperative arteriography demonstrated a patent anastomotic channel (Fig. 4). The patient has had no visual symptoms since surgery.

Case 2—In February 1979, a 72-year-old woman experienced a sensation of "gloominess" in her right eye while she was taking a hot bath. One week later she noted painless fatigue of the masseter muscles when she chewed. She had had headaches only rarely, but she experienced several in the two weeks

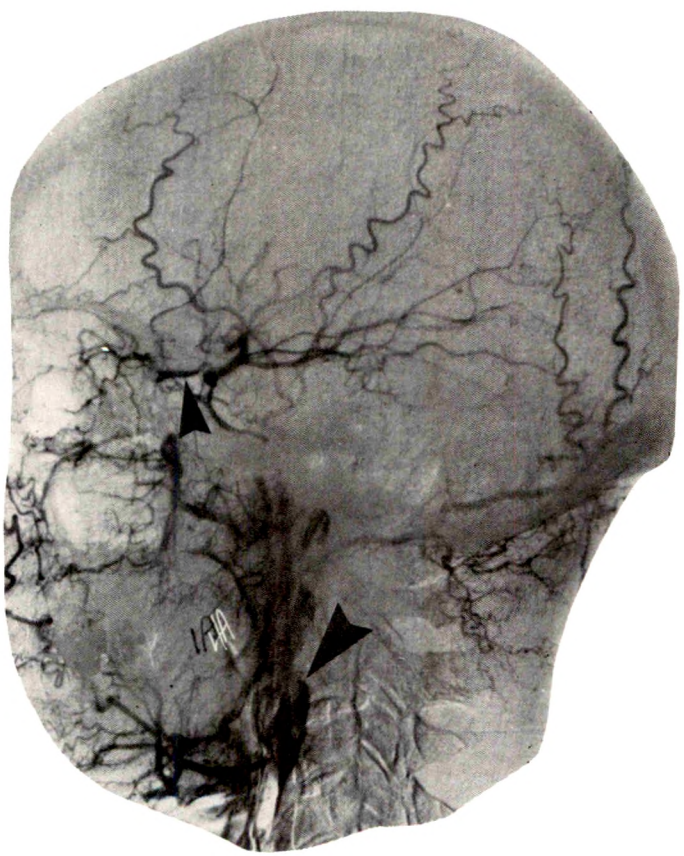


Fig. 3 (Raymond and associates). Case 1. Occlusion of left internal carotid artery (large arrow). The ophthalmic artery (small arrow) fills in a retrograde fashion by way of its orbital branches.

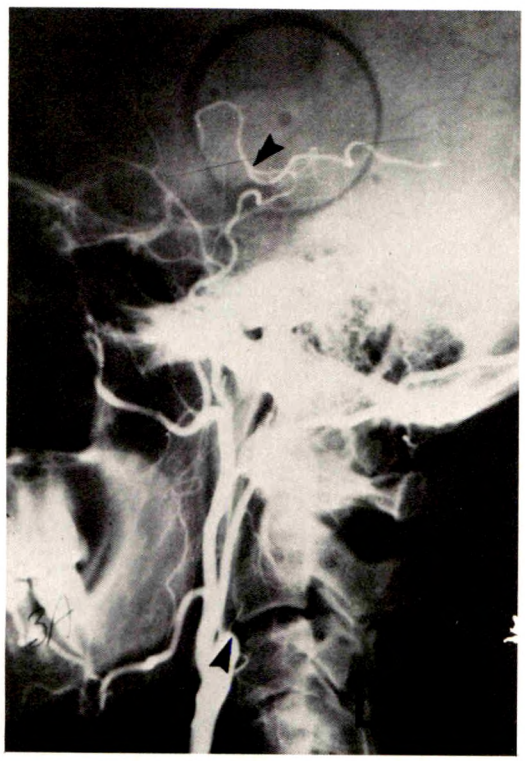


Fig. 4 (Raymond and associates). Case 1. Left carotid arteriogram performed six days postoperatively. Lower arrow points to the complete occlusion of the internal carotid artery; upper arrow shows the site of superficial temporal-middle cerebral artery branch anastomosis.

before admission here. Three days before admission she developed sudden, permanent loss of visual acuity in the right eye. She had no symptoms of anorexia, weakness, malaise, fever, or scalp tenderness.

Visual acuity was R.E.: hand motions and L.E.: 6/6 (20/20). There was an afferent pupillary defect in the right eye. The visual field of the left eye was full to the 1/1,000 white isopter and normal centrally for a red target. Intraocular pressure was R.E.: 13 mm Hg and L.E.: 12 mm Hg. The optic disk of the right eye had pallid edema extending beyond the disk edge with several hemorrhages on the disk surface. The optic disk of the left eye was normal.

Results of a neurologic examination were normal. The carotid arteries had good, equal pulses and were without bruits. Laboratory findings included a zeta sedimentation rate of 56% (upper normal limit, 54%), a Westergren erythrocyte sedimentation rate[6] of 64 mm/hr, hematocrit reading of 37.3%, and normal serum glucose values. There were no abnormalities on cerebral arteriography.

Fluorescein angiography showed filling defects in the choroidal circulation and the temporal portion of the right optic disk; the retinal circulation appeared before the choroidal circulation (Fig. 5). Late frames showed fluorescein leakage at the right optic disk. Two days after admission she developed small hemorrhages on the inferotemporal surface of the left optic disk.

A 5-cm segment[7] of the right superficial temporal artery was removed. This vessel was tortuous and irregular. Histologic evaluation showed both round-cell infiltration and giant cells in the media of the vessel wall with fragmentation of the internal elastic lamina, confirming the clinical diagnosis of cranial arteritis. She began taking 100 mg of prednisone a day.

Discussion

Although both of our patients had Uhthoff's symptom, a complaint associated with an intrinsic disease of the central nervous system, neither were so afflicted. Rather, they each had short posterior ciliary artery insufficiency. Patient 1 had a chronic form and Patient 2 an acute form, also called ischemic optic neuropathy.[8]

The fluorescein angiogram showed two kinds of evidence for ischemia: the choroidal circulation filled with dye after the retinal circulation (a reversal of the nor-

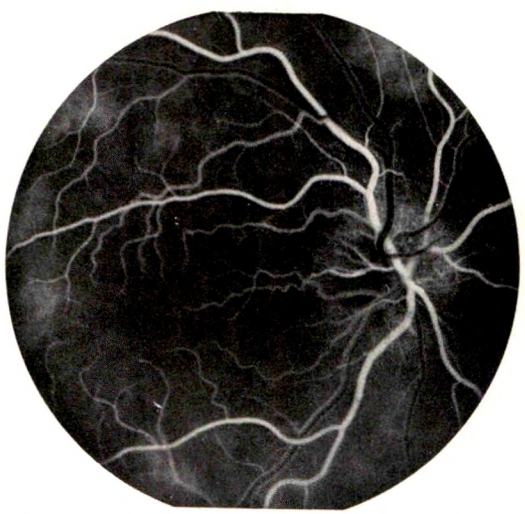


Fig. 5 (Raymond and associates). Case 2, right eye. Fluorescein angiogram shows filling defects in choroid and temporal aspect of optic disk.

mal sequence) and there were large filling defects in the choroid and optic disk.

Our patients showed that there are at least two causes of short posterior ciliary artery insufficiency; there are probably more. Patient 1 had retrograde flow in the ophthalmic artery and, therefore, tenuous perfusion of the choroidal and retinal circulations. The monocular visual blur in association with exercise probably resulted from decreased ocular perfusion when blood was shunted to the skin and muscles.[9,10] The patient's other transient visual symptoms, occurring when he turned his head to the left, were probably caused by compression of the intact right internal carotid artery by the contracting right sternocleidomastoid muscle.

In Case 2, thickening of the vessel wall of the short posterior ciliary arteries compromised blood flow. Cranial arteritis narrows the lumina of these vessels before actual occlusion.[11,12] The peripheral vasodilatation caused by her hot bath decreased her blood pressure enough to cause transient disk and choroidal ischemia.

The short posterior ciliary artery circulation appears especially vulnerable to ischemia. It supplies the optic disk.[13-15] Its branches are large-caliber, carry a heavy flow,[16] and abruptly enter the choriocapillaris adjacent to the optic disk.[17] Unlike retinal and cerebral vessels, ciliary vessels are probably not self-regulated.[18] Thus, they would not maintain a constant blood flow during a reduction in blood pressure or increase blood flow during hypoxia. These factors may explain Uhthoff's symptom in our two patients.

Decreased perfusion or occlusion of the posterior ciliary arteries deprives axons of oxygen and nutrients at the level of the optic disk[19] but does not kill ganglion cells or proximal axon segments within the inner retina. In Patient 2, continuing orthograde axoplasmic transport, or flow, resulted in an accumulation of axoplasmic debris at the border of the posterior ciliary territory, causing pallid swelling of the optic disk (acute ischemic optic neuropathy).[20]

Summary

Transient visual blurring with heat or exercise (Uhthoff's symptom) is associated with multiple sclerosis. To our knowledge, this is the first report of its occurrence in cases of documented vascular disease.

Two patients had insufficiency of the short posterior ciliary circulation. In one it was caused by a carotid occlusion and in the other by cranial (giant-cell, temporal) arteritis. The monocular blurring may have occurred during periods of relative arterial hypotension.

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Ophthalmic miniature

It is curious enough that iritis has actually been ascribed to mercury, as a cause. This notion can only have arisen from that loose kind of logic, and hasty generalization, for which, I am sorry to say, medical reasoners are too often distinguished. Mercury is perpetually exhibited for the cure of syphilis; and people who have been treated for syphilis are very liable to iritis. This seems to be the only foundation for the opinion in question. When we come to appeal to facts, we find no ground for believing that this mineral is thus, both bane and antidote. If it were so, Benvolio's advice to the slighted Romeo might be very pertinently offered to the patient in such a case:

"Take thou some new infection to thine eye,
And the rank poison of the old will die."

London Medical Gazette 27:706, 1841

TALC RETINOPATHY

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In 1972 AtLee[1] first described talc retinopathy in 17 drug addicts who had injected methylphenidate HCl (Ritalin) intravenously. The talc particles appeared as tiny glistening crystals concentrated along the small end arterioles of the perifoveal arcade in the posterior pole. Murphy and associates[2] also observed talc particles in the retinal vessels of nine out of 17 addicts who had injected suspensions of methadone HCl tablets intravenously. Isolated foreign body granuloma of the retina,[3] irreversible macular ischemia,[4] peripheral retinal neovascularization,[5,6] and optic disk neovascularization[6] have been reported as complications of probable talc microembolization from chronic intravenous injection of either powder or pulverized tablets.

We describe herein 23 patients with bilateral intraretinal talc emboli. Abnormalities of the posterior pole were noted in all of these cases. Additionally, six of these 23 patients had peripheral retinal neovascularization associated with peripheral retinal vascular nonperfusion. Although two single occurrences of peripheral retinal neovascularization secondary to talc[5,6] have been described, to the best of our knowledge this is the first report of a series of patients with talc retinopathy that shows the progressive fundus changes seen in the proliferative retinopathies and that documents these changes with fluorescein angiography. The fundus findings show a remarkable similarity to each of the progressive stages of sickle-cell retinopathy and fit the Goldberg[7] classification of proliferative sickle-cell retinopathy. Of these six patients with peripheral retinal neovascularization, two had optic disk neovascularization and traction retinal detachment and three had vitreous hemorrhage. One patient underwent bilateral pars plana vitrectomy and another patient underwent unilateral pars plana vitrectomy for nonabsorbing vitreous hemorrhage. An additional patient had argon laser panretinal photocoagulation for peripheral retinal neovascularization and secondary vitreous hemorrhage in one eye.

MATERIAL AND METHODS

We reviewed the records of patients seen here between January 1973 and December 1977 and found 85 patients with a discharge diagnosis of intravenous methylphenidate HCl drug abuse. We contacted 23 (27%) of these patients; 15 (65%) agreed to have an ocular examination. The eight patients who did not comply cited their lack of visual complaints and the discontinuation of their drug habit as the main reasons for declining the examination. Thirteen additional cases were obtained from friends or family members of this group, from inpatient consultations, and from patients referred directly to us. A total of 28 patients with a history of intravenous methylphenidate HCl drug abuse were examined, of whom 23 had fundus involvement; 14 were men, ages 26 to 51 years, and nine were women, ages 28 to 52 years. The mean age was 39 years and the median age was 38 years. Twenty-one patients were black and two

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were white. A thorough drug abuse history was obtained, including duration of abuse, average number of tablets per injection, and frequency and method of injection. Cumulative numbers of tablets injected were then calculated from these data.

Ophthalmoscopic examination included visual acuity, refraction, slit-lamp examination, applanation tonometry, and fundus examination using direct and indirect ophthalmoscopy and the Goldmann three-mirror contact lens. Tangent screen visual fields were performed on all patients. Fundus photography and fluorescein angiography were obtained in 21 of the 23 patients with talc retinopathy. One patient declined the procedure and the other had sclerotic peripheral veins that precluded fluorescein injection intravenously. The number of intraretinal talc particles in the posterior pole was graded on a scale from 0 to 4 by careful analysis of stereoscopic fundus photographs taken at 2× magnification. Blood pressure was measured in all patients. Twenty patients underwent a complete medical examination, which included general physical examination, pulmonary function tests, and chest X-ray. Hemoglobin electrophoresis, complete blood cell count, intermediate strength purified protein derivative (PPD) skin testing, chest X-ray, angiotensin-converting enzyme, and two-hour postprandial glucose were performed on the six patients with retinal neovascularization to exclude other potential causes of proliferative retinopathy such as diabetes mellitus,[8] sickle-cell hemoglobinopathies,[7,9] leukemia,[10,11] sarcoidosis,[12,13] and polycythemia.[14]

Results

Of the 28 patients with a history of intravenous methylphenidate HCl drug abuse, 23 (82%) had talc retinopathy. Most of the patients in this series were chronic, long-term drug abusers, and multi-drug abuse was common. Although the authenticity of their drug habit cannot be verified or quantitated with certainty, most of these patients admitted to injecting an average of 20 to 35 tablets of methylphenidate HCl daily for one to ten years (Table 1). The average cumulative number of tablets injected ranged from 720 to 280,000 tablets.

The patients were divided into three groups (Table 1). Group 1 included 11 patients (Cases 1 to 11) who showed evidence of Grade 2 or less (Fig. 1) intraretinal talc particles in one or both fundi and whose visual acuity was 6/9 (20/30) or better in each eye. None of these patients had visual symptoms, nor was there any fluorescein angiographic evidence of retinal or choroidal vascular abnormalities.

Group 2 included 12 patients (Cases 12 to 23) who showed evidence of Grade 2 or more (Fig. 2) intraretinal talc particles in both fundi and additional ophthalmoscopic and fluorescein angiographic abnormalities of the fundus (Table 2). Five of these patients in Group 2 had reduced visual acuity of 6/12 (20/40) or worse in one or both eyes. Patient 12 had normal vision but also bilateral pigmentary mottling in the macula. Fluorescein angiography showed small hyperfluorescent dots corresponding to the pigment epithelial abnormalities. This patient was hospitalized with an acute left hemiparesis that was presumably secondary to cerebral talc embolization. The predominant fundus abnormalities in Cases 13 to 17 consisted of perifoveal capillary abnormalities, including perifoveal capillary nonperfusion, enlargement of the foveal avascular zone, closure and enlarged terminal segments of arterioles, venular loops, and microaneurysms. Additionally, four of these patients (Cases 13 to 15, and 17) showed peripheral retinal vessel nonperfusion. Patient 13 also showed a segmental area of atrophy and hyperpigmenta-

TABLE 1

DRUG ABUSE DATA AND CLINICAL CORRELATION IN PATIENTS WHO INJECTED METHYLPHENIDATE HCL (RITALIN)*

Case No., Sex, Age (yrs), Race	Duration (mos)	Culmulative No. of Tablets	Method of Injection	Visual Acuity		Talc Particles (Grade 0-4)	
				R.E.	L.E.	R.E.	L.E.
			Group 1				
1, F, 28, B	12	4,800	w	6/7.5 (20/25)	Enucleated	1	0
2, M, 48, B	24	36,000	w	6/9 (20/30)	6/9 (20/30)	1	1
3, F, 31, W	24	16,000	w, w/o	6/7.5 (20/25)	6/7.5 (20/25)	1	1
4, F, 50, B	24	10,000	w	6/7.5 (20/25)	6/7.5 (20/25)	1	1
5, F, 36, B	12	5,400	w, w/o	6/6 (20/20)	6/6 (20/20)	1	1
6, F, 31, B	36	54,000	w, w/o	6/7.5 (20/25)	6/7.5 (20/25)	2	1
7, M, 31, B	30	15,000	w, w/o	6/9 (20/30)	6/9 (20/30)	1	2
8, F, 32, B	24	45,000	w, w/o	6/7.5 (20/25)	6/7.5 (20/25)	2	2
9, F, 52, B	48	30,000	w, w/o	6/9 (20/30)	6/9 (20/30)	2	2
10, F, 36, B	12	3,500	w/o	6/7.5 (20/25)	6/7.5 (20/25)	2	2
11, F, 30, B	12	11,000	w	6/6 (20/20)	6/6 (20/20)	2	2
			Group 2				
12, M, 39, B	16	10,000	w	6/6 (20/20)	6/6 (20/20)	2	2
13, M, 51, W	120	162,000	w, w/o	6/9 (20/30)	6/9 (20/30)	3	3
14, M, 40, B	12	4,000	w/o	6/12 (20/40)	6/18 (20/60)	2	2
15, M, 40, B	24	20,000	w, w/o	6/6 (20/20)	6/6 (20/20)	2	2

16, M, 35, B	36	8,400	w, w/o	6/30 (20/100)	6/24 (20/80)	2	2
17, M, 39, B	36	9,700	w, w/o	6/30 (20/100)	6/12 (20/40)	4	4
18, M, 45, B	24	13,000	w/o	6/7.5 (20/25)	6/7.5 (20/25)	3	3
19, M, 48, B	48	280,000	sc, w/o	6/9 (20/30)	6/9 (20/30)	4	4
20, M, 41, B	24	10,800	w/o	6/9 (20/30)	6/9 (20/30)	3	3
21, M, 40, B	36	20,000	w, w/o	6/6 (20/20)	6/6 (20/20)	3	3
22, M, 26, B	24	8,600	w, w/o	6/6 (20/20)	6/30 (20/100)	3	3
23, M, 32, B	36	98,000	w, w/o	HM	6/120 (20/400)	3	3
			Group 3				
24, F, 21, B	6	720	w	6/6 (20/20)	6/6 (20/20)	0	0
25, F, 21, B	6	780	w	6/6 (20/20)	6/6 (20/20)	0	0
26, M, 32, B	6	720	w	6/6 (20/20)	6/6 (20/20)	0	0
27, M, 36, B	10	1,000	w	6/6 (20/20)	6/6 (20/20)	0	0
28, M, 41, B	8	800	w, w/o	6/7.5 (20/25)	6/7.5 (20/25)	0	0

*B designates black; W, white; HM, hand movements; w or w/o designates with or without filtration of drug suspension; sc, subcutaneous injection after all veins became sclerotic.

TABLE 2

FUNDUS FINDINGS IN PATIENTS (GROUP 2)* WHO INJECTED METHYLPHENIDATE HCL (RITALIN)†

Case No.	Eye.	Perifoveal Capillary Abnormalities‡	Peripheral Vessel Closure	Arteriolar-Venular Anastomosis	PRN	Vitreous Hemorrhage	Traction RD	NVD	Remarks
12	R.E.	−	−	−	−	−	−	−	Retinal pigment epithelial defects in both eyes
	L.E.	−	−	−	−	−	−	−	
13	R.E.	+	+	−	−	−	−	−	Presumed choroidal infarct in right eye
	L.E.	+	+	−	−	−	−	−	
14	R.E.	+	+	−	−	−	−	−	
	L.E.	+	+	−	−	−	−	−	
15	R.E.	+	+	−	−	−	−	−	
	L.E.	+	+	−	−	−	−	−	
16	R.E.	+	−	−	−	−	−	−	Flame-shaped hemorrhages and cotton-wool spots in both eyes
	L.E.	+	−	−	−	−	−	−	
17	R.E.	+	+	−	−	−	−	−	
	L.E.	+	+	−	−	−	−	−	
18	R.E.	+	+	+	+	−	−	−	
	L.E.	+	+	+	+	−	−	−	
19	R.E.	+	+	+	+	−	−	−	
	L.E.	+	+	+	+	−	−	−	
20	R.E.	+	+	+	+	−	−	−	
	L.E.	+	+	+	+	−	−	−	
21	R.E.	+	+	+	+	+	−	−	
	L.E.	+	+	−	−	−	−	−	
22	R.E.	+	+	+	+	+	−	−	
	L.E.	+	+	+	+	+	+	+	
23	R.E.	+	+	+	+	+	−	+	
	L.E.	+	+	+	+	+	+	+	
No. eyes involved (Total: 45)		22 (49%)	20 (44%)	11 (24%)	11 (24%)	5 (11%)	2 (4.4%)	3 (6.7%)	

*All patients in this group had intraretinal talc particles.

†PRN designates peripheral retinal neovascularization; NVD, optic disk neovascularization; RD, retinal detachment; +, present; −, absent.

‡Perifoveal capillary abnormalities consisted of perifoveal capillary nonperfusion, enlargement of foveal avascular zone, closure and enlarged terminal segments of arterioles, venular hairpin loops, microaneurysms.

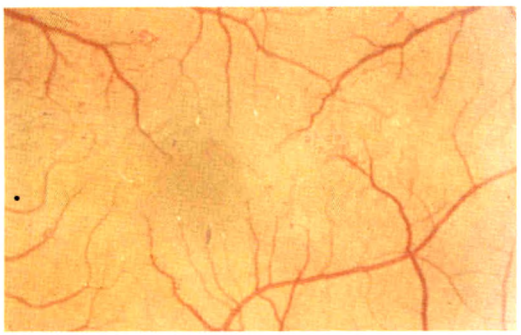


Fig. 1 (Tse and Ober). Fundus photograph showing characteristic location and appearance of intraretinal talc particles in the macula (Grade 2).

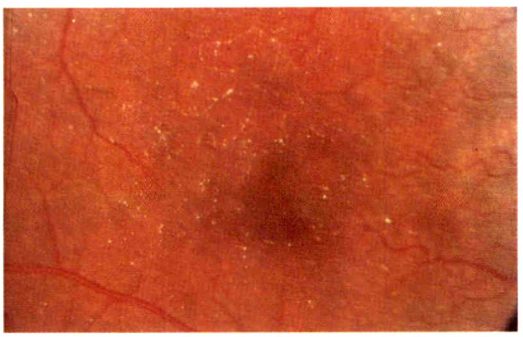


Fig. 2 (Tse and Ober). Fundus photograph showing Grade 4 talc retinopathy.

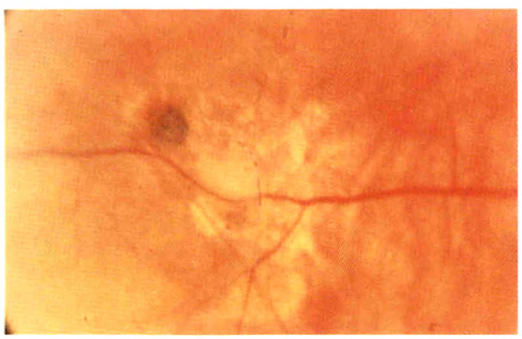


Fig. 3 (Tse and Ober). Case 13. Fundus photograph showing segmental area of subretinal pigment alteration in superotemporal quadrant of right eye, which may represent healed choroidal infarct.

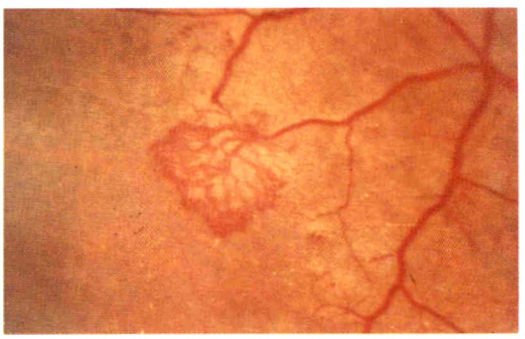


Fig. 6 (Tse and Ober). Case 20. Fundus photograph of neovascular sea-fan in temporal periphery of right eye. Peripheral to the neovascularization is the zone of avascular retina.

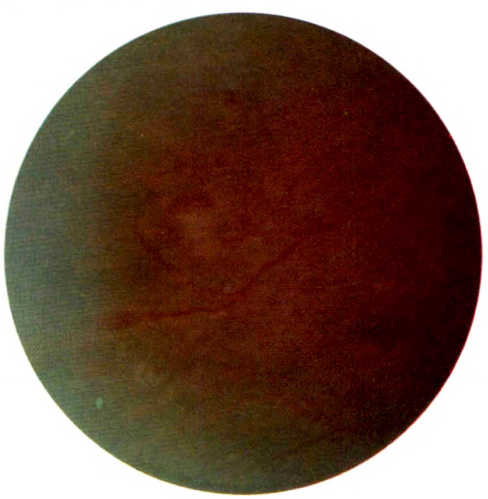


Fig. 11 (Tse and Ober). Case 22. Fundus photograph of right eye showing peripheral retinal neovascularization in the inferotemporal quadrant.



Fig. 12 (Tse and Ober). Case 23. Postoperative fundus photograph of right eye at one month showing remnant of optic disk fibrovascular stalk that was excised during vitrectomy procedure.



Fig. 4 (Tse and Ober). Case 16. Fluorescein angiogram of left eye showing blockage of dye by intraretinal hemorrhages and capillary closure in areas of soft exudate. Several terminal arteriolar segments are occluded (arrows). Note perifoveal capillary nonperfusion and enlargement of foveal avascular zone.

tion of the retinal pigment epithelium in the superotemporal quadrant of the right eye that appeared to be a healed choroidal infarct (Fig. 3). Fluorescein angiography showed hyperfluorescence in areas of pigment epithelial atrophy and blockage of dye corresponding to areas of hyperpigmentation. Patient 16 had acute blurring of vision and, in addition to perifoveal capillary abnormalities, showed cotton-wool exudates, retinal hemorrhages, and other signs of hypertensive retinopathy (Fig. 4). Six patients in Group 2 (Cases 18 to 23), in addition to perifoveal capillary abnormalities, showed peripheral retinal vessel nonperfusion, arteriolar-venular anastomoses, and peripheral retinal neovascularization. Results of laboratory tests done to exclude other causes of peripheral neovascularization were normal (Table 3). Patient 21 had a unilateral vitreous hemorrhage, whereas patients 22 and 23 had bilateral vitreous hemorrhages, traction retinal detachment in one eye, and optic disk neovascularization in one or both eyes.

The five patients in Group 3 (Cases 24 to 28) had no ophthalmoscopic evidence of talc particles. Fluorescein angiograms were not performed on these patients.

Case reports

Four cases are described in more detail to show the predominant fundus findings of talc retinopathy, to show the similari-

TABLE 3

Summary of laboratory data in patients with peripheral retinal neovascularization*

Case No.	Hgb Electrophoresis %A	%A_2	%F	Hgb, g	Hct, %	PPD	CXR	ACE (IU/l)	Two-hr PPG (mg/100 ml)
18	96.0	3.2	0.8	13.0	39.0	+	A†	30	100
19	97.1	2.9	0.0	13.6	41.7	−	N	32	84
20	95.5	4.5	0.0	15.0	45.0	−	N	32	86
21	96.8	3.0	0.2	16.4	47.6	+	N	36	84
22	96.9	2.6	0.5	16.0	40.9	−	N	30	90
23	96.1	3.0	0.9	14.5	43.7	−	N	30	84

*Hgb designates hemoglobin; Hct, hematocrit; PPD, purified protein derivative of *Mycobacterium* tuberculosis (note that patients had been previously treated on the basis of + PPD without clinical evidence of tuberculosis); CRX, chest X-ray; ACE, angiotensin-converting enzyme (normal, 12-36 IU/l); two-hr PPG, two-hour postprandial glucose; +, positive; −, negative; A, abnormal; and N, normal.

†Hilar adenopathy with calcification.

ties of the maculopathy and proliferative retinopathy to sickle-cell retinopathy, and to describe several treatment modalities for secondary complications.

Case 14—A 40-year-old black man had injected methylphenidate HCl for one year. When examined he had decreased vision in both eyes. Visual acuity was R.E.: 6/12 (20/40) and L.E.: 6/18 (20/60). Ophthalmoscopic examination showed bilateral intraretinal talc particles in the posterior pole. Fluorescein angiography of the left macula showed perifoveal capillary closure and an enlarged foveal avascular zone (Fig. 5.) The spaces in the surrounding capillary meshwork were more variable in size than normal. One pathologic avascular zone[15] was located temporal to the foveal avascular zone, and the border of the pathologic avascular zone showed beads of fluorescence that appeared to be hairpin vascular loops. The fluorescein angiogram of the right eye showed similar but slightly milder changes.

Case 20—A 41-year-old black man had injected heroin and crushed methylphenidate HCl tablets for two years. The patient had hypertension and asthma, which were well controlled with methyldopa (Aldomet), hydrochlorthiazide (HydroDIURIL), and oxtriphylline (Choledyl).

Results of physical examination were normal except for blood pressure measuring 140/90 mm Hg. Visual acuity was 6/9 (20/30) in both eyes. The fundi showed Grade 3 talc particles. Just anterior to the equator at 9 and 11 o'clock in the right fundus, neovascularization was seen, which was identical in appearance to the neovascular sea-fans described in proliferative sickle-cell retinopathy (Fig. 6).[7,9] Similar tufts of neovascularization were seen in the superotemporal quadrant of the left eye. Fluorescein angiography showed perifoveal capillary abnormalities in both eyes (Fig. 7). In the periphery of the right eye arteriolar-venular anastomoses (Fig. 8) and sea-fans (Figs. 9 and 10) were noted at the junction between perfused and nonperfused retina.

Case 22—A 26-year-old black man complained of blurred vision and periorbital swelling of the left eye after being struck by a fist. He admitted to having injected 200 to 700 mg of methylphenidate HCl daily for the past two years. His general health had been excellent, and there was no history of systemic disease.

Visual acuity was R.E.: 6/6 (20/20) and L.E.: 6/30 (20/100). The left periorbital area was mildly swollen and ecchymotic. There was a small subconjunctival hemorrhage in the left eye. Ophthalmoscopic examination of the right eye showed numerous tiny, yellow-white, refractile crystalline dots of varying sizes in the retinal arterioles of both fundi, especially concentrated in the end arterioles of the perifoveal arcade. In the right eye, numerous small, elevated neovascular tufts were seen anterior to the equator in the superotemporal and inferotemporal quadrants (Fig. 11). Intraretinal hemorrhage and a diffuse, resolving vitreous hemorrhage were noted in the inferior quadrants. The left eye showed a fresh preretinal hemorrhage as well as a diffuse central vitreous hemorrhage that partially obscured the posterior pole. Peripheral to the hemorrhage, sever-

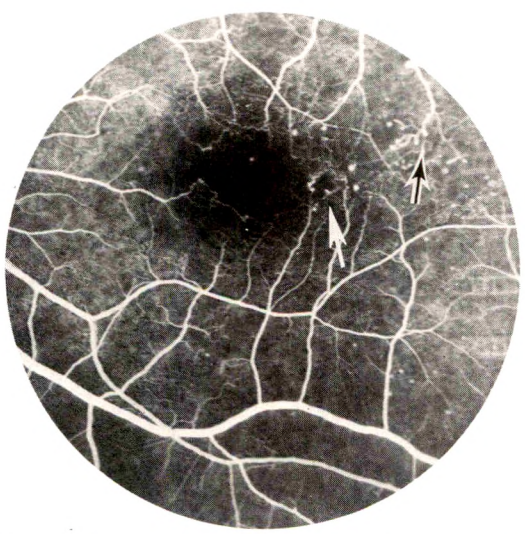


Fig. 5 (Tse and Ober). Case 14. Fluorescein angiogram of left eye showing an enlarged foveal avascular zone. One pathologic avascular zone (white arrow) is located temporal to the fovea, surrounded by beads of fluorescence and abnormal vessel loops. Several vessels show hairpin loops (black arrow).

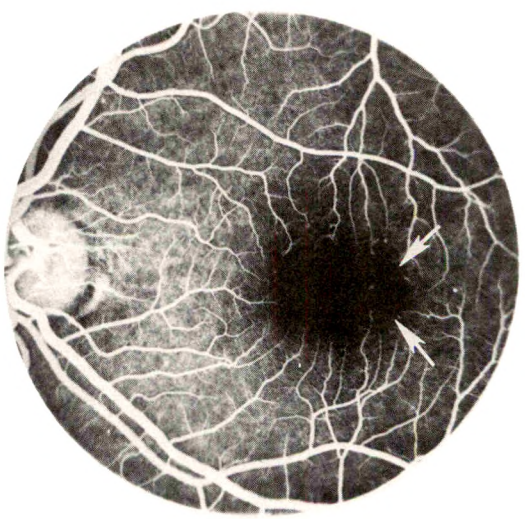


Fig. 7 (Tse and Ober). Case 21. Fluorescein angiogram of left eye showing pathologic avascular zone (arrows), vascular loops, and perifoveal capillary nonperfusion.

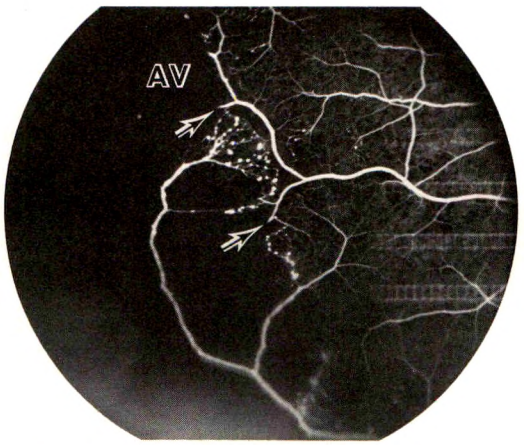


Fig. 8 (Tse and Ober). Case 20. Fluorescein angiogram of temporal periphery of the right eye. Abrupt arteriolar obstruction is seen in the periphery (arrows). An arteriolar-venular anastomosis (AV) is also present; that is, Stage I and II are seen side by side. The peripheral retina (at left) appears totally ischemic.



Fig. 9 (Tse and Ober). Case 20. Fluorescein angiogram in arteriovenous phase showing neovascular sea-fan in the temporal periphery of the right eye that has developed in area of arteriolar-venular anastomosis.

al small areas of elevated retinal neovascularization were visible in the superotemporal quadrant. Fluorescein angiography showed leakage of dye from these new vessels and capillary nonperfusion peripheral to them.



Fig. 10 (Tse and Ober). Case 20. Fluorescein angiogram in early venous phase showing diffuse leakage of dye from sea-fan in right eye. The retinal vessels show nonperfusion anterior to the zone of abrupt closure. The capillaries are dilated and form microaneursym-like dots in the zone of closure.

Panretinal photocoagulation with the argon laser was performed in the right eye. On Sept. 20, 1979, a pars plana vitrectomy was performed on the left eye because of recurrent, nonabsorbing vitreous hemorrhage. Optic disk neovascularization and a localized peripapillary traction retinal detachment were noted at surgery. An encircling scleral buckle procedure with 360 degrees of cryoretinopexy was also performed. Best corrected postoperative visual acuity was 6/12 (20/40). Follow-up examination showed involution of neovascular fronds in the temporal quadrants of the right eye as well as in the superotemporal quadrant of the left eye. However, the patient developed a rhegmatogenous retinal detachment in the right eye that was repaired on Nov. 21, 1979. Recurrent vitreous hemorrhage also occurred in the left eye after blunt trauma and has persisted.

Case 23—A 32-year-old black man had a 14-year history of drug abuse with bilateral decreased vision for several months. He had intravenously injected aqueous suspensions of crushed methylphenidate HCl tablets in daily doses of about 2 g for at least three years. The patient reported several episodes of shortness of breath, malaise, and chills after injections.

The patient had been treated for syphilis and gonorrhea six years before examination. He had also been hospitalized for blood culture-proven *Staphylococcus aureus* septicemia four years previously, and had received six weeks of antibiotic therapy intravenously. Results of direct ophthalmoscopic examination performed on admission were normal. There was no history of diabetes mellitus, sickle-cell hemoglo-

binopathy, sarcoidosis, or hypertension. Family history was noncontributory.

Examination on July 21, 1978, showed visual acuity of R.E.: hand movements at two feet and L.E.: 6/120 (20/400). Ophthalmoscopic examination showed bilateral vitreous hemorrhages, which completely obscured retinal details in the right eye. In the left eye, neovascularization could be seen in the superotemporal quadrant peripheral to the central vitreous hemorrhage. Ultrasonography indicated the presence of fibrous stalks originating from the optic disks in both eyes.

Results of laboratory studies that were done to exclude other causes of proliferative retinopathy were normal (Table 3). Results of additional studies, including sedimentation rate, urinalysis, automated chemical profile, lupus erythematosus preparation, antinuclear antibodies, and electrocardiogram, were normal.

On March 5, 1979, because of nonabsorbing vitreous hemorrhage, a pars plana lensectomy and vitrectomy were performed on the right eye. During surgery, numerous talc particles were noted throughout the posterior retina. The retina was moderately atrophic, and a fibrovascular stalk extended from the optic disk onto the posterior hyaloid surface (Fig. 12). After the fibrovascular stalk was excised and bleeding controlled by intraocular bimanual-bipolar diathermy,[16] 219 intraocular xenon photocoagulation[17] lesions were applied. One month later, the patient developed rubeosis iridis, intraocular hemorrhage, and peripheral traction retinal detachment. Irrigation of the hemorrhage, further endophotocoagulation, peripheral cryopexy, and 360-degree encircling scleral buckling were performed on April 16, 1979. His subsequent course was further complicated by persistent ocular hypertension, which was not controlled with medical therapy. A pars plana filtering procedure[18] was performed on Sept. 21, 1979. Intraocular pressure fell below 20 mm Hg for about three weeks. Subsequent increase of intraocular pressure, however, necessitated cyclocryotherapy on Oct. 29, 1979. Best corrected postoperative visual acuity was 6/60 (20/200) on Nov. 28, 1979.

Pars plana vitrectomy was performed on the left eye on Aug. 10, 1979. Optic disk and peripheral neovascularization were noted, in addition to peripapillary and localized nasal traction retinal detachments. The macula appeared atrophic and contained large numbers of presumed talc particles. Intraocular xenon photocoagulation was applied. A postoperative hemorrhage occurred and has persisted.

Discussion

Intravenous abuse of stimulant drugs intended for administration orally has shown a dramatic increase in recent years.[19] The drug user prepares a suspension for injection by dissolving the crushed tablets in water and boiling them. The turbid suspension may be passed through cigarette filters, cotton fibers, or gauze, with considerable individual variation in the amount of crushing and extent of filtration. The filtrate is then injected intravenously, subcutaneously, or intramuscularly. Showers of insoluble tablet fillers, principally talc (hydrous magnesium silicate), cornstarch, filtering materials, and other contaminants embolize to the pulmonary vasculature, where the particles are trapped. If sufficient microembolization occurs, talc-induced pulmonary granulomatosis may produce pulmonary hypertension and cor pulmonale.[20] Rydell and Hoffbauer[21] postulated that as pulmonary hypertension increases collateral vasculature develops, which eventually allows talc particles to bypass the lungs and enter the systemic circulation directly. Even before the development of these shunts, however, it appears that particles smaller than 7 μ in size can travel to the retina, presumably by traversing the intact pulmonary capillary bed.[19]

AtLee[1] was the first to note a characteristic type of retinopathy caused by talc and cornstarch microemboli. Histologic examination confirmed the presence of talc and cornstarch particles in the retina and choroid, as well as in the lung and brain. Kresca, Goldberg, and Jampol[5] described the first case of widespread peripheral retinal capillary nonperfusion with neovascular proliferation at the junction of perfused and nonperfused retina. Brucker[6] described a single case with disk and peripheral retinal neovascularization and fluorescein angiography that showed widespread retinal capillary nonperfusion. Friberg, Gragoudas, and Regan[4] showed the first case of visual loss secondary to irreversible macular ischemia from presumed talc microvascular occlusion. Schatz and Drake[19] described 12 additional patients with talc retinopathy and macular involvement. None of their cases, however, showed peripheral vessel

occlusion, arteriolar-venular anastomoses, or neovascular proliferation.

To the best of our knowledge, this is the first series of patients with talc retinopathy who show all of the peripheral retinal vascular changes that are seen in proliferative sickle-cell retinopathy and can thus be considered a model for sickle-cell retinopathy as suggested by Kresca, Goldberg, and Jampol.[5] The nonproliferative retinal vascular changes present in the posterior pole in our patients are also similar to those described in patients with sickle-cell hemoglobinopathies.[15,22-24] Abnormalities in the vasculature of the macular and perimacular region occurred in 22 (49%) of 45 involved eyes (Cases 13 to 23). Microaneurysm-like dots, enlarged arteriolar segments, hairpin-shaped venular loops, pathologic avascular zones, and widening and irregularities of the foveal avascular zone were noted (Figs. 4, 5, and 7). Most of the talc emboli are trapped in the retinal circulation of the posterior pole, possibly because of the denser capillary net[25] and the greater blood flow to this region.[26] This results in vascular occlusion of the capillary bed, with consequent disruption in the fine vasculature of this region. The salmon patch and black-sunburst sign described with sickle-cell retinopathy,[9] and presumably related to intraretinal and preretinal hemorrhages, were not noted in our patients. We cannot explain the absence of these signs in talc retinopathy unless it relates to a significant difference in the nature of the occluding particles, or to the relatively small number of patients examined.

Schatz and Drake[19] showed nonfilling of the choriocapillaris and leakage of dye at the level of the retinal pigment epithelium in the maculas of three patients with talc retinopathy, which they attributed to choroidal occlusion. Patient 13 in our series had a pigmented subretinal scar in the superotemporal quadrant of the right eye suggestive of a healed choroidal infarct (Fig. 3). This may have been secondary to occlusion of several choriocapillaris lobules by talc particles, but must remain speculative because the acute lesions were not observed. This patient had the longest history of methylphenidate HCl drug abuse (ten years) and the second highest cumulative number of tablets injected. Choroidal ischemia and subsequent retinal pigment epithelial alteration have also been noted in patients with sickle-cell hemoglobinopathies and was postulated to be secondary to posterior ciliary artery occlusions or focal occlusions of precapillary arterioles.[27] Similar findings have also been reported as complications of photocoagulation in patients with proliferative sickle-cell retinopathy.[28]

Although Patient 16 was normotensive when examined, he showed acute hypertensive retinal vasculopathy associated with talc microembolization, a finding which has been reported by others.[1,19,29] Dilatation and leakage of dye from retinal capillaries was noted on fluorescein angiography (Fig. 4). Capillary occlusion and damage to retinal capillaries may be caused by the talc particles alone; however, the chance of retinal damage may be greater when systemic hypertension is present.[19] In our series, six patients were receiving antihypertensive medications, but all were normotensive at the time of initial examination, except for Patient 20 who was mildly hypertensive.

Proliferative sickle-cell retinopathy has been classified by Goldberg[7] into the five following progressive stages: Stage I, peripheral arteriolar occlusion; Stage II, peripheral arteriolar-venular anastomosis; Stage III, neovascular and fibrous proliferation; Stage IV, vitreous hemorrhage; and Stage V, retinal detachment. Each of these five progressive stages can be shown in this series of patients with

talc retinopathy. Twenty eyes (44%) showed peripheral vascular nonperfusion (Stage I), which was confirmed by fluorescein angiography (Figs. 8 to 10). Peripheral arteriovenous anastomoses occur as a response to peripheral vascular occlusion,[7,9] and thus Stage II can be expected to coexist with Stage I, as shown in several of these patients (Fig. 8). Eleven eyes (24%) showed this stage, and the peripheral neovascularization (Stage III) present in these same 11 eyes was seen at the junction between perfused and nonperfused retina. Fluorescein angiography showed diffuse leakage of dye from the new vessels (Figs. 9 and 10). The peripheral neovascularization seen in Case 20 (Fig. 7), was morphologically indistinguishable from the sea-fan seen in sickle-cell retinopathy.[9] The predominant location of the neovascular proliferation in the superotemporal periphery in our cases and in the previously reported cases[5,6] is similar to hemoglobin sickle-cell retinopathy[7] and suggests a similar pathogenetic mechanism. Vitreous hemorrhage (Stage IV) was present in five eyes (Cases 21 to 23) (11%). Trauma may have contributed to the vitreous hemorrhage seen in the left eye of Patient 22. The persistence of the vitreous hemorrhage with impairment of visual acuity necessitated operations in two patients (Cases 22 and 23). Traction retinal detachment (Stage V) was noted in the left eyes of Patients 22 and 23.

Although the mechanisms that lead to new vessel formation in proliferative retinopathies are not clear, vascular occlusion with resultant retinal hypoxia and production of a presumed vasoproliferative factor probably exert a prominent role.[30,31] In talc retinopathy, talc emboli or other inert fillers, or both, probably act to produce vaso-occlusion in a manner similar to sickled erythrocytes in the sickle-cell hemoglobinopathies.[9] Talc particles range in size from 5 to 10 μ,[4] comparable to that of red blood cells.[32] These particles, however, are irregularly shaped and lack the distensibility of red blood cells, which impedes their passage through retinal capillaries. Once lodged in the capillaries, they may elicit endothelial proliferation or granuloma formation, or both,[33] which, combined with further embolization, may occlude the lumen completely. This obstruction leads to sluggish blood flow and retinal hypoxia, and presumably precipitates neovascular proliferation. Vitreous hemorrhage may then occur as a result of mechanical shearing of these thin-walled friable vessels because of contraction of the vitreous.[34] A sequence of cell-mediated events characterized by vitreous organization and vitreoretinal traction can ultimately lead to retinal detachment.

Proliferative retinopathy has been found in a wide variety of diseases associated with retinal vascular occlusion and ischemia, such as diabetes mellitus, sickle-cell hemoglobinopathies, blood dyscrasias, sarcoidosis, carotid insufficiency, and pulseless disease (Takayasu's disease).[35,36] Both peripheral retinal and optic disk neovascularization have been reported in all of these conditions, although new disk vessels appear to be relatively rare in hemoglobin SC disease.[37] Patients 20 and 21 both showed optic disk as well as peripheral retinal neovascularization. General physical examination and laboratory tests (Table 3) were used to exclude other causes of retinal neovascularization. Because of the small number of reported cases, it cannot be determined if optic disk neovascularization in talc retinopathy is as rare as it is in sickle-cell retinopathy, or whether the different and foreign nature of these obstructing particles compared to sickled erythrocytes in some way predisposes to this expression of retinal ischemia.

The duration of drug abuse and cumulative number of tablets injected influ-

enced the number of talc particles detectable in the fundus and the severity of the retinopathy, a finding which concurs with previous reports.[1,2,19] No retinal talc particles were visible in the five patients who had injected less than 1,000 tablets (Cases 24 to 28); all of these patients had been methylphenidate HCl drug abusers for less than one year.

Although most of the drug abusers filtered the drug suspension before injection, five patients (Cases 10, 14, and 18 to 20) preferred the unfiltered preparation because it gave them a greater "talc flash."[1] They appeared to have more severe fundus involvement than those who injected filtered preparations. For example, Patient 20, who injected more than 10,000 tablets, always unfiltered, had Grade 3 talc retinopathy and peripheral retinal neovascularization. Patient 14, who injected only about 4,000 tablets, had Grade 2 talc retinopathy and decreased visual acuity in both eyes as a result of perifoveal capillary nonperfusion. In contrast, Patient 2, who injected 36,000 tablets, but often with meticulous filtration, had only Grade 1 talc retinopathy.

Neither extent of drug abuse or degree of filtration, however, appeared to markedly affect visual acuity. Most patients in this series did not have significant visual symptoms, with visual acuities of 6/9 (20/30) or better in 18 patients. This finding agrees with previously reported series.[1,2,19] We cannot explain why several patients in Group 1, although visually asymptomatic, did not have 6/6 (20/20) visual acuity with normal fluorescein angiograms. Additional tests of macular function may help to explain this observation, which may be secondary to capillary damage undetected by fluorescein angiography.

Several of our patients had obvious focal areas of macular nonperfusion by fluorescein angiography (Cases 13 and 18 to 21), but retained visual acuity of 6/9 (20/30) or better. The preservation of central vision despite lack of normal macular perfusion has also been noted in ischemic diabetic maculopathy[38] and ischemic hypertensive maculopathy.[39] This discrepancy indicates that there may be redundancy of the blood supply to the macula either from adjacent retinal vascular arcades, the choroid, or both.[39]

Five patients (24%) in our series, however, had visual acuities of 6/12 (20/40) or worse in one or both eyes. The cause of decreased visual acuity in three patients (Cases 14, 16, and 17) was probably related to perifoveal capillary nonperfusion, indicating that, with sufficient embolization, even the rich macular blood supply can be damaged to the point where central visual acuity will be diminished. Vitreous hemorrhage accounted for the decreased vision in the remaining two patients (Cases 22 and 23). Although Patient 21 had vitreous hemorrhage in the right eye, it was located in the inferotemporal periphery, off the visual axis, and therefore did not contribute to decreased visual acuity. Previous series have also noted several cases with decreased visual acuity believed to be related to capillary nonperfusion[19] and retinal hemorrhages.[1,29] Although the decrease in visual acuity in the case described by Murphy[2] was believed to be secondary to retinal pigment epithelial abnormalities, no fluorescein angiography was performed; therefore, macular vessel occlusion cannot be excluded. Patient 12 in our series did show retinal pigment epithelial abnormalities, but had normal visual acuity.

All six patients who showed neovascularization had Grades 3 or 4 talc particles in their fundi. The greater number of particles may have caused more vascular occlusion, with consequent greater ischemia and resultant neovascularization. Only two of the 17 patients who did not

show proliferative retinopathy had sufficient numbers of talc particles present to be considered Grades 3 or 4. Even these two patients, however, had significant vascular damage in the form of a presumptive choroidal infarction (Case 13) and perifoveal capillary nonperfusion (Case 17).

Of the 11 eyes with neovascularization in this series, five had vitreous hemorrhages and two had traction retinal detachment. Because eyes with neovascularization may develop vitreous hemorrhage and traction retinal detachment, consideration must be given to therapy. In the right eye of Patient 22, who had previously had a vitreous hemorrhage, peripheral panretinal ablation with the argon laser was performed. The rationale behind this therapy is the elimination of the ischemic peripheral areas of retina to which the neovascularization is presumably a response. Peripheral ablation was effective in causing involution of the neovascular tissue; however, rhegmatogenous retinal detachment later occurred because of persistent vitreoretinal traction. Another possible treatment is the direct ablation of neovascular fronds and their feeder vessels as has been advocated in proliferative sickle-cell retinopathy.[40,41] Boase[42] however, has expressed some reservation about photocoagulation in sickle-cell retinopathy, because of cases of spontaneous closure of the neovascular fronds[43] and because properly controlled studies of photocoagulation have not been conducted. Even less information is available on the natural course of proliferative talc retinopathy; there is a possibility of spontaneous regression of the neovascular tissue if drug abuse is discontinued.

In those cases where neovascularization persists and results in visually disabling and nonabsorbing vitreous hemorrhage, vitrectomy may be considered. Involution of neovascular tissue in other proliferative retinopathies has been reported after vitrectomy,[44,45] presumably by elimination of the scaffolding on which the vessels were proliferating. This was noted in the left eye of Patient 22, where neovascular tissue regressed after vitrectomy alone. Vitrectomy, however, has been associated with complications such as retinal tears, recurrent hemorrhage, rubeosis iridis, and phthisis bulbi. Those eyes with a severe underlying ischemic retinopathy[44,45] are more likely to develop rubeosis iridis. Rubeosis iridis developed in the left eye of Patient 23 despite the application of endophotocoagulation at the time of surgery to prevent this complication. Because of the small number of cases seen with talc retinopathy, treatment must be individualized in each case, with full consideration of the potential complications.

Prophylactic peripheral retinal photocoagulation was considered in Patients 18 to 20 because of their continuous drug abuse, but they refused therapy. This refusal indicates the difficulties we have encountered in the management of this population of patients. They are often itinerant, poorly compliant when they can be contacted, and are reluctant to accept therapy until visually disabling complications develop. Despite being repeatedly warned of the potential ocular and physical consequences of their drug abuse, all but six continued their habit. Of these six, three are in prison and thus would have more difficulty in obtaining this drug.

All but two patients in our series were black, the population at risk for development of sickle-cell retinopathy. Presumably, the coexistence of these two conditions could be expected to cause more severe retinopathy than either condition alone.[46]

With the recent rise in parenteral

abuse of medications taken orally,[19] an increase in the incidence of talc retinopathy can be expected. Previous series have indicated incidences of ocular involvement of 28%[1] and 44%.[19] In our series, 23 (82%) of methylphenidate HCl drug abusers had some evidence of talc retinopathy. Possibly only those with heavier addiction, and thus greater concern, responded to our attempts to contact them. More accurate figures will be obtained with larger case studies. However, our study shows there is a significant incidence of ocular involvement in methylphenidate HCl drug abusers. Although probably of limited usefulness, greater patient education should be undertaken. Close follow-up is necessary because of risk of progression of the disease. Additionally, removal of, or substitution for, the insoluble fillers currently present in these frequently abused tablets should be considered by the pharmaceutical industry.[19]

Summary

Twenty-three patients had bilateral intraretinal talc microemboli, which were seen on ophthalmoscopic examination. Twelve of these patients had additional retinal vascular or choroidal abnormalities in the posterior pole that were confirmed by fluorescein angiography. Six patients had peripheral retinal neovascularization associated with peripheral retinal vascular nonperfusion. Of these six patients, three had associated vitreous hemorrhage, and two had optic disk neovascularization and traction retinal detachment. The progressive fundus changes seen in talc retinopathy were documented by fluorescein angiography and compared to those seen in sickle-cell retinopathy. One eye in one patient was treated by argon laser photocoagulation for proliferative retinopathy, and three eyes in two patients with vitreous hemorrhage were treated by pars plana vitrectomy.

Acknowledgment

Edward F. Cherney, M.D., supervised patient care in Cases 22 and 23.

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VISUAL LOSS AS A RESULT OF PRIMARY OPTIC NERVE NEUROPATHY AFTER INTRANASAL CORTICOSTEROID INJECTION

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Transient or permanent visual loss after injections to the head and neck has seldom been reported. In virtually all cases, ophthalmoscopic examination implicated inadvertent intravascular injection with subsequent retinal embolism. We describe a patient with marked visual loss seen immediately after corticosteroid injection to the concha nasalis inferioris. At no time did the ophthalmoscopic examination suggest retinal embolic disease. To our knowledge, this is the first case report of visual loss secondary to an optic nerve insult after injection to the head or neck.

CASE REPORT

A 25-year old woman came here for an emergency consultation after acute loss of visual acuity in the right eye. The patient had a long history of sinus disease and was being treated for intermittent nasal obstruction secondary to multiple allergies. As part of her therapy, she periodically received corticosteroid injections to the conchae nasalis inferioris to decrease congestion of the nasal mucosa. Her physician inserted cotton pledgets containing 1% phenylephrine HCl to minimize bleeding and 2% tetracaine to anesthetize the mucosa. Then, using direct visualization, he injected 0.5 ml of a solution containing 20 mg of methylprednisolone acetate into each concha nasalis inferioris at two or three different sites with a 25-gauge sharp needle. He then reinserted the cotton pledgets until hemostasis was adequate. She received such injections on Jan. 25 and Feb. 4, 1980. There were no complications and her symptoms were relieved. On Feb. 19, 1980, she received a third set of injections. She noted a "cloud" over her right eye immediately after the injection and experienced severe visual loss within a few minutes.

She was hospitalized and underwent a neuro-ophthalmologic examination within one hour of the injection. At the time of the initial examination, her visual acuity was R.E.: hand motion, temporally, and L.E.: 6/6 (20/20). There was a 3+ right afferent pupillary defect. There was no blanching or erythema of the ipsilateral face, no blepharoptosis, and no extraocular motor disturbance. The anterior segments were normal. Applanation tonometry showed an intraocular pressure of 16 mm Hg in each eye. The results of an ophthalmoscopic examination were normal; vascular pattern and vessel caliber were normal, without evidence of intravascular emboli, intraretinal hemorrhages, edema, exudates, or nerve fiber layer infarction (Fig. 1). The optic disks were normal.

Visual fields disclosed an extensive loss nasally in the right eye, with less constriction temporally, and loss of central fixation (Fig. 2). The left visual field was normal. Orbital B-scan and computed axial tomography displayed a grossly normal optic nerve (Fig. 3).

Treatment, already begun by her otolaryngologist (J. A. Z.), consisted of 20 mg of prednisone, orally four times a day; 12 mg of nylidrin HCl, orally twice a day; 2.75 mg of histamine, intravenously; and intermittent 5% carbon dioxide inhalation.

The visual acuity in her right eye improved over a

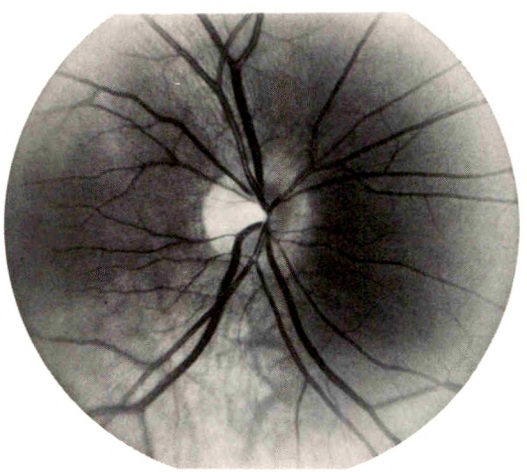


Fig. 1 (Evans, Zahorchak, and Kennerdell). Right eye within two hours of acute visual loss.

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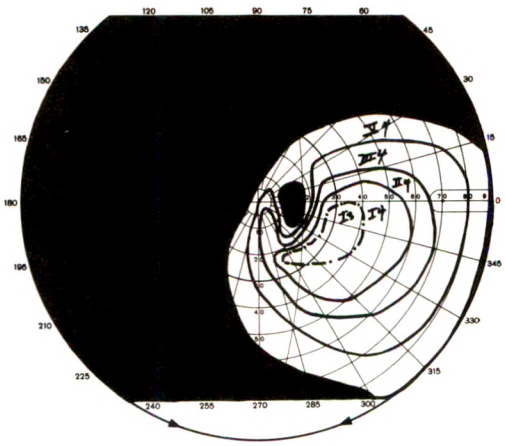


Fig. 2 (Evans, Zahorchak, and Kennerdell). Visual field within two hours of acute visual loss.



Fig. 3 (Evans, Zahorchak, and Kennerdell). Computed axial tomographic scan showing right optic nerve (American Science and Engineering 510 matrix).

period of several days, stabilizing at 6/15− (20/50−) with a small myopic overcorrection. The afferent pupillary defect became much less prominent but persisted. Her nasal peripheral visual field did not expand but she did regain central fixation (Fig. 4).

Discussion

Transient or permanent visual loss after injections to the head and neck is an unusual phenomenon that has been reported with increasing frequency in the past several years. A recent review implicated paraffin, alcohol, local anesthetics, angiographic contrast dye, procaine penicillin, corticosteroid suspensions, and multiple drug combinations.[1] Injections of corticosteroid suspensions have been used in the peritonsillar fossae to decrease postoperative pain,[2,3] in the conchae nasalis to treat allergic rhinitis,[4] and in the scalp to treat alopecia areata, psoriatic plaques, discoid lupus erythematosus, and lichen planopiliaris. Subtenon and, less commonly, retrobulbar injections of corticosteroids have been used for a variety of ophthalmic conditions. Intranasal,[1,5-8] peritonsillar,[9] scalp,[10,11] and retrobulbar[12] injections have all been implicated as occasional causes of visual loss. The incidence of visual loss following injection into the concha nasalis inferioris has been estimated at 0.006%.[13]

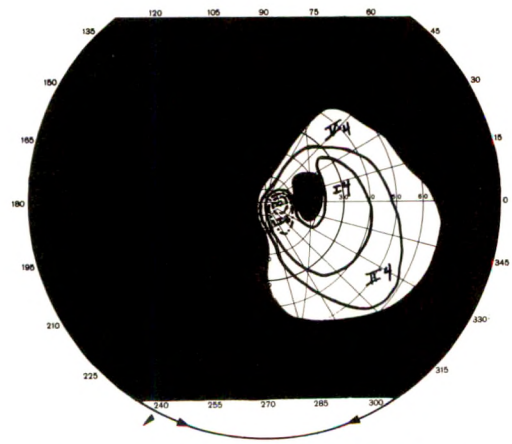


Fig. 4 (Evans, Zahorchak, and Kennerdell). Visual field one month after acute visual loss.

Several authors[13-15] have reviewed this phenomenon. They believe that visual loss occurs as the result of high-pressure injection of material with embolic potential into highly vascular tissue. Retrograde arterial flow allows emboli to lodge in vascular beds far distant from the site of injection. Therefore, injections to such

diverse areas as the scalp, peritonsillar fossa, and conchae nasalis can cause visual loss from retinal embolic disease. Results of ophthalmoscopic examinations, which are almost always consistent with embolic disease, support this theory. Retinal vasospasm in the absence of emboli[9] and optic nerve damage may also play a role. Optic nerve damage may be caused by emboli in the optic nerve vasculature, by optic nerve vasospasm, or by direct neurotoxic effects. Occasional reports of afferent pupillary defects suggest optic nerve involvement in addition to retinal disease, although this could be anticipated with florid retinal embolic disease alone.

This clinical pattern can be reproduced in animals by injecting a corticosteroid suspension and epinephrine together. Injection of either agent alone will not reproduce the retinal changes.[15] This suggests that the corticosteroid suspension, with its poor aqueous solubility and, therefore, embolic potential in an aqueous medium, coupled with the vasoconstricting properties of epinephrine greatly enhances the risk of embolic occlusive disease.

Many previously described patients have had symptoms suggesting embolic disease, including transient central nervous system defects (altered state of consciousness, focal neurologic defects), and blanching or erythema of the ipsilateral face.

The most frequently reported sites of facial injection are all highly vascular and therefore present a small but definite risk of inadvertent intra-arterial injection, regardless of technique. Injecting a significant volume of material into a small space requires sufficient force to propel retrograde intra-arterial emboli to distant vascular beds. Pretreating the nasal mucosa with vasoconstrictors (1% phenylephrine, 5% cocaine) theoretically lowers the risk of intravascular injection.

Subclinical emboli may be more common than recognized, for this diagnosis is usually considered only when the function of vital structures is affected, that is, the retina or central nervous system, and it is confirmed by direct visualization only in the retina.

The patient described here is, to the best of our knowledge, unique in that her retina and optic disk were normal throughout the entire course of observation. The afferent pupillary defect, less prominent as visual acuity improved, strongly suggested that the optic nerve was the site of damage. We believe this is the first case report of visual loss after facial injection caused by optic nerve injury. Whether the insult causes emboli in the optic nerve vasculature or whether they are secondary to vasospasm is speculative, although the former seems more likely. Why the optic nerve circulation is so profoundly affected without any effect on the retinal circulation is also unclear.

Summary

A 25-year-old woman experienced acute visual loss after injection of methylprednisolone suspension into the concha nasalis inferioris. Results of ophthalmoscopic examinations were normal throughout the observation period, a finding not reported previously in cases of visual loss after head or neck injections. An afferent pupillary defect, which diminished as visual acuity improved, implicated optic nerve damage, as did the visual field defect which also improved slightly. This is, to our knowledge, the first case report of acute visual loss caused by optic nerve neuropathy after injection to the head or neck.

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POTENTIAL ARTIFACTS IN SCANNING ELECTRON MICROSCOPY OF THE TRABECULAR MESHWORK IN GLAUCOMA

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The scanning electron microscope is a useful tool for studying the trabecular meshwork surface and abnormalities that may contribute to the malfunction of this tissue in glaucoma.[1-3] Trabeculectomy specimens are one important source of pathologic tissue, but these specimens require special care in order to prevent contamination of the tissue surface during collection and processing for scanning electron microscopy.

While studying the trabecular meshwork from patients with primary open-angle glaucoma, we became familiar with the scanning electron microscopic appearance of some potential problems or artifacts caused by methods of collecting and preparing trabecular tissues. In an attempt to identify the source of contamination, we studied the effects of decompression of the globe, bleeding, and other complications introduced by trabeculectomy, and compared the appearance of washed and unwashed trabeculectomy specimens. We used whole enucleated eyes or eyes obtained at autopsy to show that the meshwork in these eyes is not coated by the materials found in the unwashed trabeculectomy specimens. We examined glaucomatous trabecular tissues from whole eyes obtained at enucleation or autopsy to determine whether the meshwork of such patients is obstructed by a blocking substance as reported by others.[1]

We studied the scanning electron microscopic appearance of the substances that coat and apparently obstruct the trabecular interstices in trabeculectomy specimens. We interpreted these findings as artifacts caused by specimen collection or processing. These data do not confirm the reported finding that an "amorphous blocking substance" is found in the trabecular meshwork in primary open-angle glaucoma.[1] We offer an alternative explanation.

MATERIAL AND METHODS

This study included 17 specimens of two types: surgical specimens obtained at trabeculectomy and whole eyes obtained at autopsy or enucleation. There were ten trabeculectomy specimens. The assistant surgeon washed five of them during collection by continuously irrigating the tissues with a balanced saline solution; the other five were collected without washing. For comparison, we studied seven whole eyes, including five eyes without glaucoma obtained at autopsy and two glaucomatous eyes. One glaucomatous eye was enucleated because of a posterior choroidal melanoma in a patient with open-angle glaucoma and exfoliation. Another globe, obtained at autopsy, had primary open-angle glaucoma. Originally it had been fixed in formalin. We submerged the seven whole eyes in fixative for one to two hours and dissected pie-shaped pieces that included the trabecular meshwork. Immediately after

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excision, we immersed all of the specimens in a 2% glutaraldehyde and 1% paraformaldehyde solution at pH 7.4, buffered with 0.067 M of sodium cacodylate containing 5 mM of calcium chloride.

All the specimens were kept in the fixative solution overnight at 4°C; the next day they were transferred to a freshly prepared batch of the same fixative. We kept all the tissues in this fixative for two to four days and then rinsed them in a cold solution of 0.5 M of sucrose at pH 7.4, buffered with 0.1 M of sodium cacodylate. They were dehydrated in a series of alcohols increasing in concentration up to 100% ethyl alcohol; the specimens then were critical-point dried through carbon dioxide. We mounted the specimens on metal stubs in the usual manner, using a conducting silver adhesive, and coated them with approximately 15 nm of gold in a Hummer II sputter-coater. All the specimens were viewed with a scanning electron microscope at 20 kV and photographed with Polaroid 55 P/N film.

Results

Studies using trabeculectomy specimens are limited by the difficulty of obtaining appropriate controls, since ideally these consist of trabeculectomy specimens from nonglaucomatous patients. In an attempt to circumvent this problem, we compared washed to unwashed trabeculectomy specimens in order to study the presence of a tightly adhering blocking material in the meshwork. Additionally, we compared the scanning electron microscopic findings in nonglaucomatous and glaucomatous whole eyes (obtained at autopsy or enucleation) in regard to the presence of this same blocking substance. Finally, we examined the scanning electron microscopic appearance of probable artifacts introduced after tissue collection and specimen preparation.

The trabecular meshwork of washed specimens was free of substances that might block the flow of aqueous through the uveal and corneoscleral meshwork (Fig. 1). The unwashed trabeculectomy

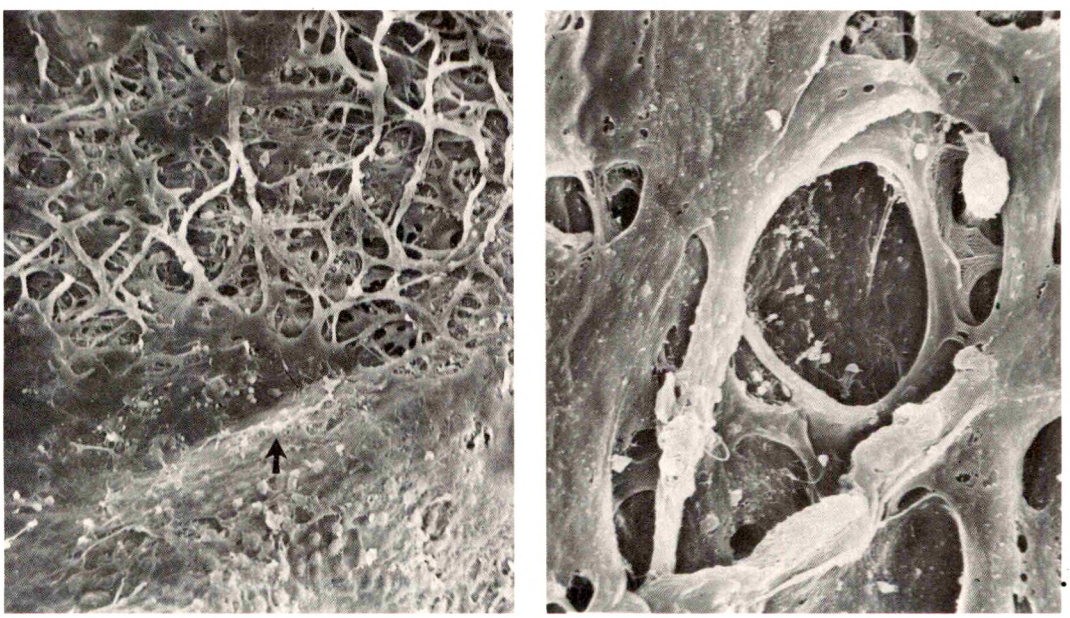


Fig. 1 (Maglio and associates). Washed trabeculectomy specimen. Left, Schwalbe's line (arrow). Uveoscleral and corneoscleral meshworks contain no amorphous material obstructing the interstices (× 200). Right, A higher-magnification view, showing meshwork interstices (× 1,500).

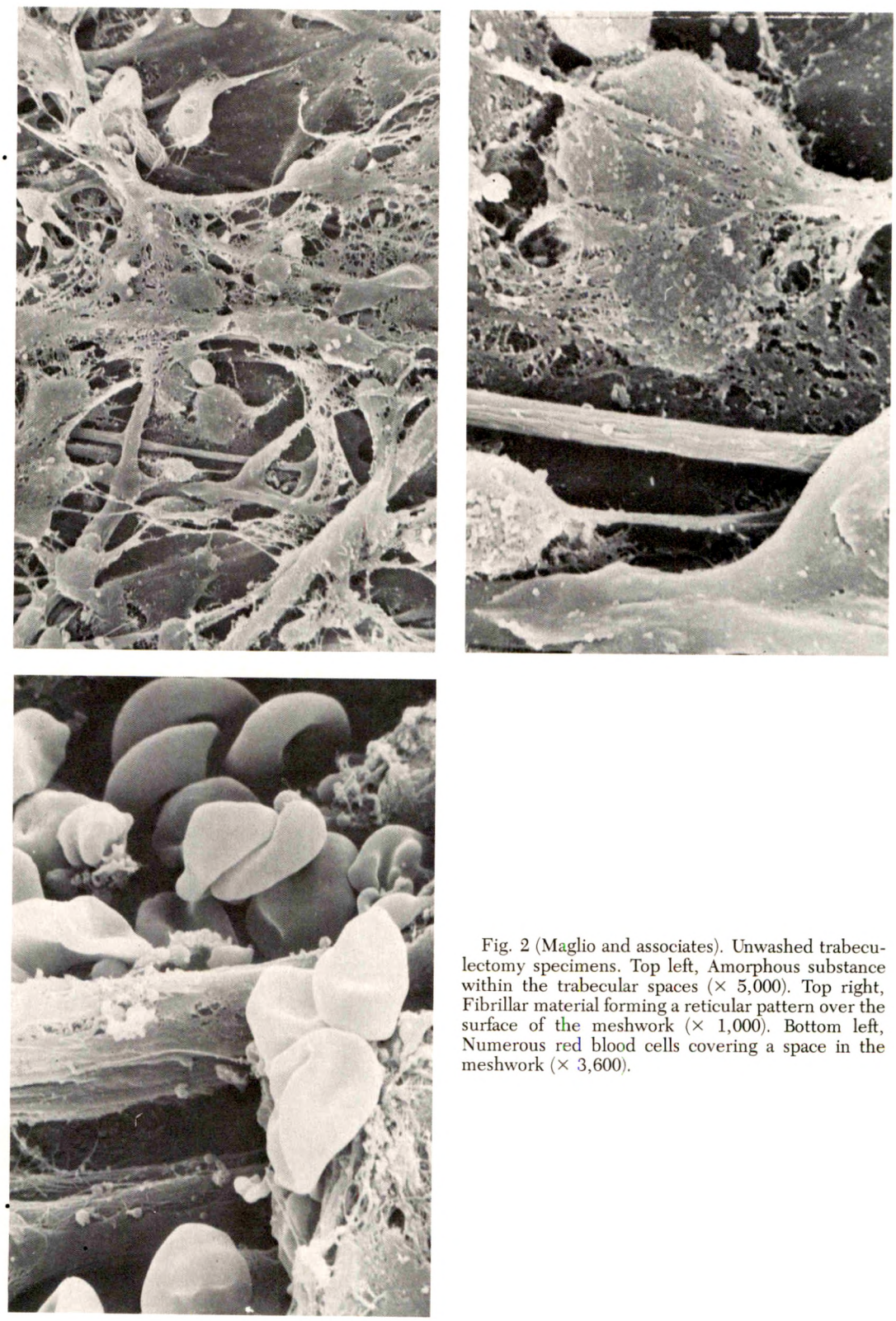


Fig. 2 (Maglio and associates). Unwashed trabeculectomy specimens. Top left, Amorphous substance within the trabecular spaces (× 5,000). Top right, Fibrillar material forming a reticular pattern over the surface of the meshwork (× 1,000). Bottom left, Numerous red blood cells covering a space in the meshwork (× 3,600).

specimens had a number of substances lodged in the trabecular interstices (Fig. 2). One of these materials was amorphous (Fig. 2, top left) and closely resembled some of the materials shown by other investigators.[1] Another material was fibrillar in nature and looked like fibrin (Fig. 2, top right). These materials were observed in most unwashed specimens even when there had been no apparent bleeding during the trabeculectomy. When noticeable bleeding had occurred, they were present in abundance and were usually accompanied by red blood cells (Fig. 2, bottom left). Thus, these materials may have come from blood vessels and were readily removed from the trabeculectomy specimens by washing.

Figure 3 shows the appearance of the trabecular meshwork in one normal eye obtained at autopsy and Figure 4 shows that of an enucleated specimen from a patient with open-angle glaucoma and exfoliation. Neither specimen shows the presence of the materials observed in the unwashed trabeculectomy specimens. The only substance found in the trabecular interstices in the enucleated specimen consists of the exfoliation material (Fig. 4, top right and bottom left), which is formed by numerous fibrils gathered into a ball resembling a tumbleweed. Although we suspect that patients with the exfoliation syndrome have primary open-angle glaucoma made worse by this obstructive exfoliation material, our comparison might be questioned by others

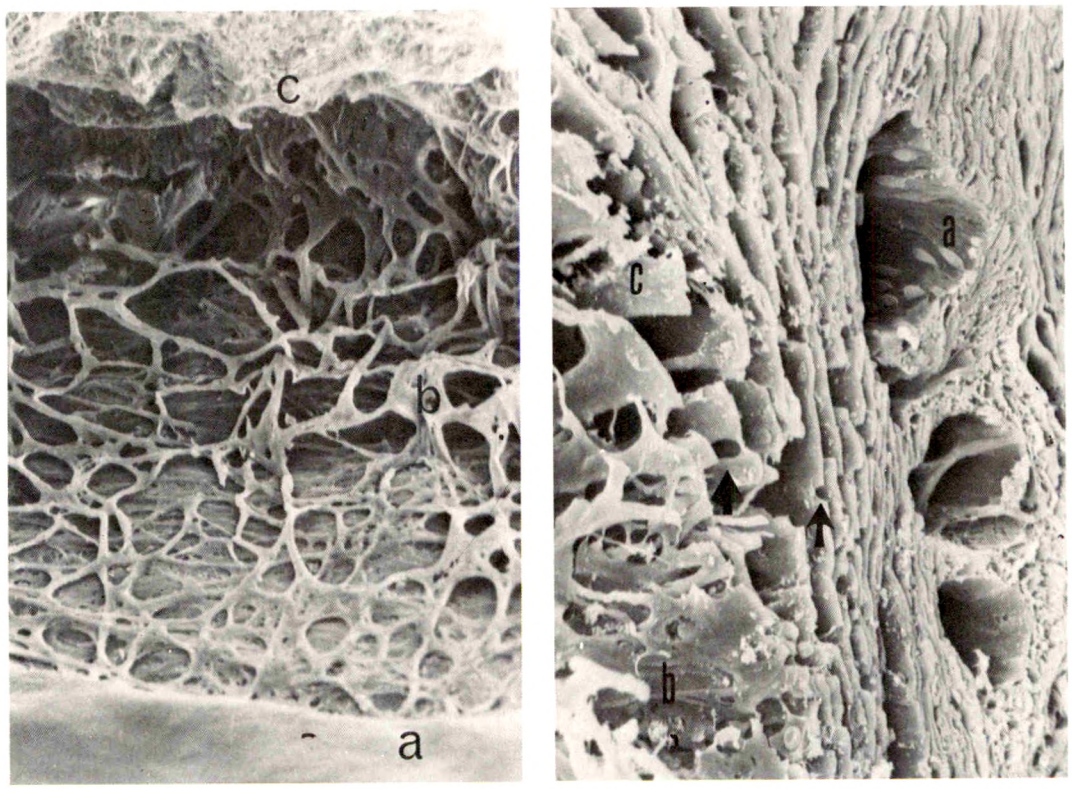


Fig. 3 (Maglio and associates). Normal autopsy specimen from the eye of a 28-year-old individual. Left, Endothelial surface of the cornea (a), uveoscleral meshwork (b), and iris surface (c) (× 1,500). Right, Side view of same specimen shows trifurcated Schlemm's canal (a), the sheets of the corneoscleral meshwork (b), and the uveoscleral meshwork (c). Note the open trabecular pores (arrows) (× 440).

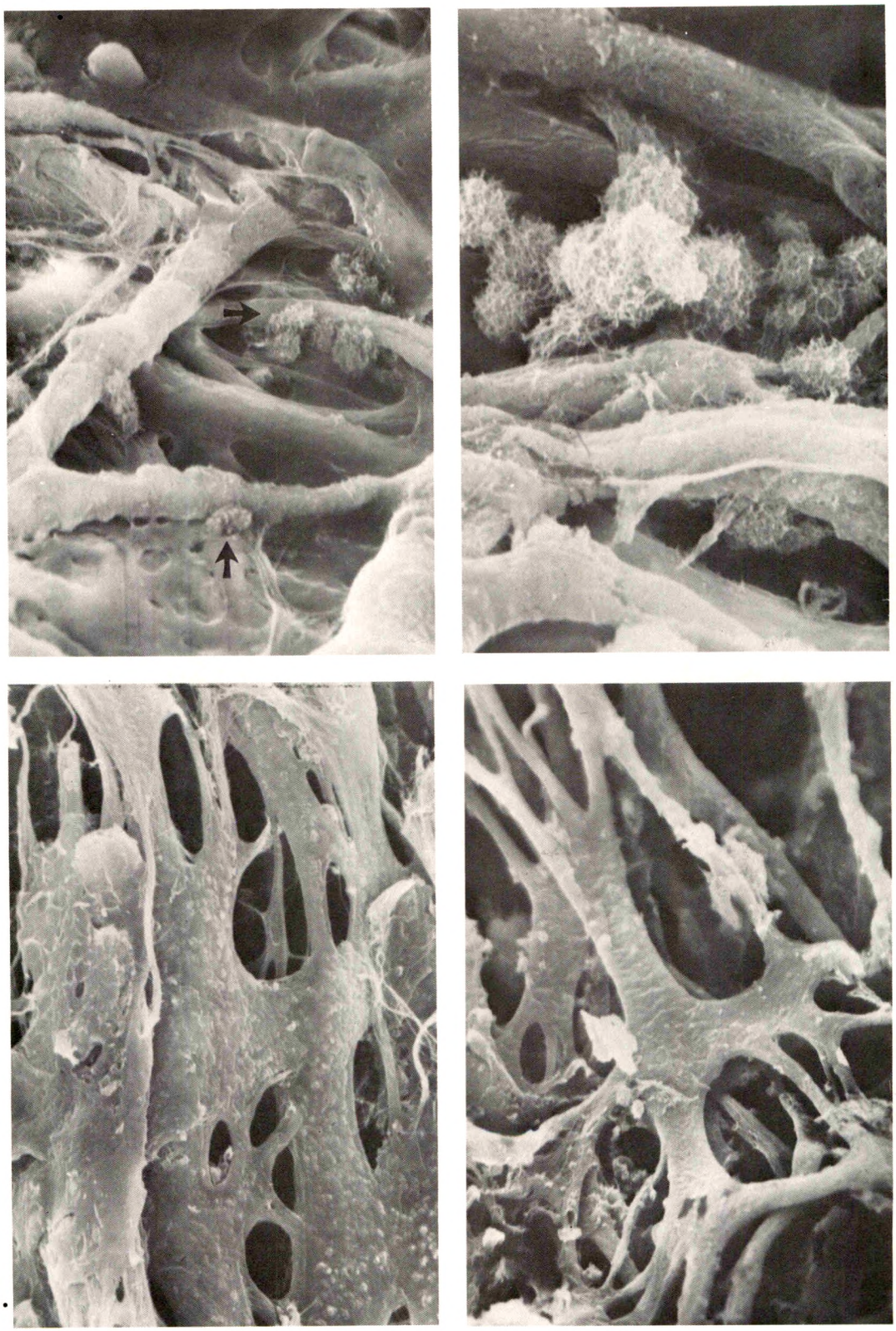


Fig. 4 (Maglio and associates). Autopsy specimen from a patient with open-angle glaucoma and the exfoliation syndrome. Top left, Trabecular spaces are open and the only substance found consists of the fibrillar exfoliative material (arrows) (× 1,500). Top right, Higher-power view of exfoliative material (× 3,000). Bottom left, Trabecular cells with clear intercellular spaces; the nodular surface of these cells is presumably related to the presence of ingested melanin granules (× 1,100). Bottom right, Uveoscleral meshwork; there is a loosely adherent material of unknown nature but the meshwork has clear interstices (× 1,100).

who consider this condition secondary open-angle glaucoma. Therefore, we examined by scanning electron microscopy another eye with primary open-angle glaucoma obtained at autopsy from the University of California Eye Pathology Laboratory, San Francisco. Figure 5 shows that there was no material blocking the meshwork of this specimen.

Although we accounted for the presence of most of the blocking materials by the formation of plasma and other proteins during trabeculectomy, we still had not demonstrated the presence of another substance with a smooth appearance and many cracks on its surface found by other investigators.[1] Such cracks might have been the result of incomplete carbon dioxide drying or of the removal of some volatile substance in the vacuum chamber of the scanning electron microscope. Acetone or butyl acetate are volatile substances present in the silver paint adhesive used to fasten the tissue specimens to metal stubs before viewing. When we liberally applied this adhesive to trabecular tissues similar in size to the typical trabeculectomy specimen (2 × 4 mm), the adhesive seeped into the hollow trabecular interstices, completely blocking them. Figure 6 shows the smooth appearance of this adhesive and the many cracks on its surface. With the aid of a dissecting microscope, small amounts of this adhesive can be applied, thus preventing contamination of the small pieces of tissue obtained at trabeculectomy.

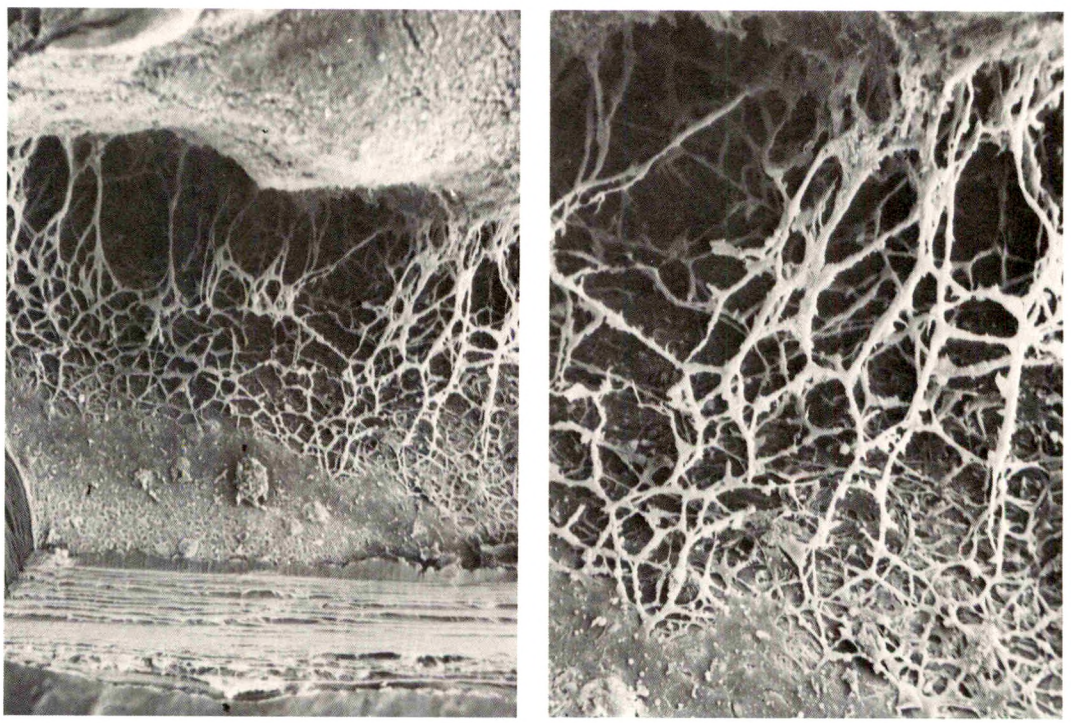


Fig. 5 (Maglio and associates). Eye obtained at autopsy from a patient with primary open-angle glaucoma. Left, Despite formalin fixation, the usual three-dimensional architecture of the meshwork is evident (× 48). Right, Detail of uveoscleral meshwork showing that the trabecular interstices are not covered with amorphous material (× 100).

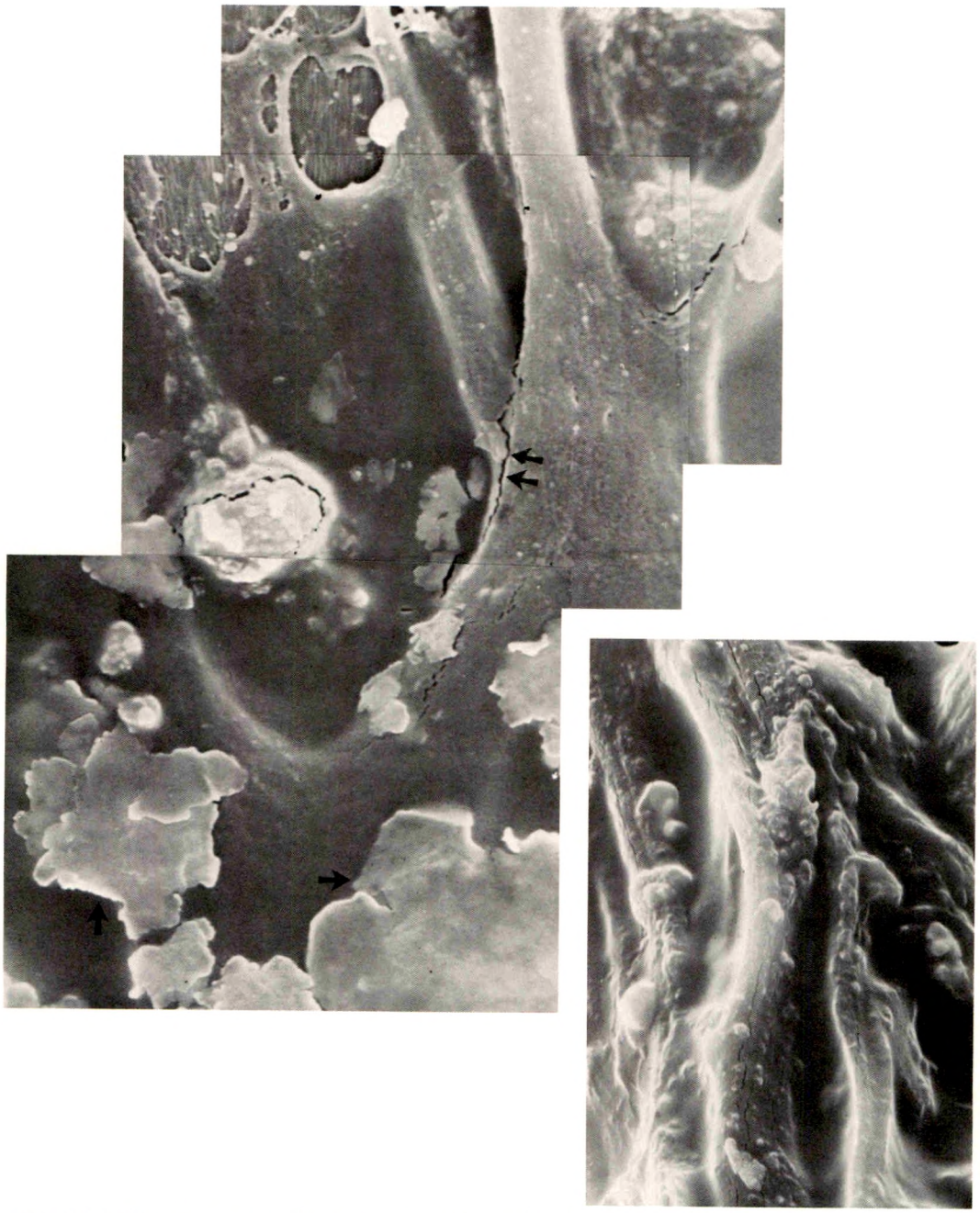


Fig. 6 (Maglio and associates). Contamination of the meshwork with the silver paint adhesive. Left, Surface of the trabecular cells appears to be coated with the adhesive; particles shown at arrows represent large deposits of silver. The cracks along the surface (double arrows) are produced by outgassing in the vacuum chamber (× 7,800). Right, A specimen contaminated by a light application of the adhesive. The surface, through which nodular structures in the trabecular cells are visible, is smooth. Note the minute cracks along a uveal band (× 2,000).

Discussion

The appearance of the trabecular meshwork surface as shown by the scanning electron microscope varies considerably depending on the conditions at specimen collection and on the source of the tissue specimens. The surfaces of specimens obtained at trabeculectomy are often covered by an amorphous substance, a fibrillar material, and, occasionally, red blood cells. These materials can be easily removed in most cases by washing the specimens during collection. When whole eyes obtained at autopsy or enucleation are used as the source of tissue samples, the trabecular meshwork surface is free of these substances and washing is not necessary. We found this to be so in normal as well as glaucomatous specimens. These findings seem to support Svedbergh's[4] suggestion that abnormal substances can be released from the circulation during trabeculectomy and may be precipitated onto tissue surfaces.

A recent series of studies has shown that there is a marked alteration of the various blood barriers with decompression of the globe in primate and human eyes.[5-10] Raviola[5] was the first to show that plasma proteins reflux through Schlemm's canal and the trabecular meshwork into the anterior chamber after paracentesis. Okisaka[6] demonstrated that leakage of proteins into the posterior chamber also occurs with decompression of the globe, and that this is related to a breakdown of the blood-aqueous barrier localized to the anterior portion of the pars plicata. Bartels and associates[7] studied the sequence of events in the breakdown of the blood-aqueous barrier with paracentesis. They reported that protein leaks first into the posterior chamber and a few minutes later into the anterior chamber from Schlemm's canal and the meshwork. Another important finding was the determination that the degree of protein leakage is influenced more by the rapidity with which the intraocular pressure is decreased than the level to which it decreases.[8] To our knowledge, there have been no studies comparing the rate of pressure decrease at trabeculectomy with pressure decrease at paracentesis and other surgical procedures. Tso and Shih,[9] however, reported that there was a breakdown of the blood-retina and blood-pigment epithelium barriers after trabeculectomy in monkeys. Possibly, most procedures in which the anterior chamber is entered produce a rapid decompression and much protein leakage. This is supported by the report of Zimmerman and associates[10] who measured a significant degree of protein leakage into the anterior chamber in humans undergoing most anterior segment surgical procedures. We believe that plasma proteins, as well as other substances released by bleeding, constitute the major contaminants observed in our trabeculectomy specimens.

The appearance of the trabecular meshwork after paracentesis and enucleation in monkeys has been studied by scanning electron microscopy.[7] This report agrees with our findings, and both studies show that the meshwork of enucleated eyes is free of materials coating or blocking the trabecular meshwork. This is consistent with the idea that at trabeculectomy the intraocular pressure decreases much more rapidly than it does in enucleated eyes, and that this difference is an important factor in determining the degree of protein leakage.[8]

We needed data from both enucleated eyes and trabeculectomy specimens in order to examine the provocative finding of an amorphous blocking substance in the trabecular meshwork associated with primary open-angle glaucoma.[1] Enucleated and autopsy specimens from patients with primary-open angle glaucoma are difficult to obtain. Nevertheless, we had

an opportunity to study eyes from three patients with primary open-angle glaucoma who had never undergone ocular surgery. We performed scanning electron microscopy in the two specimens reported here; the third specimen was studied only by transmission electron microscopy (I. Wood, unpublished data). Our scanning electron microscopy studies found no substances deposited over the trabecular tissues. A review of the transmission electromicrographs from the third specimen gave similar findings.

The data from the trabeculectomy specimens and from eyes of patients with primary open-angle glaucoma suggest that the blocking material is released at the time of the trabeculectomy procedure as a result of the sudden decompression of the globe. We observed a blocking substance similar to that previously reported[1] only when the trabecular tissues were contaminated with the adhesive used in the preparation of the tissue samples for viewing by scanning electron microscope. Whether this accounts for the presence of a blocking material in the previous report[1] is not certain. We believe, however, that special care must be taken in the analysis of all trabeculectomy specimens because contamination often produces misleading effects.

Summary

We examined the trabecular meshwork surface by scanning electron microscopy to investigate possible artifacts appearing during collection and preparation of trabeculectomy specimens. As controls, trabecular tissue samples from normal and glaucomatous patients were dissected from whole eyes obtained at enucleation or at autopsy. Our results suggest that the major contaminants are released during decompression of the globe at trabeculectomy; these consist of an amorphous substance, a fibrillar material, and, occasionally, red blood cells. Small tissue samples may also be contaminated by the conducting adhesive used to fasten them to metal stubs. This silver paint may coat the surface of the meshwork and appear to be a smooth substance blocking the outflow of aqueous humor. We were unable to confirm the finding reported by others that an amorphous blocking material is present in the trabecular interstices in patients with open-angle glaucoma.

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AN EVALUATION OF THE ACCURACY OF COMMUNITY-BASED PERIMETRY

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Although there is widespread agreement that visual field tests are a valuable clinical tool, to our knowledge there are no published reports on how effectively they are performed.

We studied a sample of community perimetrists in order to assess (1) their theoretical understanding of and ability to perform perimetry, (2) their performance on a series of patients with visual field defects unknown to them, and (3) the effect of an intensive instructional course on their performance.

SUBJECTS AND METHODS

The perimetrists—Eight of the 11 nonphysician perimetrists employed in a single community agreed to participate in a research study and course in visual field testing. Of these eight, three had had less than six months' experience in visual field testing and were excluded from the study, although they attended the course. The five experienced perimetrists included four women and one man, with one to five years (average, 2.5 years) of experience doing visual field testing. Four were former registered nurses and one a physician's assistant. For the most part, they had been trained on the job and had not taken formal courses in visual field testing; none were certified ophthalmic assistants or technicians. During a typical week, they performed two to eight visual field tests. Their employer, an ophthalmologist, occasionally commented upon their results and suggested modifications in technique, but the perimetrists conducted almost all the field examinations without assistance or supervision. More than 90% of the tests were for glaucoma or suspected glaucoma, with retinal or visual pathway diseases accounting for less than 10%.

The patients—We selected 14 patients from one of our practices (J. D. T.). All had visual field defects established by prior Goldmann perimetry supervised by one of us (J. D. T.). These earlier tests served as reference visual fields. Six patients had hemianopic visual field defects associated with compressive chiasmal lesions verified by craniotomy. Eight patients had prechiasmal (nonhemianopic nerve fiber bundle) visual field defects associated with clinically unequivocal optic neuropathies (four were glaucomatous, three ischemic, and one hereditary). We selected the patients for their willingness to undergo repeated examinations and because we believed their field defects represented a realistic range of diagnostic difficulty. Each patient was a paid volunteer.

The protocol—The first day of the study consisted of an interview with each perimetrist designed to assess background knowledge and approach to perimetry. This was followed by a pre-test, during which each perimetrist examined

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ten to 12 eyes, using the instrument with which he or she had had the greatest experience (three used a Goldmann, one an Autoplot, and one a tangent screen instrument). The matching of perimetrist and patient was random, except that no more than 75% of the eyes tested by each perimetrist could have either hemianopic or nonhemianopic defects; at least 25% had to come from the other group. Each patient was examined by more than one perimetrist. The perimetrist was told only the best corrected visual acuity and refractive error for each patient; this information was not volunteered, but had to be requested. Each perimetrist was timed and permitted a maximum of 40 minutes to examine each eye.

The second and third days of the study consisted of an intensive course on visual fields which taught the general background theory of perimetry and a procedure for performing visual field tests on patients with visual loss of uncertain localization.[1] This procedure stressed the following steps: (1) selecting an initial target with a threshold immediately temporal to the blind spot; (2) measuring the blind spot; (3) exploring the vertical fixational meridian to detect hemianopic defects[2,3]; and (4) exploring the nasal horizontal meridian and paracentral areas for nonhemianopic defects.[4,5]

During this two-day period, the perimetrists practiced carrying out these maneuvers under instructors' supervision on patients they had not previously examined. Each perimetrist examined two patients with prechiasmal and two with chiasmal defects, and results were discussed in relation to the true defects.

On the fourth day, the protocol of the pre-test was duplicated exactly in a post-test, with the perimetrists repeating their examinations on the same patients they tested on day 1. They completed 55 visual field test papers on day 1 and 55 on day 4. The reference visual fields corresponding to these test visual fields had previously disclosed 22 hemianopic defects, 20 nonhemianopic (nerve fiber bundle) defects, 14 of which were nasal steps and 13 normal fields in fellow eyes (Table 1).

The performance evaluation—An ophthalmologist experienced in perimetry but unfamiliar with the study evaluated the perimetrists' performance in the tests in two categories. (1) Qualitative perimetry: the ability to differentiate normal

TABLE 1

CORRECT VISUAL FIELD DIAGNOSES IN EYES EXAMINED BY PERIMETRISTS*

Perimetrist	Correct Visual Field Diagnosis (No. of Eyes)				
	Normal	Abnormal			Total
		Hemianopic	Nonhemianopic		
			Nasal Steps	Other	
A	1	6	2	2	11
B	3	4	2	2	11
C	3	3	4	1	11
D	2	4	3	1	10
E	4	5	3	0	12
	13	22	14	6	55

*Based on prior quantitative perimetry performed by one author (J.D.T.).

from abnormal, and to identify two important localizing defects—hemianopic, involving the vertical meridian, and nerve fiber bundle, involving the horizontal meridian (nasal step). (2) Quantitative perimetry: the ability to define reasonably the extent, depth, and the slope of the margins of significant visual field defects.

The evaluator used the following criteria to judge the presence or absence of localizing defects: (1) hemianopic—a step of at least 5 degrees within 15 degrees of either side of the vertical fixational meridian in at least one isopter; or a step of less than 5 degrees if present in two or more isopters; (2) nasal step—the same as for hemianopic defects, using the nasal horizontal meridian instead of the vertical fixational meridian.

In judging the adequacy of quantitative perimetry, the evaluator noted if only one isopter was used to map a defect (single-isopter mapping), if more than one was used, but mapping was incomplete (incomplete mapping), or if mapping included the largest and smallest visible targets (complete mapping).

Results

General background knowledge and approach to perimetry—Using the interview and observations during the pre-test, we found that the perimetrists used a stereotyped procedure on all patients, regardless of the suspected lesion. This consisted of introducing a moving target from outside the field of vision and noting its threshold. Those using Goldmann perimeters selected a standard initial target arbitrarily, usually an I_4 or larger, and only explored inside its boundary with the initial target, hoping to have the patient signal the target's disappearance into a scotoma. Those using campimeters (Autoplot, tangent screen) selected initial targets with thresholds at 30-degree eccentricity and stopped at the limits of this isopter. Suprathreshold static techniques were generally unfamiliar and rarely used. The perimetrists were unaware of the importance of localizing defects (those with borders along either the horizontal or vertical meridians) compared to nonlocalizing defects. They did not understand the concept or purpose of mapping the physiologic blind spot or of quantitating the depth and slope of defects (Table 2).

Qualitative perimetry—Detecting any visual field defect—The five perimetrists achieved a pre-test average score of 69% (range, 50% to 88%) in separating 42 abnormal from 13 normal visual fields (Table 3). This score improved to 95% (range, 88% to 100%) in the post-test. False-positives also fell from 20% to 10%. This improvement was statistically significant ($P = .039$). We obtained a pre-test and a post-test error count for each patient and applied the Boyett-Shuster test[6] for matched pairs.

Identifying hemianopic defects—The average pre-test score for identification of the 22 hemianopias was 45% (range, 0 to 75%); this improved to 84% (range, 67% to 100%) in the post-test. This improvement was associated with a modest fall in specificity, with false-positives rising from 3% to 12%, and was not quite statistically significant ($P = .11$), based on the Boyett-Shuster test.[6]

In the pre-test, hemianopic defects were missed without regard to size, extent, or depth in five of the six patients with chiasmal lesions. In the post-test, the missed hemianopic defects were confined to only two patients. One had a subtle hemianopic defect only to I_2. The other had gross nystagmus which interfered with fixation; although a defect with

TABLE 2

ERRORS IN VISUAL FIELD TECHNIQUE

Error	No. of Perimetrists
Stereotyped (nonselective) approach	5
Choice of kinetic targets that were too large	5
Moving from seeing to nonseeing field in search of defects	3
Unfamiliarity with suprathreshold static presentation	4
Failure to measure full extent, depth, and slope of defects (quantitation)	4
Failure to recognize significance of localizing (hemianopias, nasal steps) vs nonlocalizing (all other) field defects	4
Failure to measure physiologic blind spot	3

a vertical step was invariably plotted, the step did not fall within 15 degrees of the vertical meridian and the evaluator, adhering strictly to the set criteria for a hemianopic defect, reluctantly judged it as nonhemianopic. The perimetrists therefore improved from randomly missing hemianopic defects, regardless of severity, in the pre-test to missing only the more subtle defects in the post-test.

IDENTIFYING NASAL STEPS—The 14 nerve fiber bundle defects involving the horizontal meridian (nasal steps) were correctly identified 77% of the time (range, 33% to 100%) in the pre-test; this average fell to 67% in the post-test (range, 33% to 100%), probably because the instructional course placed such a high priority on exploring the vertical meridian and central field that perimetrists shifted their attention away from the horizontal meridian in the post-test.

IDENTIFYING NONLOCALIZING NERVE FIBER BUNDLE DEFECTS—The modest

TABLE 3

ACCURACY IN IDENTIFYING VISUAL FIELD DEFECTS

	Pre-Test Accuracy				Post-Test Accuracy			
	True-Positive		False-Positive		True-Positive		False-Positive	
Identification	Mean	Range	Mean	Range	Mean	Range	Mean	Range
Normal vs abnormal visual fields*	69%	50-88%	20%	0-100%	95%	88-100%	10%	0-50%
Specific defects								
Hemianopic†	45%	0-75%	3%	0- 14%	84%	67-100%	12%	0-33%
Nasal steps‡	77%	33-100%	5%	0- 12%	67%	33-100%	0%	—
Nonlocalizing nerve fiber bundle§	20%	0-100%	10%	0- 27%	60%	0-100%	11%	0-27%

*Normal, 13; abnormal, 42.
†With specified defect, 22 fields; without specified defect, 33.
‡With specified defect, 14 fields; without specified defect, 41.
§With specified defect, 6 fields; without specified defect, 49.

decline in identifying nasal steps was offset by an improvement in identifying the six nonlocalizing central and paracentral nerve fiber bundle defects, from 20% (range, 0 to 100%) to 60% (range, 0 to 100%).

Quantitative perimetry—In the pre-test, only two (5%) of the 42 abnormal test fields had defects adequately mapped with multiple isopters; incomplete mapping occurred in 12 (29%), and only one isopter was used in the remaining 28 (66%). This rating improved significantly (P = .032) in the post-test to 24 (57%) with complete mapping, six (14%) with incomplete mapping, and 12 (29%) with single-isopter mapping (Table 4).

We used the following method to obtain scores for each perimetrist separately. For each eye, we awarded one point to the post-test group if the quantification in the post-test was superior to that in the pre-test. Similarly, we awarded one point to the pre-test group if the quantification in the post-test was inferior to that in the pre-test. We awarded no points for a tie. The total score for each perimetrist is simply the number of times the post-test was superior plus the number of times the pre-test was superior. These scores were subjected to the matched pairs test of Boyett and Shuster[6] to obtain the P value.

The marked improvement in both qualitative and quantitative perimetry in the post-test applied to all five perimetrists. This improvement occurred with an increase in examination time, from an average of 12 minutes per eye (range, four to 25) to 20 minutes per eye (range, five to 32).

Discussion

Under the testing conditions we devised, five nonphysician perimetrists without formal training but with considerable experience were able to identify only an average of 69% of abnormal visual fields. Their 77% identification of nasal steps was far better than their 45% identification of hemianopic defects. Adequate measurement of the extent, depth, and slope of identified defects was rare (5%). Accuracy in all categories improved greatly after instruction.

Their pre-test performance is explained, we believe, by two fundamental deficiencies.

Limited understanding of the basic psychophysics of perimetry—The perimetrists were often unaware that mapping a normal peripheral isopter did not guarantee that the more internal contours of the island of vision were normal. Those that did understand this concept employed a flawed technique, hunting for internal defects by moving from seeing to

TABLE 4

Accuracy in quantitating extent, depth, and slope of visual field defects

Mapping	Pre-Test Accuracy*		Post-Test Accuracy*	
	Average	Range	Average	Range
Complete mapping	5%	0-25%	57%	13-88%
Incomplete mapping	29%	0-75%	14%	0-38%
Single-isopter mapping	66%	25-100%	29%	13-75%

*No. of visual fields with defects = 42.

nonseeing areas. Once they found a deficit, the perimetrists did not grasp the difference between identifying and defining its full extent, depth, and slope with many isopters.

Stereotyped approach to visual field examination, regardless of the working diagnosis and expected defects—None of the perimetrists understood the concept of selective exploration of the visual field in search of defects that establish the location of the causative lesion within a specific segment of the visual pathway. Although several were aware of the importance of the nasal horizontal meridian visual fields in glaucoma, none realized that this applied to other optic neuropathies. A minority knew that the vertical meridian was a threshold boundary for chiasmal and postchiasmal defects. No one realized that such defects with borders involving the horizontal or vertical meridian should have a high detection priority because of their importance in localizing disease to the prechiasmal (horizontal) or chiasmal (vertical) areas. Few recognized that the central 30 degrees of the visual field contained the majority of these defects, and that suprathreshold static techniques, as well as small kinetic targets, are an efficient means of exploring these areas (Table 2).

Is this an accurate profile of community perimetry as practiced nationwide? The sample is small, but we believe it is representative. It is our impression that most visual field tests in this country are not performed by ophthalmologists or by certified technicians with formal education in perimetry, but by assistants trained on-the-job, like the five we tested. We selected the patients to include not just problem cases, but commonly observed visual field defects.

If we are to judge by our small sample, present-day office perimetry is largely adequate in identifying typical glaucomatous defects—a task frequently called for in clinical practice. However, adequate quantification of these glaucomatous defects is necessary to measure progression, and this goal may not now be properly met. Additionally, we question whether informally trained perimetrists are skilled at identifying the hemianopic defects which signal the presence of possible life-threatening intracranial disease.

Additional formal training should help. In this study, the two-day course resulted in significant improvements in both of these areas. Such courses must teach perimetrists that the visual field examination is not just a mechanical exercise; the procedure must be adapted to fit the suspected location of the lesion. For example, a patient with visual loss of uncertain origin should have the vertical meridian intensively explored first; a patient with suspected nutritional or hereditary optic neuropathy must have the caecocentral area examined first; visual fields in established glaucoma require scrupulous quantitation for meaningful follow-up. Such a flexible approach demands a good understanding on the part of the perimetrist of the working diagnosis, the expected visual field defects, and their localizing value.

Finally, if formal visual field courses of this type are to be meaningful, they must include monitored practice sessions in mapping representative defects. We found these sessions essential for identifying persistent errors in both concept and technique.

Summary

We assessed the accuracy of five office-based perimetric technicians in the examination of 14 patients with prechiasmal and chiasmal defects unknown to them. A

pre-test was followed by two days of instruction and supervised practice; the technicians were then retested on the same patients (post-test). An independent evaluator scored their results.

The identification of any field defect rose significantly (P = .039) from 69% in the pre-test to 95% in the post-test. The identification of the hemianopic configuration of a defect rose from 45% in the pre-test to 84% in the post-test (P = .11); initial identification of nasal steps was 77%, falling to 67% in the post-test.

Adequate definition of extent, depth, and slope of defects was rare (5%) on the pre-test, but rose significantly (P = .032) to 57% on the post-test.

The monitored patient examinations were essential for correcting recurrent flaws in technique. This study shows that such teaching efforts, including emphasis on identifying hemianopic defects, are necessary to raise visual field examination to the level of a consistently reliable diagnostic determinant.

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HYPERTROPHY OF THE RETINAL PIGMENT EPITHELIUM ASSOCIATED WITH GARDNER'S SYNDROME

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Congenital hypertrophy of the retinal pigment epithelium, characterized by isolated, flat, well-demarcated, hyperpigmented lesions at the level of the retinal pigment epithelium, is usually asymptomatic.[1-3] The shape of the lesions is generally round to oval, and their edges may be smooth or scalloped. A narrow, hypopigmented halo around the periphery of the lesions and depigmented lacunae may be present. Bilateral occurrence[3] and enlargement over time are rare.[4] Alterations of the overlying retinal vasculature may occur.[5] Histopathologic study of the lesions has shown an increase in concentration of pigment granules in enlarged, but not hyperplastic, retinal pigment epithelial cells and degeneration of the overlying photoreceptors.[2,6] To our knowledge, there have been no previous reports of familial transmission of congenital hypertrophy of the retinal pigment epithelium or its association with systemic disease.

Gardner's syndrome is an autosomal dominant condition characterized by multiple intestinal polyps, hard-tissue abnormalities, and soft-tissue abnormalities.[7-9] Many patients do not manifest all three components. The polyps, which are benign adenomas histologically, are most often located in the colon and rectum, but sometimes occur in the stomach and small intestine as well. Because most patients with colonic polyposis develop carcinoma of the colon, they require close surveillance, and prophylactic colectomy is usually performed. The hard-tissue abnormalities include osteomas, particularly of the mandible and other skull bones, cortical thickening in the long bones and ribs, and abnormal dentition.[10] Soft-tissue abnormalities include epidermal and sebaceous cysts, pigmented spots on the torso and limbs,[11] fibromas, lipomas, desmoids, and diffuse postsurgical intra-abdominal fibromatosis. Other malignancies, including fibrosarcomas,[12] carcinomas in the region of the ampulla of Vater,[13] carcinoma of the thyroid,[14] and skin cancer,[15] also appear to be associated with Gardner's syndrome. Increased tetraploidy in cultured skin fibroblasts, which may be related to the widespread neoplastic tendency of this condition, has been observed.[16]

The only reported ocular lesion occurring in Gardner's syndrome of which we are aware is exophthalmos due to orbital osteoma,[17] although Awan[18] described one case of angioid streaks in a patient with an unspecified type of multiple polyposis. Herein we describe the occurrence of congenital hypertrophy of the retinal pigment epithelium in three affected members of a family with Gardner's syndrome.

SUBJECTS AND METHODS

We examined nine surviving members of a kindred with Gardner's syndrome (Fig. 1) for the features of this condition. We performed sigmoidoscopic and barium enema examinations on all except for two children under 8 years of age who showed no extraintestinal manifestations. We performed careful ophthalmoscopic examinations on all nine patients, includ-

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ing Patient II-3, who was not in the line of transmission of the Gardner's syndrome gene.

Case reports

Patient IV-2—The proband, a 6-year-old girl, was referred to us for examination of a pigmented fundus lesion in the right eye. The lesion was suspected of being a malignant melanoma. The lesion was discovered during an examination for stigmata of Gardner's syndrome, which was present in her mother. The girl's visual acuity was 6/6 (20/20) in each eye; the results of a complete ocular examination were unremarkable except for the ophthalmoscopic findings. Several discrete, well-demarcated, flat, darkly pigmented lesions were seen at the level of the retinal pigment epithelium of each eye (Fig. 2). Five such lesions were present in the right eye and 12 in the left eye. The lesions were scattered randomly over the fundus and not grouped. They were oval to round and varied in diameter up to approximately 2 disk diameters; some had hypopigmentation around the periphery. These lesions have remained stationary during the last three years. The girl has colonic polyps and osteomas of the skull.

Patient III-2—This 25-year-old woman had Gardner's syndrome. We performed a complete ocular examination because of the findings in her daughter, Patient IV-2. Results of this examination disclosed four pigmented lesions in the right eye and six in the left. These lesions resembled those of her daughter, except that the areas of depigmentation tended to be more prominent (Fig. 3). Fluorescein angiography disclosed areas of blocked choroidal fluorescence, window defect, choriocapillaris nonperfusion, and retinal capillary caliber irregularities over some of the lesions (Fig. 4). The electroretinogram, electrooculogram, and visual-evoked response were normal in each eye. The lesions have not changed during two years of follow-up. This patient had previously undergone a total proctocolectomy and ileostomy for multiple polyposis. The excised specimen had 545 benign adenomatous polyps. At the time of surgery, a gastric polyp was palpated. Other features of Gardner's syndrome present in this patient included osteomas in the skull, ethmoid bone, and mandible, endosteal cortical thickening in the femur, and multiple sebaceous cysts. She also had cirrhosis related to excessive ethanol intake, and had had surgery for a ventricular septal defect.

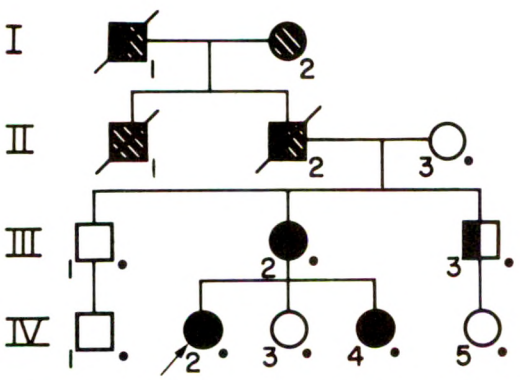


Fig. 1 (Blair and Trempe). Pedigree of family described. Note generation-to-generation transmission, consistent with autosomal dominance, of both Gardner's syndrome and congenital hypertrophy of the retinal pigment epithelium. The absence of congenital hypertrophy of the retinal pigment epithelium in Patient II-3 supports the view that the hypertrophy was inherited from the side of the family with Gardner's syndrome. Square, male; circle, female; slash, deceased; dot at lower right, examined; arrow, proband. I-1 and II-2: Gardner's syndrome, hypertrophy status unknown; I-2 and II-1: status unknown; II-3, III-1, IV-1, IV-3, and IV-5: unaffected; III-2, IV-2, and IV-4: Gardner's syndrome and hypertrophy; III-3: Gardner's syndrome, no hypertrophy.

Patient IV-4—This patient, two years younger than her sister, Patient IV-2, was first examined here at the age of 6 years. Results of her ocular examination disclosed four pigmented lesions in the right eye and one in the left eye. A double-contrast barium enema disclosed many small, filling defects, which may have represented small polyps, throughout the colon. The girl had several sebaceous cysts on the scalp, but no osteomas.

Other family members—Patient III-3 had no eye lesions, osteomas, or skin lesions, and the results of sigmoidoscopy and barium enema were normal. He was considered to be affected on the basis of an infiltrating angiolipoma of the thigh. Patients I-1 and II-2 both died of bowel cancer. Patient II-1 died in World War II at the age of 19 years; no manifestations of Gardner's syndrome were known to have been present.

Discussion

Ophthalmologists should be aware of the association of congenital hypertrophy of the retinal pigment epithelium with Gardner's syndrome. Since the fundus lesions appear to be congenital, an ophthalmologic examination can provide information as to which children in a kindred with Gardner's syndrome are affected and therefore at risk of developing polyposis and carcinoma of the colon. Gardner's syndrome within a family may be diagnosed for the first time when an ophthalmologist observes the fundus lesions and initiates further diagnostic in-

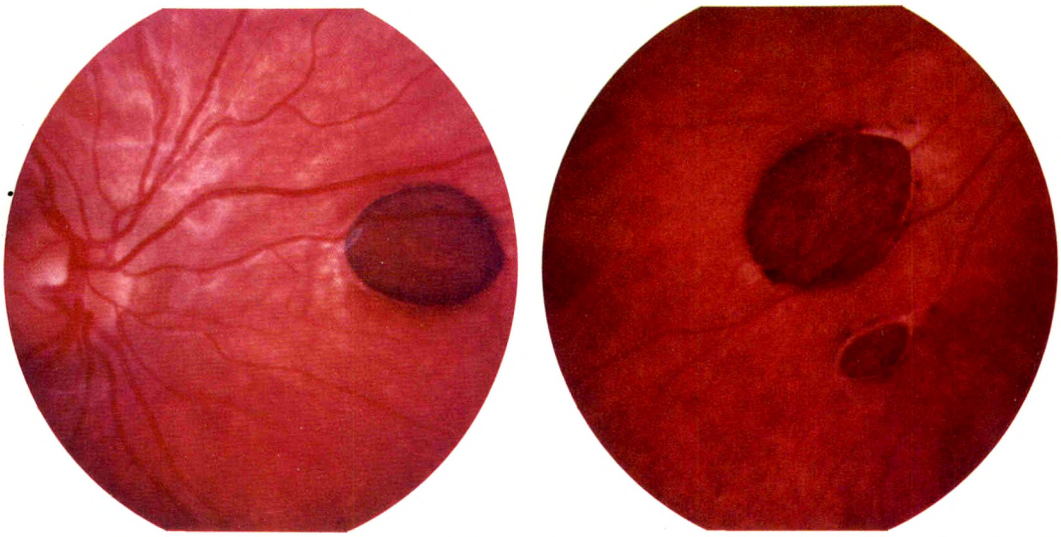


Fig. 2 (Blair and Trempe). Patient IV-2. Congenital hypertrophy of the retinal pigment epithelium. Left, Lesion nasal to the disk in right eye. Right, Two lesions inferonasal to the disk in left eye. Note mottled pigmentation with hypopigmented halo at the periphery.



Fig. 3 (Blair and Trempe). Patient III-2. Congenital hypertrophy of the retinal pigment epithelium in the left eye. Note well-defined, irregular area of depigmentation within the lesion which exposes a large choroidal vessel (arrow). Less discrete areas of pigmentation alteration surround the lesion.



Fig. 4 (Blair and Trempe). Fluorescein angiogram of lesion seen in Figure 3 (Patient III-2). Note hypofluorescence where there is increased pigmentation. Hypofluorescence in the lower left of the lesion, due to choriocapillaris nonperfusion, permits clear visualization of a large choroidal vessel. The small retinal vein over this area has a fusiform caliber alteration (arrow). The area just to the lower left of the lesion shows a window-defect type of hyperfluorescence.

vestigation. He should question the patient about rectal bleeding, diarrhea, abdominal pain, and mucous discharge. He should examine the skin for cysts and masses and the bones for osteomas and perform a sigmoidoscopy and barium enema when he suspects Gardner's syndrome. A family history and examination of other family members are also important.

The appearance of the individual fundus lesions associated with Gardner's syndrome is typical of congenital hypertrophy of the retinal pigment epithelium, but there are several atypical aspects: familial transmission, bilateral occurrence, multiple lesions per eye, and association with systemic disease. No one, to our knowledge, has reported congenital hypertrophy of the retinal pigment epithelium occurring in more than one member of a family. In our pedigree, it occurred in two sisters and their mother. This suggests an autosomal dominant mode of transmission, which is consistent with the condition's being a manifestation of Gardner's syndrome. The lesions were bilateral in all three of these patients. We are aware of only one other bilateral case.[3] In all the previous reports we could locate, only one lesion per eye was present. The lesions were multiple in five of the six affected eyes of our patients.

Grouped pigmentation of the retina and congenital hypertrophy of the retinal pigment epithelium are similar conditions histologically.[2,19] In grouped pigmentation of the retina, pigmented spots occur at the level of the retinal pigment epithelium. The spots are arranged in a cluster, often suggestive of animal tracks. The individual lesions seldom have fenestrations or halos and are usually smaller than those seen in congenital hypertrophy of retinal pigment epithelium.[20] The individual lesions in our cases resembled those of congenital hypertrophy of the retinal pigment epithelium more than those of grouped pigmentation of the retina, and their distribution was unlike that seen in grouped pigmentation of the retina. Furthermore, grouped pigmentation of the retina is usually unilateral, has not been convincingly associated with systemic disease,[20] and has been found to be familial in only one atypical instance.[21]

The type of lesions seen in our patients must also be differentiated from malignant melanoma, choroidal nevi, reactive retinal pigment epithelial hyperplasia, the sunburst lesion of sickle-cell retinopathy, and geographic dark posterior fundus patches.[22] Modern diagnostic techniques make differentiating these conditions from congenital hypertrophy of the retinal pigment epithelium straightforward.

We examined the 24-year-old maternal uncle of the proband (Patient III-3) for

Gardner's syndrome; results of sigmoidoscopy, barium enema, and indirect ophthalmoscopic examination were normal. We classified him as affected with the syndrome because an infiltrating angiolipoma[23] had been removed from his thigh two years previously. We are unaware of this type of tumor being reported in Gardner's syndrome. Nevertheless, because a wide variety of connective tissue tumors occur in Gardner's syndrome, we assume that the angiolipoma was a manifestation of the abnormal gene.

Several forms of intestinal polyposis are known, and their classification is in dispute.[15,24-27] Classically, familial polyposis coli has been differentiated from Gardner's syndrome by the absence of extracolonic manifestations. Recently, gastric polyps, duodenal polyps, mandibular osteomas, and dental abnormalities consistent with Gardner's syndrome have been discovered in patients previously thought to have familial polyposis coli.[24-26] Thus, the distinction between the two has become blurred. Furthermore, Binder and associates[27] have reported the case of a family with colonic polyps, sebaceous cysts, gastric polyps, and malignant brain tumor. This suggests an overlap between Gardner's syndrome and Turcot's syndrome, which consists of colonic polyposis and brain tumor.[28]

The Peutz-Jeghers syndrome consists of gastrointestinal polyposis and melanotic macules on the lips, buccal mucosa, eyelids, and digits.[29,30] Although these pigmented spots occasionally occur on the conjunctiva, we are unaware of such lesions occurring in the fundus. The pigmented skin lesions of Gardner's syndrome have a different distribution, being confined to the torso and limbs.[11] The polyps in the Peutz-Jeghers syndrome are hamartomas, and are thus different from the polyps of the above syndromes both histologically and prognostically.

In conclusion, the presence of many bilateral polyps or familial hypertrophy of the pigment epithelium should alert the ophthalmologist to the possible presence of Gardner's syndrome. Appropriate examinations can then be carried out to substantiate the diagnosis and to determine the presence of malignant or premalignant lesions.

Summary

Congenital hypertrophy of the retinal pigment epithelium was seen in three affected members of a kindred with Gardner's syndrome. The latter consists of a triad of many intestinal polyps, hard-tissue abnormalities, and soft-tissue abnormalities. Although the appearance of the individual lesions in our patients was typical of hypertrophy of the retinal pigment epithelium, the following atypical features were present: multiple lesions per eye; bilateral occurrence; familial transmission; and association with systemic disease. Ophthalmoscopic examination can help identify children who are at risk of developing polyposis and carcinoma of the colon. In some instances, Gardner's syndrome may be diagnosed in a patient and his family as a result of observing the fundus lesions.

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APPLANATION TONOMETRY WITH AND WITHOUT FLUORESCEIN

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Some investigators have suggested that it is unnecessary to use fluorescein when doing applanation tonometry and that simply viewing the mires with white light is adequate.[1,2] This idea conflicts with the original technique described in 1957 by Goldmann and Schmidt[3] who recommended instilling the dye by briefly placing a dry strip impregnated with fluorescein in the conjunctival sac.

Moses[4] explained why fluorescein is necessary in 1960. Only the central 3.06 mm of the applanation prism is in contact with the cornea, not the entire surface as is sometimes mistakenly believed. A triangular tear meniscus forms in the area bounded by the applanating surface and the cornea with the apex at this junction (Fig. 1). It is the apex of the tear meniscus which defines the applanated area and it is indistinguishable from the cornea unless stained with a sufficient amount of fluorescein.[4,5] Moses pointed out that if the fluorescein concentration is too weak, only the thickest part of the triangle and not the apex will be visible (Fig. 2, top left). The true applanated area will then be less than the required 3.06 mm and the pressure will be underestimated. The thin apex contains less total fluorescein than the thicker base and, therefore, fluoresces less brilliantly. Most topically applied anesthetics are weakly acidic, which tends to inhibit the fluorescence of fluorescein.[5] If an anesthetic is used to moisten the fluorescein-impregnated paper strip, a significant amount of the fluorescence is quenched. This may drop the apical portion of the mires below the fluorescence threshold and have the same effect as too little fluorescein.

When no fluorescein is used, only the very base of the tear meniscus is seen on end and the pressure can be grossly underestimated (Fig. 2, top right). The ideal situation is to have the apex of the meniscus clearly visible, delineating the applanated area (Fig. 2, bottom left). My purpose was to demonstrate that these arguments are correct in a clinical situation.

SUBJECTS AND METHODS

I used two groups of 25 patients (50 eyes) as test subjects. In Group A, I measured applanation pressures after applying proparacaine HCl to both eyes. I used white light and recorded the pressure when the inner aspects of both mires just touched. I lightly touched both inferior conjunctival sacs with a dry, commer-

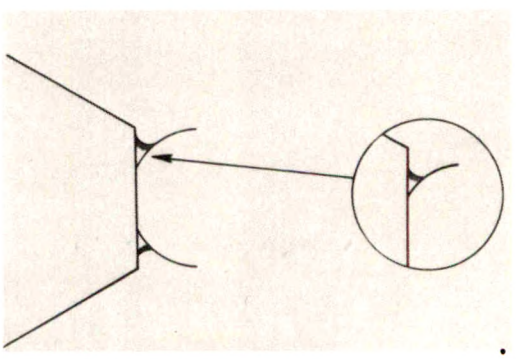


Fig. 1 (Roper). Cross-section of the cornea-applanation surface interface. Insert, tear meniscus bounded by the cornea and the prism with its apex where they come in contact.

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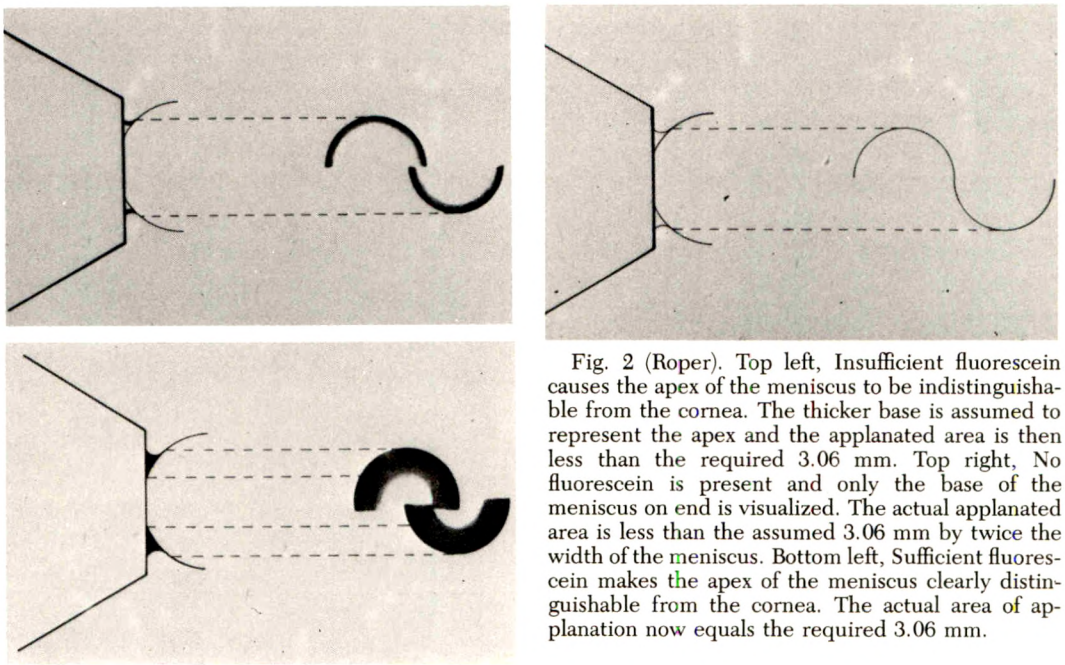


Fig. 2 (Roper). Top left, Insufficient fluorescein causes the apex of the meniscus to be indistinguishable from the cornea. The thicker base is assumed to represent the apex and the applanated area is then less than the required 3.06 mm. Top right, No fluorescein is present and only the base of the meniscus on end is visualized. The actual applanated area is less than the assumed 3.06 mm by twice the width of the meniscus. Bottom left, Sufficient fluorescein makes the apex of the meniscus clearly distinguishable from the cornea. The actual area of applanation now equals the required 3.06 mm.

cially prepared fluorescein strip, and then measured the applanation pressure with a cobalt blue light. A second observer recorded both readings, which were then analyzed in a masked fashion.

I then measured the applanation pressures in the 25 subjects (50 eyes) in Group B with white light as described above. Then I administered one drop of a combined preparation of 0.25% fluorescein sodium and 0.4% benoxinate HCl to each eye and measured the applanation pressures with the cobalt blue light.

Results

In Group A, the mean pressure was 11.1 ± 3.41 mm Hg in the eyes tested without fluorescein compared with a mean pressure of 12.78 ± 3.29 mm Hg in the eyes tested with a small amount of fluorescein. The average difference of 1.68 mm Hg was significant at the $P < .05$ level.

In Group B, the mean pressure was 11.72 ± 3.20 mm Hg in the eyes tested without fluorescein compared with a mean pressure of 17.34 ± 4.07 mm Hg in the eyes tested with the combined fluorescein-anesthetic solution. The average difference of 5.62 mm Hg was highly significant at the $P < .001$ level.

Discussion

I chose a combined anesthetic-fluorescein preparation for this investigation over a larger amount of fluorescein applied from a paper strip because of its standardized concentration.[6-9] I could have used a sterile solution of 0.25% fluorescein separately from the anesthetic to achieve the same consistency.[10] I assumed partial staining of the tear meniscus occurred when I briefly touched a dry fluorescein-impregnated paper strip to the lower conjunctival sac. I compared the applanation obtained with both of these techniques with pressures obtained when no fluorescein was used. The completely stained eyes had a mean 5.62 mm Hg increase over the pressures recorded immediately before staining. The partially stained group had a mean 1.68 mm Hg

increase over the pressures recorded immediately before staining. Moses and Lua[11] and Thornburn[12] have shown that repeated applanation can decrease the pressure an average of 0.5 mm Hg. I did not take this effect into consideration because it would have tended to magnify the difference already demonstrated.

Because the use of fluorescein is objectionable to some ophthalmologists and patients, the construction of a tonometer that would take the width of the tear meniscus into account seems desirable. This is probably not practical, however, as the width of the tear meniscus would have to remain constant in all cases. Some factors which can alter the size of the tear meniscus are (1) the viscosity of the tears (tear viscosity may vary from patient to patient and can be altered by the topical application of anesthetics or other medications[13]); (2) the amount of tears (this is variable and can also be influenced by the amount and irritative ability of the topically applied anesthetic); (3) the surface of the tonometer (the finish of the applanating surface can affect the width of the meniscus); (4) the surface of the cornea (the variation in smoothness from one cornea to another can affect the width of the meniscus); (5) the angle between the prism and cornea (this angle varies with the corneal curvature; a flatter cornea causes a broader meniscus than does a steep cornea); and (6) the intraocular pressure (there may be some change in the size of the meniscus as intraocular pressure varies).

Although an exhaustive treatment of fluid dynamics involving the cornea-applanating surface interface is beyond the scope of this article, I hope these comments will serve to illustrate some possible pitfalls in trying to take the width of the meniscus into account when calibrating an applanation prism.

It is apparent from this and other studies[3,5,7,10,12,14,15] that an adequate amount of fluorescein is essential for obtaining accurate applanation pressures; therefore, it only remains to determine which form to use.

Many ophthalmologists prefer to use fluorescein-impregnated paper strips either moistened with some fluid or applied to the conjunctiva dry.[16] One reason for this preference is that individually wrapped strips are sterile. Another reason is that a small amount of fluorescein creates discrete mires, giving a sharp endpoint. However, this technique can underestimate applanation pressure by 4.6 mm Hg.

Grant[10] has recommended that a drop of 0.25% sodium fluorescein solution be applied after the topical anesthetic agent to avoid the variation in fluorescein concentration that occurs with the paper strip method. Grant[10] and Moses[4] have both determined that the optimal concentration of fluorescein is 0.125% to 0.5%. More than 0.5% causes residual staining of the cornea-prism interface and less than 0.125% fails to stain the apex of the meniscus adequately. Fluorescein solution is rarely used today because of fear of contamination with *Pseudomonas aeruginosa*.[17]

Many investigators have advocated combining fluorescein solution with a topically applied anesthetic.[6-9] The use of benoxinate HCl stabilized by polyvinylpyrrolidone has eliminated problems with quenching of the fluorescence and precipitate formation.[9] Adding 1% chlorobutanol provides bacteriocidal activity with the solution in a 1:4 dilution; it is bacteriostatic in a 1:14 dilution.[9] The average time to self-sterilization of 2 ml of the compound after inoculation with 1×10^6 organisms of *P. aeruginosa* was five minutes.[9,18]

Aside from standardized concentration and elimination of an extra step in the procedure, the combined preparation offers several other advantages. The

mires are of an optimal size, defined by Tanton[7] as 10% of the diameter of the circular pattern. The definition of the inner margin of the tear meniscus is much better with the compound containing polyvinylpyrrolidone than with other agents such as methyl cellulose, polyvinyl alcohol, or saline.[9] Polyvinylpyrrolidone by virtue of its increased viscosity induces a slight error in applanation tonometers which employ a constant force over a variable applanation area (Maklakoff type), but this effect is not clinically apparent in applanation tonometers using variable force with a constant applanation area (Goldmann type).[6,19,20] This increased viscosity has the advantage of holding the tear film on the cornea for a longer period of time.[9] Quickert,[9] Holtz,[21] and Stewart[22] have shown that the risk of inducing an infection with this compound is negligible.

Summary

Some investigators have suggested that it is not necessary to use fluorescein when doing applanation tonometry. This is incorrect because, in order to visualize the apex of the tear meniscus which defines the applanated area, the tears must be stained with fluorescein.

I found an average pressure of 11.72 ± 3.20 mm Hg in eyes tested without fluorescein compared with an average pressure of 17.34 ± 4.07 mm Hg when pressures were immediately remeasured with fluorescein.

Failure to use fluorescein during applanation tonometry will result in an underestimation of approximately 5.62 mm Hg.

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RECURRENT KERATOCONUS AFTER KERATOPLASTY

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Keratoconus is an axial ectasia of the cornea of unknown origin. Although keratoconus has been reported in association with a host of localized and systemic disorders, no one association has pointed to a probable pathogenesis; the diversity of associations seems to rule out any single underlying genetic or metabolic defect. Approximately one third of all patients with keratoconus have no apparent systemic disease.

Advanced keratoconus, with contact lens intolerance and poor visual acuity, may be treated surgically with penetrating keratoplasty; clear-graft rates have exceeded 80% in several series.[1-3] We report a case of successful penetrating keratoplasty for keratoconus, complicated 16 years later by keratoconus in the graft.

CASE REPORT

A 35-year-old woman came here in May 1975 with a two-year history of decreased visual acuity in her left eye. She had had bilateral penetrating keratoplasties for keratoconus (left eye, 1959; right eye, 1962). The patient admitted rubbing both eyes with her knuckles before and after her keratoplasties because of itching from an allergic diathesis.

Her best corrected visual acuity was R.E.: 6/9 (20/30) and L.E.: 6/60 (20/200). The left eye showed a well-healed, 8.5-mm corneal graft, Fleischer's ring, vertical subepithelial white lines, Munson's sign, mild central corneal thinning, and stromal scarring (Fig. 1). The lens and media were clear, the intraocular pressure normal, and the retina unremarkable. Keratometry readings disclosed irregular astigmatism of 11 diopters.

The right eye was white and quiet with a clear, 8.0-mm, well-healed penetrating corneal graft. There was no evidence of keratoconus in the right eye; keratometry readings disclosed 1.5 diopters of astigmatism.

We performed an 8.5-mm penetrating keratoplasty on the left eye and examined the excised button with light and electron microscopy.

RESULTS

Routine histopathologic examination of the excised button disclosed thinning, fragmentation, and fibrillation of the basement membrane and Bowman's layer. Gaps in Bowman's layer were filled with elastoid connective tissue (Fig. 2), which extended through the gaps into the epithelium. The stroma showed thinning and fibrillation in areas adjacent to the dehiscences in Bowman's layer. Deep stromal folds were present in Descemet's membrane.

By electron microscopy, we observed many breaks in the epithelial basement membrane, which were filled with downward protrusions from basal epithelial cells (Fig. 3). In some areas, basement membrane material had accumulated in what appeared to be abortive attempts at reformation (Fig. 3). In the stroma, there were many abnormal keratocytes with accumulations of extracellular material in depressions on the cell surfaces (Fig. 4). In some areas, collagen fibrils appeared to be embedded in the surface of these accumulations (Fig. 4). We found drifting lamellae of irregular collagen fibrils (Fig. 5) in the thinned stroma. Colla-

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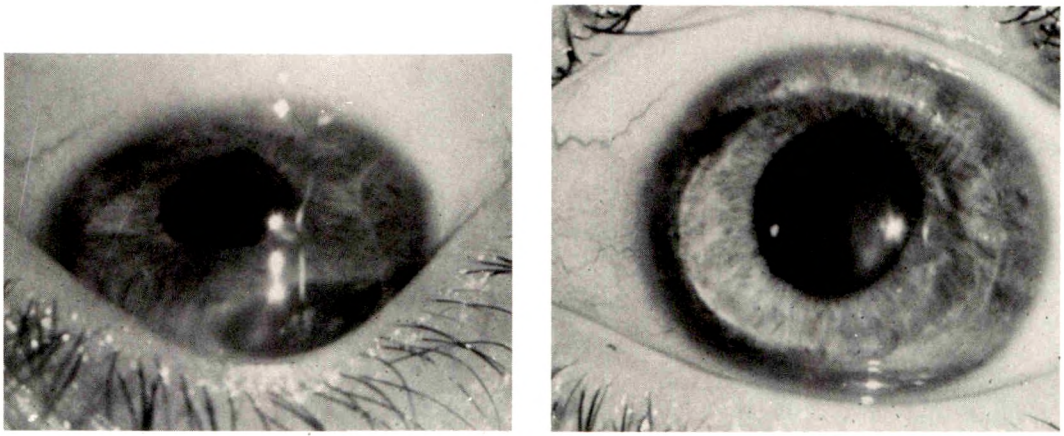


Fig. 1 (Abelson and associates). Left eye of patient with recurrent keratoconus. Left, Munson's sign and corneal configuration characteristic of keratoconus. Right, Stromal scarring.

gen fibrils in some areas were completely degenerated (Fig. 5).

Discussion

This is, to our knowledge, the first documented case report of keratoconus in a penetrating graft. Other accounts of keratoconus in penetrating grafts for keratoconus have appeared in published reports,[4-7] but none of these cases were confirmed histopathologically. The clinical and histopathologic findings in the present case complement each other and are unlike those in any other corneal dystrophy or degeneration.

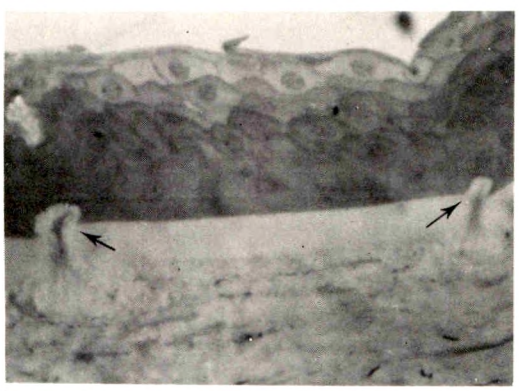


Fig. 2 (Abelson and associates). Section of excised corneal graft. Breaks in Bowman's layer are filled with elastoid connective tissue (arrows) (hematoxylin and eosin, × 600).

The development of keratoconus in the previously successful penetrating graft in this case represents either continuation of a degenerative process present in the donor corneal button at the time of keratoplasty, or true recurrence of keratoconus in the host eye. The incidence of keratoconus in the general population has been estimated at 0.15% to 0.6%,[8] so a keratoconic donor cornea is possible. Assuming that the donor button was normal, the occurrence of keratoconus in the graft may have pathogenetic implications. If the primary defect in keratoconus resides in the basal epithelial cells, as Teng[9] first suggested in 1963, the host epithelium, expected to replace the donor epithelium, may be implicated in the eventual ectasia of the donor button. Changes in the stroma of the button might be caused by a diffusible substance originating in the defective host epithelium.

Alternatively, if the primary defect in keratoconus resides in the stromal collagen itself, as recently suggested by Cannon and Foster,[10] any hypothesis about the sequence of events in this case must

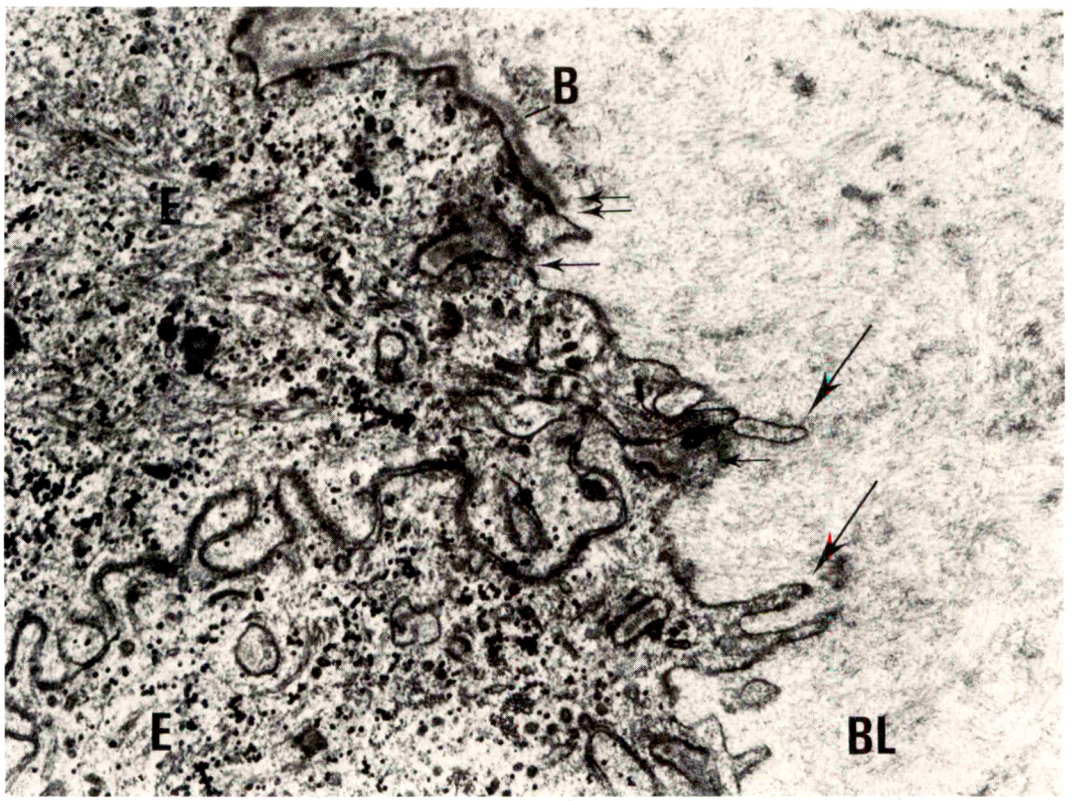


Fig. 3 (Abelson and associates). Section of corneal graft. B, basement membrane; BL, Bowman's layer; E, basal epithelial cells. Breaks in basement membrane (double arrow) are filled with protrusions of basal epithelial cells (long arrows). Note accumulations of basement membrane material (short arrows) (× 23,500).

include a mechanism by which presumably normal donor collagen is exchanged for defective collagen. One such mechanism is a slow but progressive replacement of donor keratocytes by abnormal host keratocytes, leading to abnormal collagen manufacture and eventual ectasia.

There may be an unidentified systemic ectatic factor in the host. The patient admitted to vigorous eye rubbing both before and after her initial keratoplasties; the influence of this rubbing and her allergic diathesis on the recurrence of keratoconus in the left eye can only be conjectured, but both rubbing and allergy are strongly correlated with keratoconus.[11]

The absence of clinically detectable keratoconus in the graft from a different donor in the right eye argues against the hypothetical systemic factor in the host, and can be cited as evidence of a pathologic condition of the donor cornea of the left eye. Unilateral keratoconus is the rule rather than the exception, however, and we think that this case of keratoconus in an initially normal donor graft represents true recurrence of keratoconus. Published reports of other such cases support our thesis, although they lack histopathologic confirmation.

Whatever the specific origin in our case, it is clear that keratoconus can occur in a grafted cornea. As more and more successful grafts are observed for longer periods, similar cases may be noted. Keratoconus of a successful graft should be added to the list of late complications of keratoplasty.

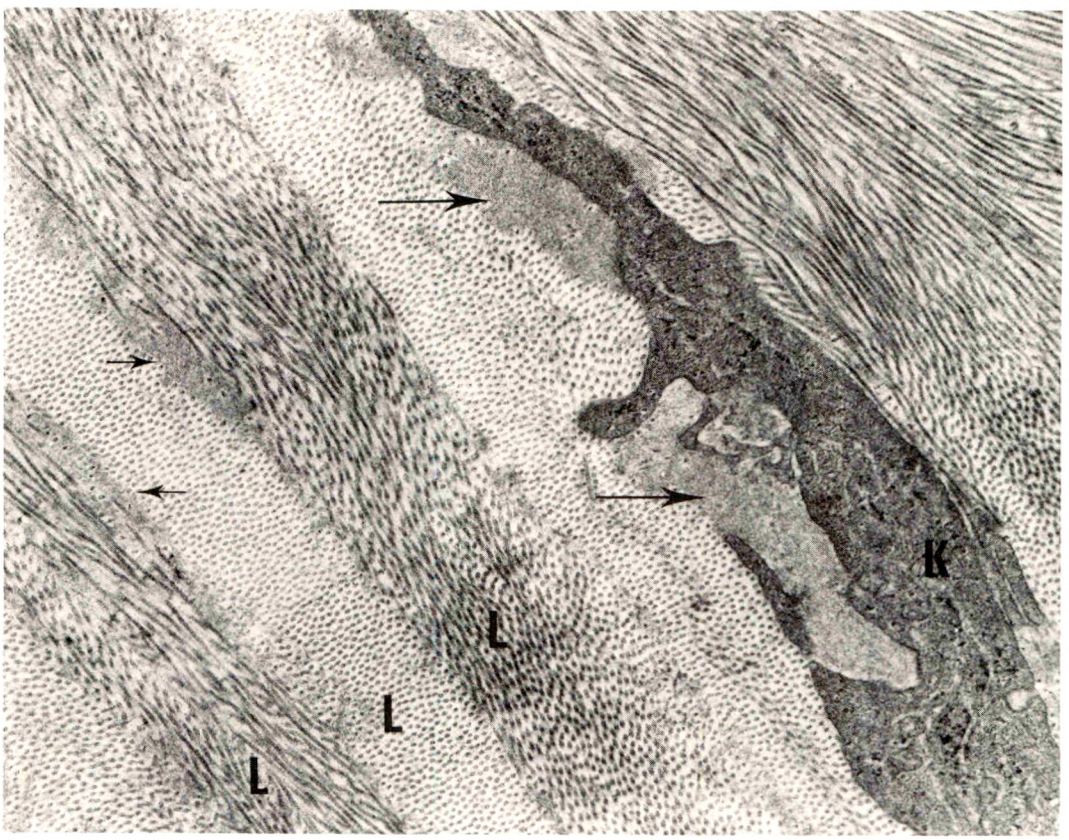


Fig. 4 (Abelson and associates). Stroma of corneal graft. K, keratocytes; L, collagen lamella. There are accumulations of extracellular material in depressions of keratocytes (long arrows). Collagen fibrils appear anchored in surface of extracellular accumulations (short arrows) (× 25,000).

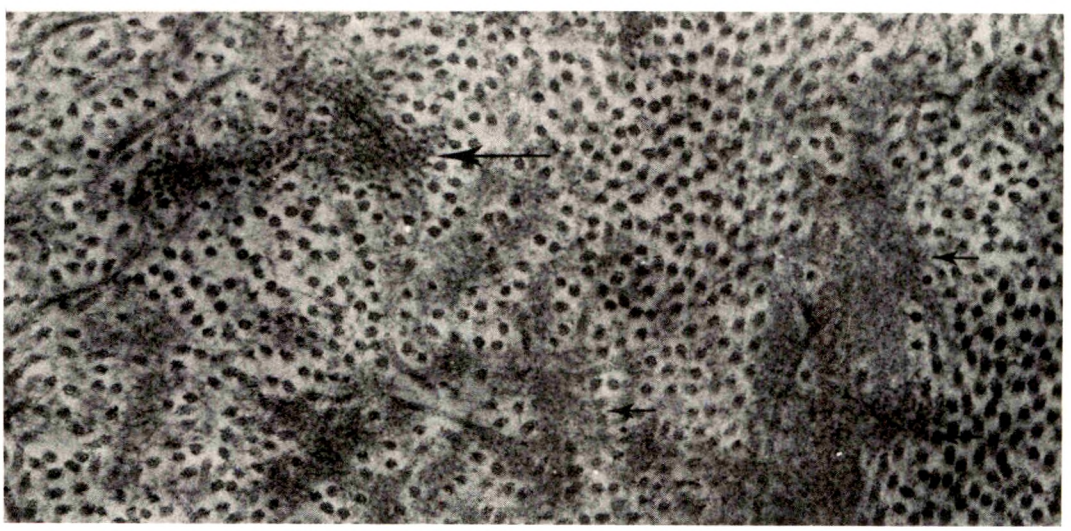


Fig. 5 (Abelson and associates). Stroma of corneal graft. Note fibrillation and degeneration of collagen fibrils, drifting lamella of irregularly sized fibrils (long arrow), and degeneration of normal-sized fibrils (short arrows) (× 59,000).

SUMMARY

A 35-year-old woman who had undergone bilateral penetrating corneal grafts for keratoconus had keratoconus in the eye that had received a graft 16 years earlier. A second penetrating keratoplasty was performed, and the excised button was examined with light and electron microscopy. Both the light and electron microscopic findings were consistent with the clinical diagnosis of keratoconus. Keratoconus in a graft for the same disease should be added to the list of late complications of successful keratoplasties.

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PENETRATING KERATOPLASTY IN PSEUDOPHAKIC BULLOUS KERATOPATHY

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Although the cornea remains clear in most patients who undergo cataract extraction with intraocular lens implantation, a small percentage of patients develop chronic corneal edema, or pseudophakic bullous keratopathy. Estimates range from 0 to 10%.[1,2] Comparative studies suggest that corneal edema occurs significantly more often when cataract extraction is followed by intraocular lens implantation.[3,4] Specular microscopic studies of cataract extraction with intraocular lens implantation have found that endothelial cell loss varies from 7%[5] to 62%.[6] The extent of endothelial cell loss appears to correlate directly with the amount of surgical trauma at the time of lens implantation.[7] Damage to the endothelium occurs as a result of contact with the intraocular lens, leading to cellular disruption.[8] Similarly, contact between the intraocular lens and corneal endothelium may occur during the postoperative period as a late complication, eventually resulting in chronic corneal edema.

There is little information about the effectiveness of penetrating keratoplasty in the management of pseudophakic bullous keratopathy. Fine[9] described a series of 16 patients, 15 of whom had clear grafts after one year; six patients had final corrected visual acuities of 6/12 (20/40) or better. Taylor, Khaliq, and Maxwell[10] reported nine clear grafts in a series of 11. In order to gain more information, we retrospectively studied 25 consecutive patients who underwent penetrating keratoplasties for pseudophakic bullous keratopathy.

SUBJECTS AND METHODS

All 25 patients underwent surgery at the University of Michigan Medical Center between 1977 and 1979. Follow-up time ranged from six to 41 months with a mean of 20 months. The patients' ages ranged from 52 to 82 years with a mean of 71 years. Ten patients were men and 15 were women. Eighteen patients underwent intracapsular cataract extraction and seven extracapsular extraction. The intraocular lenses were implanted at the time of cataract extraction in 24 patients; one patient had a lens implanted two years after cataract surgery. The implants included nine Medallion lenses with iris suture fixation, eight Fyodorov (Sputnik) lenses, four Copeland lenses, and four Binkhorst four-loop lenses.

Ten patients required secondary operative procedures after intraocular lens implantation. These included five patients with surgical repositioning of the lens implants, three patients with discission of retrolens membranes, and two patients with wound leak repairs.

The time from intraocular lens implantation to onset of corneal edema ranged from 0 to 60 months (mean, 13 months). In the 15 patients who did not undergo postimplantation procedures, time to onset of corneal edema ranged from 0 to 60 months (mean, 18 months). In contrast, ten patients who did undergo

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postimplantation procedures experienced onset of corneal edema from 0 to 13 months later (mean, five months).

The keratoplasty procedure was essentially the same as that used in the aphakic eye.[11] We were careful, however, to avoid contact between the intraocular lens and donor corneal endothelium. We routinely used a Flieringa ring. We incised the edematous host cornea to the level of the deep stroma with an 8.0-mm trephine, avoiding entrance into the anterior chamber. We aspirated 0.5 ml of fluid vitreous through the pars plana; this resulted in a soft eye with the lens implant well behind the corneal plane. We entered the anterior chamber with a razor-blade knife and excised the edematous corneal button with curved corneal scissors. We prepared a disparate-sized donor corneal button with 8.5-mm trephine, punching the donor cornea from the endothelial side. Donor corneas had been stored in McCarey-Kaufman medium. We sutured the donor corneal button into position with either 16 interrupted 10-0 nylon sutures or double running sutures of 10-0 and 16- to 18-μ nylon.[12] After fluid vitreous aspiration, the intraocular lens remained well back in all cases, allowing a large bubble of air to protect the donor endothelium during graft suture placement. After suturing the graft into position, we aspirated the air bubble and filled the anterior chamber with balanced saline solution. We injected antibiotics and corticosteroids subconjunctivally at the end of the surgical procedure.

We removed the intraocular lens at the time of keratoplasty in 11 patients either because of preoperative contact of the implant with the cornea which we could not correct or because of chronic uveitis. The implants we removed at keratoplasty included six Medallion lenses, four Sputnik lenses, and one Copeland lens. We had no difficulties with removal of the lens implants. We easily lysed iris, vitreous, and lens capsule adhesions from the implant by blunt dissection, retaining a round pupil. After removing the lens implant, we routinely performed anterior vitrectomy with a vitreous cutting instrument. We used cellulose sponges to ensure that no vitreous strands remained in the anterior chamber. We then prepared the donor corneal button and sutured it into position as described. We implanted no secondary intraocular lenses at the time of keratoplasty.

Results

The penetrating keratoplasty procedures outlined were no more difficult to perform than routine aphakic keratoplasty. In no case was there detectable contact between the lens implant and graft endothelium during or after surgery. We easily controlled postoperative inflammation with 1% prednisolone acetate drops instilled four times per day. The remainder of the postoperative course was essentially the same as that after aphakic keratoplasty with frequent postoperative visits and close observation. We removed the continuous 10-0 nylon sutures three months after surgery while we left the 16- to 18-μ nylon suture in place. We removed the interrupted 10-0 nylon sutures one year after surgery. We found no cases of wound separation. We prescribed spectacles or contact lenses, or both, when repeat refractions became stabilized.

Of the 25 corneal transplants, 22 (88%) remained clear. Irreversible corneal clouding caused by allograft rejection occurred in three cases (12%) at two, four, and nine months after keratoplasty. There were no cases of primary donor tissue failure, infection, or failure caused by mechanical problems.

Glaucoma occurred in seven patients (28%) during the postoperative period.

Of these, four had medically controlled glaucoma before keratoplasty; three additional patients developed increased intraocular pressure after keratoplasty which required treatment. All seven patients required continued medical therapy; none required surgical intervention or cyclocryotherapy for control of intraocular pressure.

We found no differences in final visual acuities between patients whose lens implants were removed at keratoplasty and patients whose lens implants were not removed (Table).

All patients with clear corneal transplants and normal retinas achieved final visual acuities of 6/12 (20/40) or better. When final visual acuity was less than 6/12 (20/40), the reason was readily apparent. Cystoid macular edema was present in six patients, corneal clouding secondary to allograft rejection in three, senile macular degeneration in two, and retinal detachment in one.

Discussion

Pseudophakic bullous keratopathy may be defined as chronic corneal edema caused by endothelial decompensation and resulting in epithelial bullae in a pseudophakic eye. Symptoms include decreased visual acuity, frequently worse in the morning, caused by corneal stromal and epithelial edema, opacification, and irregular surface astigmatism. Pain, caused by rupture of epithelial bullae, is a common complaint. Slit-lamp examination shows that the epithelium is thickened and edematous with bulla formation; a subepithelial connective tissue pannus may be present in chronic cases. The corneal stroma appears gray, thickened, and edematous, and frequently has superficial peripheral vascularization. Descemet's membrane and endothelium appear wrinkled and thickened with guttate excrescences.

TABLE

Final visual acuities

Visual Acuities	No.	%
All patients	25	—
6/6 (20/20) to 6/12 (20/40)	13	52
6/15 (20/50) to 6/30 (20/100)	4	16
6/60 (20/200) or worse	8	32
Lens implants retained	14	—
6/6 (20/20) to 6/12 (20/40)	7	50
6/15 (20/50) to 6/30 (20/100)	2	14
6/60 (20/200) or worse	5	36
Lens implants removed	11	—
6/6 (20/20) to 6/12 (20/40)	6	55
6/15 (20/50) to 6/30 (20/100)	2	18
6/60 (20/200) or worse	3	27

Pseudophakic bullous keratopathy may occur in the early or late postoperative period after cataract extraction with intraocular lens implantation. Patients who require secondary postimplantation surgical procedures appear to develop bullous keratopathy sooner, as shown by ten patients who developed corneal edema at an average of five months, compared to 15 patients without postimplantation procedures whose edema occurred at an average of 18 months after lens implantation. Undoubtedly, the underlying lens implant problem requiring secondary surgical correction causes additional endothelial damage leading to chronic corneal edema earlier in the postimplantation period. Patients without postimplantation surgical procedures may develop chronic corneal edema as a result of endothelial cell attrition caused by aging, with cell loss enhanced by surgical trauma at the time of lens implantation and postoperative uveitis.

In the treatment of pseudophakic bullous keratopathy, the timely removal of an obvious underlying cause, such as an anteriorly dislocated lens implant, may allow spontaneous resolution without development of chronic corneal edema. Topically applied corticosteroids may

help suppress the inflammatory component of temporary corneal edema, but are of little benefit in chronic edema. Topically applied hypertonic agents may temporarily reduce edema early in the disease process, but eventually are of decreasing benefit. Reduction of intraocular pressure may be effective if intraocular pressure is increased; its effect is small and transient when intraocular pressure is normal. Hydrophilic contact lenses may reduce discomfort and may improve visual acuity in early cases. A conjunctival flap or Bowman's membrane cautery may relieve pain in severe cases, but may further reduce visual acuity. Because penetrating keratoplasty may relieve pain and restore visual acuity, it appears to be the best treatment for pseudophakic bullous keratopathy.

The success rate of penetrating keratoplasty in pseudophakic bullous keratopathy, 88% in this series, was similar to that reported in aphakic bullous keratopathy.[13] The lens implant, whether retained or removed at keratoplasty, did not appear to adversely affect final graft clarity. Postoperative complications, including graft rejection and glaucoma, were similar to those in aphakic grafts. Final visual acuity was 6/12 (20/40) or better in all patients with clear grafts and normal retinas. Intraocular lens removal or retention at keratoplasty did not appear to affect final visual outcome. Visual acuity was limited by retinal pathology (cystoid macular edema, senile macular degeneration, and retinal detachment) in nine (36%) patients.

The anatomical results with clear corneal grafts in penetrating keratoplasty for pseudophakic bullous keratopathy were excellent. The visual results, however, were somewhat less satisfying because of accompanying retinal pathology. If retinal pathology could be eliminated, the prognosis for rehabilitation of patients with pseudophakic bullous keratopathy by penetrating keratoplasty would be excellent.

Summary

We studied 25 consecutive patients who underwent penetrating keratoplasty for pseudophakic bullous keratopathy. Follow-up time averaged 20 months. Ten patients required postimplantation surgical procedures because of complications after lens implantation. The time from lens implantation to onset of corneal edema averaged 18 months in patients without postimplantation surgical procedures and five months in patients with postimplantation procedures. The lens implants were removed in 11 patients at keratoplasty. Corneal clouding caused by graft rejection occurred in three patients, while 22 grafts remained clear. Final visual acuities were 6/6 (20/20) to 6/12 (20/40) in 13 patients, 6/15 (20/50) to 6/30 (20/100) in four patients, and 6/60 (20/200) or less in eight patients. We found no difference in final visual acuity between patients with lens implants removed at keratoplasty and those with lens implants left in place. Significant retinal pathology included cystoid macular edema, macular degeneration, and retinal detachment.

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Ophthalmic miniature

It is impossible to overestimate the importance of this event on the boy's maturing sensibilities. Through the miraculous little windows that now gripped his nose, the world leaped into pristine focus, disclosing an infinity of detail, of color, of nuance, and of movement just when the screen of his mind was at its most receptive. One of the best features of his adult descriptive writing—an unsurpassed joy in things seen—dates back to this moment; while another—his abnormal sensitivity to sound—is surely the legacy of the myopic years that came before.

Edmund Morris, *The Rise of Theodore Roosevelt*
New York, Random House, 1979

TOXIC CORNEAL EPITHELIAL EDEMA FROM EXPOSURE TO HIGH ATMOSPHERIC CONCENTRATION OF TOLUENE DIISOCYANATES

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Corneal toxicity caused by exposure to atmospheric concentrations of organic polyisocyanates has not, to our knowledge, been previously described. This report describes corneal edema in employees of a polyurethane-producing company who were exposed to high atmospheric concentrations of organic isocyanates.

CASE REPORTS

All patients were employees of the polyurethane-foam-producing company and were seen in the Department of Ophthalmology at the University of Kentucky Medical Center.

Nine people had a complete ophthalmic examination. Abnormalities were limited to the cornea and conjunctiva. The first four patients were seen because of specific ocular symptoms associated with their environment at work. The remaining five patients worked in the same environment and were examined upon our request.

Case 1—A 29-year-old man had decreased visual acuity and red, irritated eyes. He had been employed as the manager of the company for three weeks. He noted that all employees who worked in the areas with high atmospheric concentrations of organic isocyanates were complaining of eye irritation and blurred vision. He also stated that greater amounts of prepolymer chemicals and finished premold, a polyurethane foam material, were being stored in the building because of increased production and, additionally, that the hot weather was straining an inadequate ventilation system. An ocular examination 14 hours after his last exposure showed a visual acuity of R.E.: 6/6 (20/20) and L.E.: 6/4.5 (20/15) with moderate corneal epithelial edema and conjunctival injection of both eyes concentrated in the palpebral fissures (Fig. 1). He did not return for follow-up.

Case 2—A 27-year-old man had blurred vision at work. He had begun work ten days before examination. One day later he noticed foggy vision which cleared over the weekend but recurred when he returned to his job. An ocular examination 14 hours after exposure disclosed a visual acuity of R.E.: 6/12 (20/40) and L.E.: 6/9 (20/30) with moderately severe microcystic edema of both corneas. He did not return for follow-up.

Case 3—A 48-year-old woman had loss of vision in both eyes of one day's duration. She had begun work three weeks previously at the company and had noticed a decrease in visual acuity "like being in a smoky room" three days before her initial visit. An ocular examination disclosed a visual acuity of no light perception in either eye, although she had a normal response to optokinetic targets in each eye. We found severe microcystic edema of the epithelium of each cornea and moderate injection of the conjunctiva with accentuation in the exposed area of

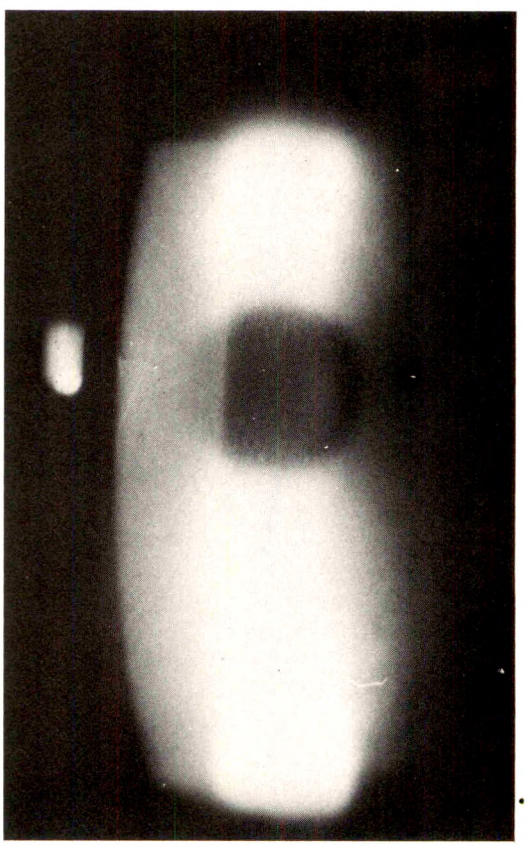


Fig. 1 (Luckenbach and Kielar). Case 1, right eye. Corneal epithelial edema.

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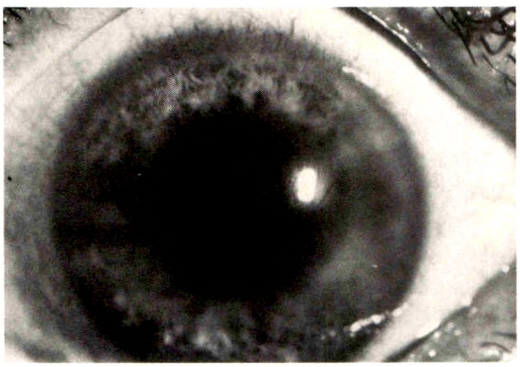


Fig. 2 (Luckenbach and Kielar). Case 3, left eye. Conjunctival injection and corneal edema accentuated in palpebral fissure.

the palpebral fissure (Fig. 2). We could not perform ophthalmoscopy adequately in the left eye because of the severe corneal edema. We believed the visual acuity of no light perception was a conversion reaction. During a six-day period, the microcystic edema of the right eye cleared, and visual acuity improved to 6/7.5 (20/25). Although the left eye had no abnormality except minimal microcystic edema eight days after her initial examination, her subjective visual acuity remained no light perception. She did not return for follow-up.

Case 4—A 40-year-old woman had blurred vision at work. She had begun working at the company two weeks before examination and had noticed hazy vision each evening which at first resolved overnight. However, by the middle of her second week, the haziness did not resolve. Ocular examination 38 hours after her last exposure disclosed a visual acuity of 6/7.5 (20/25) in each eye with moderate corneal epithelial edema and conjunctival injection of both eyes. On reexamination after three days without exposure, both corneas and conjunctivas were normal.

Case 5—A 33-year-old man had begun working in a similarly exposed area two years previously. At the time of examination, he was helping the local company for a few weeks, doing office work in an enclosed area of the building where there was no exposure to organic isocyanates. In the past, when he worked in an area of exposure, his vision had become very cloudy, requiring a weekend to clear. He estimated that for the past two years he had spent an average of two days per week with foggy vision. Ocular examination disclosed a visual acuity of 6/6 (20/20) in each eye and a complete examination, including a normal Schirmer I tear test and tear break-up time, showed no abnormalities.

Case 6—A 20-year-old woman had been working for several months when the company decided to change to the manufacture of premold. She remembered having been warned about the new product and how it would make her eyes "foggy." She claimed that after one day of excessive exposure it might take three to four days to clear. Ocular examination, including Schirmer I tear test and tear break-up time, seven days after exposure disclosed a visual acuity of 6/6 (20/20) in each eye with no abnormalities. Pachymetry showed corneal thicknesses of R.E.: 0.78 mm and L.E.: 0.76 mm. She later returned, after 1.5 days of exposure, with a visual acuity of R.E.: 6/7.5 (20/25) and L.E.: 6/6− (20/20−). She also had mild microcystic edema and conjunctival injection of both eyes, most pronounced in the palpebral fissures. Pachymetry measurements were R.E.: 0.80 mm and L.E.: 0.78 mm.

Case 7—A 30-year-old woman who had worked in premold production for one year gave a history of foggy vision clearing in one to three days. The fogginess occurred primarily when she was trimming the newly formed foam and when finished premold was stored in the building. She claimed goggles partially eliminated the foggy vision. Ocular examination after one day of exposure disclosed a visual acuity of 6/6− (20/20−) in each eye, with moderate microcystic edema and conjunctival injection. Results of the remainder of the ocular examination, including Schirmer I tear test, were normal. Pachymetry readings were 0.64 mm in each eye. Ocular examination 12 hours later, after no further exposure, disclosed persistent microcystic edema and conjunctival injection.

Case 8—A 19-year-old woman had worked for seven months, beginning as a trimmer. On the worst days her "smoky vision" required five days to clear; at other times it took a weekend to return to normal. She claimed the goggles only helped "a little." Ocular examination after one day of exposure disclosed a visual acuity of R.E.: 6/6 (20/20) and L.E.: 6/7.5 (20/25) with a trace of microcystic edema and no conjunctival injection in either eye. Results of a Schirmer I tear test and tear break-up time were normal. Pachymetry measurements were 0.76 mm in each eye. She returned at the end of one week of more extensive exposure with a visual acuity of R.E.: 6/6− (20/20−) and L.E.: 6/9 (20/30) with moderate microcystic edema and mild conjunctival injection of each eye. Pachymetry measurements were R.E.: 0.72 mm and L.E.: 0.70 mm.

Case 9—A 21-year-old woman had worked for three months pouring the prepolymer chemicals into the mold and complained that her foggy vision usually took two days to clear. She often wore goggles during the hot part of the afternoon. This seemed to help. She complained that the smoky vision was cumulative, so that at the end of the work week she needed someone to drive her home. Ocular examination after two days of exposure disclosed a visual acuity of 6/4.5− (20/15−) in both eyes, mild microcystic edema and conjunctival injection, and a normal Schirmer I tear test and tear break-up time. Pachymetry measurements were R.E.: 0.60 mm and L.E.: 0.58 mm.

Discussion

Organic diisocyanates are toxic, volatile, and highly reactive chemicals neces-

sary for the production of molecular-weight polymers in the form of laquers, adhesives, rigid or elastic polyurethane foams, synthetic rubbers, and a variety of plastics. The finished product is so physiologically inert that polyurethane is commonly used within the body without inciting an inflammatory reaction.[1] However, Bruckner and associates[2] found that isocyanates in vapor form inflame mucous membranes, especially the conjunctiva and respiratory tract, by chemical irritation. Peters and associates[3] found that long-term exposure to levels measuring less than one-half the current government threshold causes a significant decrease in ventilatory capacity, as demonstrated by acute and cumulative changes in the forced expiratory volume in one second on pulmonary function tests. Allergic skin sensitization and respiratory sensitization have also occurred.[1] In higher concentrations, various respiratory symptoms and asthmatic wheezing syndromes are common.[2] Axford and associates[4] found that a single severe exposure to toluene diisocyanates can cause acute gastrointestinal complications such as nausea and vomiting, abdominal discomfort, and diarrhea. In another report, LeQuesne and associates[5] described euphoria, ataxia, and loss of consciousness after acute exposure, followed by headache, difficulty in concentrating, poor memory, and confusion. Most of the affected patients still complained of a poor memory, personality change, irritability, or depression four years after exposure. No ocular findings in humans other than a chemical conjunctivitis have been described previously, but it is quite possible that corneal epithelial edema was overlooked in previous studies as slit-lamp examinations were not performed.

The company employing the individuals described in this report used a common method of polyurethane manufacture which has been described by Duncan and associates.[6] The premold is produced by pouring mixture A, a prepolymer, and mixture B, a polymeric isocyanate, into a mold. A reaction occurs, producing carbon dioxide which is trapped within the plastic, producing a foam. An excess of isocyanate is usually present as the mixture heats up, becomes more viscous and then more rigid, and finally sets. At this point, the excess is trimmed away and the polymer is removed from the mold, although the foam continues to "cure" and harden for 24 hours.[1] Peters and associates[3] proved that the atmosphere in buildings closest to pouring and trimming areas has substantially higher concentrations of isocyanates than other areas.

The results of our study indicate that another potential source of substantial concentrations of isocyanates may be in the gases released when the finished parts are "curing." Swarbrick[7] stated that keeping released isocyanate levels below the threshold requires that a building have effective ventilation (ten air changes per hour), an effective fume extraction system, and local exhaust facilities for molding and casting of foam. We did not measure actual atmospheric concentrations of isocyanates, but the symptoms of the employees exposed to isocyanates in areas of inadequate ventilation suggest that they were above the threshold of .02 parts per million. Atmospheric concentrations rose when unusual quantities of finished premold were stored in these areas.

We found that significant corneal microcystic edema occurred in every employee exposed to high atmospheric concentrations for one working day. Clearing time after moderate exposure varied from 12 to 48 hours, but the toxicity caused by repeated, daily exposure often required several days to resolve. Moderate amount of corneal toxic

microcystic edema caused an average fall of one line of visual acuity. Pachymetry measurements never varied more than 9% when the edema was present compared to when it was not. There was no clinical evidence of stromal edema in any patient. The actual severity of the microcystic edema was directly related to proximity to the area where foam was produced, length of exposure, inadequacy of ventilation, increasing temperature in the building, increasing quantity of prepolymer chemicals and finished premold stored inside, and, perhaps, the cumulative exposures without time to clear between. We found no long-term ocular effects in the patients who had long histories of repeated exposure.

Vapors of alcohol, amines, diethyl diglycolate, diisopropylamine, 3-dimethylaminopropylamine, ethylenediamine, N-ethylmorpholine, N-methylmorpholine, morpholine, nitronaphthalene, tetramethylbutanediamine, and triethylenediamine have produced delayed corneal epithelial edema[8]; however, none of these substances were used at the polyurethane-foam-producing company described in this report.

The mechanism by which toluene diisocyanates produce corneal edema is unknown. Corneal epithelial edema can be produced by epithelial or endothelial cell dysfunction, or both. Smelser[9] concluded that hydration of the cornea depends on the metabolic activity of the endothelial and epithelial cells which actively transfer water or salts from the cornea, requiring oxygen and metabolites and expending energy to do so. The endothelium plays the major role,[10] acting as a mechanical barrier and maintaining an active transport system to pump water from the stroma to the aqueous humor.[11] Ytteborg and Dohlman[12] found that when endothelial cell dysfunction is responsible for corneal edema, fluid first leaks into the stroma, finally causing epithelial edema only after hydration has increased the stromal thickness to approximately 30% above normal. Endothelial dysfunction can thus be ruled out as the cause of the corneal epithelial edema in the present study, as the pachymetry measurements indicated no significant increase in corneal thickness.

Tear film abnormalities must also be considered as a possible cause of the corneal epithelial abnormalities. Smelser[9] found that the precorneal tear film aids corneal deturgescence by serving as the source of atmospheric oxygen for the anterior cell layers. Additionally, Mishima[13] concluded that the tears form a hypertonic osmotic pressure gradient to draw water and solutes from the epithelium. We did not measure the oxygen level and tonicity of the tear fim. However, results of the Schirmer I test and tear film break-up time were normal.

This study demonstrated that, in the case of toluene diisocyanate toxicity, corneal epithelial edema signifies a breakdown of the system that regulates the fluid content of the epithelial tissue alone. The basal layer of epithelial cells is metabolically active, producing energy aerobically to transport water and solutes out of the anterior stroma into the tears. Interference with the normal aerobic metabolism of the cornea results in epithelial edema. Toluene diisocyanate is a highly reactive chemical which interferes with normal cell metabolism. Davies and associates[14] have shown that toluene diisocyanate inhibits formation of 3′, 5′-cyclic-adenosine-monophosphate. Neufeld and Sears[15] demonstrated a high concentration of cyclic-adenosine-monophosphate in corneal epithelium. Possibly this lipid-soluble[16] chemical penetrates the corneal epithelium, is toxic to the metabolically active basal cell layers, and effectively decreases the energy produced for the transport system, perhaps involving the production of cyclic-adenosine-

monophosphate, thus resulting in the same type of corneal edema as that seen when the epithelium is forced to function anaerobically.

Summary

Exposure to organic isocyanates in the atmosphere of a polyurethane-foam-producing company resulted in microcystic corneal epithelial edema and conjunctival injection localized primarily in the palpebral fissure.

The degree of microcystic edema and decreased visual acuity appeared to be related to location, length of exposure, ventilation, temperature, and amount of chemicals and premold stored. Clearing time varied with amount of exposure. Moderate amounts of microcystic edema caused an average fall of one line in the visual acuity. Pain was absent. The toxic effect of the isocyanates on the metabolism of the corneal epithelium may be the cause of the corneal epithelial edema.

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MULTIPLE REGRESSION ANALYSIS PREDICTOR MODELS IN EXOTROPIA SURGERY

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The ability to determine preoperatively the amount of surgery required to produce the desired postoperative result has been the goal of strabismus surgeons over the years. The accumulated clinical experience, based on trial and error, of many skilled surgeons has provided general guidelines for exotropia surgery.[1-4] However, in recent years more accurate methods have been suggested.[5] Scott and associates[6-8] advocated the use of computer-derived statistical models as an aid to the strabismus surgeon. Our study was undertaken to evaluate the usefulness and limitations of multiple regression analysis predictor models in exotropia surgery.

MATERIAL AND METHODS

The methods of Scott, Mash, and Jampolsky[6] were the basis of our retrospective study. We randomly selected the ophthalmic records of 30 patients with exotropia operated on at the Hadassah University Hospital between 1967 and 1977. Criteria for inclusion in the study included: (1) primary deviation of intermittent exotropia; (2) no previous surgery; (3) surgical correction by symmetrical bilateral recession of the lateral recti muscles; (4) age less than 20 years; and (5) a follow-up period of at least seven months.

Table 1 lists the predictor variables chosen for analysis and compares our patients to those of Scott, Mash, and Jampolsky.[6] The estimates of millimeters of surgery were based on the clinical experience and judgment of senior members of the Pediatric Ophthalmology Department. Computer analysis was carried out by one of us (E. B.) using step-wise multiple regression analysis (SPSS Sub-Program[9]). We eliminated all variables found to be nonsignificant. We obtained a final statistically valid model in which one dependent variable, the effect per millimeter (difference in prism diopters between preoperative and postoperative deviations per millimeter of surgery performed), was found to be a function of four independent predictor variables with their associated weighting factors (partial regression coefficients). We calculated r^2, the coefficient of determination, which reflects the overall accuracy of the predictor equation representing the proportion of variation explained by variance of the predictor variables.

RESULTS

We used multiple regression analysis to construct a valid statistical model (Table 2). The dependent variable, the effect per millimeter in symmetrical bilateral recession surgery, was determined by the magnitude of the larger preoperative deviation, the average corrected visual acuity, the degree of anisometropia, and the average spherical equivalent. We found that the coefficient of determination, r^2, was .73.

DISCUSSION

In order to reduce the guesswork in strabismus surgery, accurate predictor

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TABLE 1

COMPARISON OF SAMPLE POPULATIONS USED FOR MULTIPLE REGRESSION ANALYSIS MODELS

Characteristic	Present Study (N=30) Mean	Present Study (N=30) S.D.	Scott, Mash, and Jampolsky[6] (N=54) Mean	Scott, Mash, and Jampolsky[6] (N=54) S.D.	Student's *t* Value	P Value
Age	9.2	±0.8	5.9	±0.5	3.630	.001
Magnitude of larger deviation	29.9	±1.9	25.5	±1.0	2.227	.05
Magnitude of smaller deviation	15.0	±2.4	10.1	±1.2	2.377	.02
Difference between larger and smaller deviations	15.2	±2.0	15.4	±1.2	0.097	.40*
Visual acuity	0.76	±0.03	—	—	—	—
Average spherical equivalent	0.29	±0.19	0.18	±0.14	0.462	.70*
Anisometropia	0.34	±0.07	0.57	±0.11	1.451	.20*
Change in deviation with upgaze	−0.1	±2.0	0.9	±0.5	0.609	.60*
Change in deviation with downgaze	7.7	±1.4	0.6	±0.6	5.320	.001
Millimeters of surgery performed	12.3	±0.4	10.7	±0.3	3.150	.005

* Not significant.

models based on statistical methods are necessary. However, the validity and usefulness of multiple regression analysis applied to exotropia surgery have yet to be demonstrated. We obtained a statistically valid model with the same methods as Scott, Mash, and Jampolsky,[6] but found different predictor variables to be significant (Table 2). Scott, Mash and Jampolsky[6] found age, the difference between near and distance deviations, and the change in deviation with upward and downward gaze to be significant; we did not. We found visual acuity and anisometropia to be significant, whereas they did not. Furthermore, one predictor variable, average spherical equivalent, had opposite effects in the two models. Our model required less surgery in more severe myopia since the effect per millimeter of surgery was greater. This observation is supported by Bedrossian[10] who cautioned against overcorrections in patients with myopic exotropia. Scott, Mash, and Jampolsky's[6] model required more surgery in more severe myopia since the effect per millimeter was less.

The observed differences in the predictor variables in the two models were undoubtedly caused by differences in the sample populations from whom the data were derived. Table 1 shows that the two

TABLE 2

COMPARISON OF MULTIPLE REGRESSION ANALYSIS MODELS FOR PREDICTION OF EFFECT PER MILLIMETER IN BILATERAL RECESSION SURGERY*

Predictor Variable	Partial Regression Coefficient	×	Patient's Measure	=	
Present study (r^2 = .73)					
Magnitude of preoperative larger deviation	0.0411	×	—	=	
Average corrected visual acuity	−3.3268	×	—	=	
Anisometropia	−0.7600	×	—	=	
Average spherical equivalent	−0.2277	×	—	=	
y intercept	Sum = effect per millimeter			=	3.6133
Scott, Mash, and Jampolsky[6] (r^2 = .96)					
Preoperative deviation for distance	0.0544	×	—	=	
Difference between distance and near deviations	0.0164	×	—	=	
Age	−0.0692	×	—	=	
Average spherical equivalent	0.0473	×	—	=	
Change in deviation with upgaze	0.0071	×	—	=	
Change in deviation with downgaze	0.0199	×	—	=	
y intercept	Sum = effect per millimeter			=	1.0490

*Calculation: millimeter of surgery for each lateral rectus muscle = $\frac{\text{Preoperative deviation} \times 0.5}{\text{Effect per millimeter}}$

sample populations differed significantly with respect to the following variables: age, magnitude of larger and smaller deviations, difference in deviation with downgaze, and the amount of surgery performed. Therefore, one must question the universality of current predictor models in which the significant predictor variables vary according to the sample population. One difficulty was the small size of the sample populations (No. = 30) used to develop the two models. The sample population must be enlarged so that the validity of the model can be increased.

Two possible sources of error in multiple regression analysis are multicollinearity and measurement unreliability. If the independent variables are highly intercorrelated, then multiple regression analysis is weakened by diminished reliability of the partial regression coefficients and marked fluctuation of regression coefficients from sample to sample.[9] If there is a correlation in the general population between average spherical equivalent and age or corrected visual acuity, then the validity of the current models may be reduced. Furthermore, partial regression coefficients are attenuated toward zero by measurement unreliability.[11] Therefore, reliably measured independent variables (age, spherical equivalent, anisometropia) influence the dependent variable,

effect per millimeter of surgery, more than less reliably measured independent variables (preoperative deviation, variation in deviation with upgaze and downgaze). The clinical significance of these possible sources of error in the current exotropia models is difficult to assess, but must be considered when evaluating the usefulness of applying multiple regression analysis to the problem of exotropia.

The current multiple regression analysis models in exotropia surgery are designed to predict only one thing—the effect per millimeter (that is, change in squint angle per millimeter of surgery performed). They are not designed to predict postoperative clinical success (residual angle of 0 ± 10 prism diopters) unless there is a direct correlation between effect per millimeter and clinical success. Current multiple regression analysis models assume such a correlation but controlled clinical trials are needed to prove it.

Discriminant analysis represents an alternative computer-based multivariate statistical method[12] that may prove useful. This method enables the surgeon to choose preoperatively the amount of surgery most likely to produce clinical success in exotropia. The application of this method requires the construction of a statistical model based on analysis of the surgical results in a large series of patients with exotropia and a determination of those variables, including amount of surgery, best able to discriminate between clinical success and failure. The computer could then develop a discriminant function, and a discriminant coefficient derived for each variable similar to the partial regression coefficients in multiple regression analysis. A discriminant score could then be calculated for any new patient with exotropia by adding the products of the discriminant coefficients and the patient's variables as measured preoperatively. This discriminant score would measure the probability of a successful outcome in this new patient compared to the group of successful cases used to develop the model. The surgeon could then choose the amount of surgery that would yield a discriminant score associated with the greatest likelihood of postoperative clinical success.

Statistical models are only of value if they accurately describe reality and possess demonstrated predictive value. Valid statistical models to improve surgical accuracy and reduce the number of second operations in strabismus surgery are important. However, multiple regression analysis applied to exotropia surgery must be used cautiously. The ultimate predictive value of multiple regression analysis must be demonstrated in future controlled clinical trials.

Summary

We developed a statistical model based on multiple regression analysis from a sample population of 30 patients with intermittent exotropia who underwent bilateral recession of the lateral recti muscles. We also identified predictor variables, which accounted for most of the change in deviation per millimeter of surgery performed. The application of multiple regression analysis to the problem of exotropia may be limited by variation in predictor variables according to sample population, multicollinearity, measurement unreliability, and the unproven assumption that the preoperative calculation of effect per millimeter correlates well with postoperative clinical success. Controlled clinical trials are needed to determine the value of multiple regression analysis in exotropia surgery. Discriminant analysis may represent an alternative statistical technique with application to this particular problem.

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Ophthalmic miniature

"My eyes have been a little weak."

"Let me see your teeth." He glanced at them, and again made the gentle, clicking sound of sympathy and disapprobation.

"Now your eye." He lit a lamp at the patient's elbow, and holding a small crystal lens to concentrate the light, he threw it obliquely upon the patient's eye.

Sir Arthur Conan Doyle,
Round the Red Lamp

ORBITAL SOCKET PAIN AFTER INJURY

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Socket secretions, superior sulcus deformities, enophthalmos, unsatisfactory motility, lower eyelid laxity, and migration of the implant are the most common sequelae of enucleation.[1] Severe orbital socket pain unrelated to the prosthesis is unusual.[1,2]

We studied a case of persistent superior orbital socket pain after enucleation. Histological examination demonstrated a neuroma with cartilaginous metaplasia.

CASE REPORT

A 55-year-old man had excruciating pain in his left orbit which had been enucleated four years previously. During the Korean War, the patient had suffered a shrapnel injury which caused corneal perforation and traumatic cataract. The left globe developed a total retinal detachment and neovascular glaucoma which could not be controlled medically. Twenty-four years after the original injury, the patient had been given a retrobulbar injection of 100% alcohol for intolerable, poorly localized orbital pain. After the injection, the patient noted that the quality and location of the pain had been modified. The vague, aching pain had become a sharp, stabbing sensation confined to the distributions of the supraorbital and supratrochlear nerves. Because of this continued discomfort, the left globe was enucleated and a spherical implant inserted.

The pain above the patient's left eyebrow persisted. On four separate occasions over a four-year period, the ocular prostheses had been refitted in an attempt to make him more comfortable. Trial periods without any prosthesis in his socket had also failed to relieve his discomfort.

When we examined him four years after the enucleation, we found a well-fitting prosthesis with good superior and inferior fornices and no migration of the implant. Palpation of the superior orbital rim medially, in the region of the supraorbital notch, produced intense pain which radiated superiorly above his eyebrow. Radiographs of the orbits, including tomography and axial and coronal computed tomography, disclosed no abnormalities. A- and B-scan ultrasonography also showed no abnormalities.

The patient chose to undergo exploratory surgery in an attempt to define and alleviate his pain. We performed an anterosuperior orbitotomy. We incised the frontal periosteum both medially and laterally to the supratrochlear notch. The supratrochlear and supraorbital nerves were isolated and retracted with silk sutures. The angular, dorsal nasal, and supraorbital veins were also isolated and retracted. We then lifted the periosteum both laterally and medially to the trochlear notch, keeping the anatomical relationships intact. We freed the arcus marginalis from the superior orbital rim, permitting a smooth subperiorbital dissection. Palpation disclosed a firm nodular lesion involving the supratrochlear nerve 1 cm posterior to the orbital rim and 1 cm lateral to the trochlea (Fig. 1). This was excised and kept for examination. We then performed a layered closure.

The patient's pain ceased immediately after the operation. Hypesthesia continued in the distribution of the supratrochlear nerve for two months postoperatively. This gradually improved, and the patient has continued to do well and has resumed wearing his ocular prosthesis.

Examination of the pathological specimen showed a traumatic neuroma (Fig. 2) with cartilaginous metaplasia (Figs. 3 and 4).

DISCUSSION

Severe, persistent socket pain unrelated to the ocular prosthesis is rare after enucleation of the globe[1,2] and should alert the clinician to a possible progressive process. In our case, the prosthesis could not have caused the pain because its removal produced no significant reduction in the pain's intensity. An unusual feature of our patient's pain was that it was best defined after a retrobulbar injection of 100% alcohol. This injection apparently blocked the component of the pain caused by the primary glaucoma, and unmasked the pain secondary to the neuroma. The original shrapnel injury, the retrobulbar injection of alcohol, or the enucleation may have caused the

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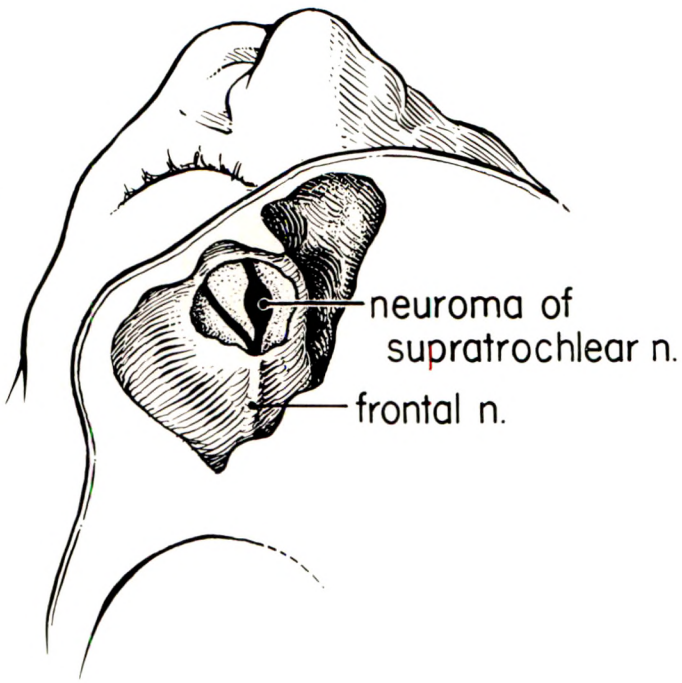


Fig. 1 (Sutula and Weiter). Schematic view of left orbit shows the location of the neuroma.

formation of the neuroma and its associated cartilaginous metaplasia. Although the exact cause of the neuroma remains unclear, it seems to have been present before the retrobulbar injection of alcohol.

Amputation neuroma is actually an abortive regeneration of a severed nerve rather than a true neoplasm. The connective tissue components—the endoneurium, perineurium, and Schwann cells—proliferate. Axon cylinders elongate, branch, and ramify to follow these pathfinders from the proximal end towards the distal portion of the nerve.[3-5] If enough soft tissue intervenes, however, the normal guidelines are obliterated and the axons become entangled in a densely fibrous matrix. In the present case, the cartilaginous metaplasia may have blocked the regenerating neurons. The proliferative response in the proximal end may persist for years, producing this clubbed overgrowth. The interval from severance of the nerve to the development of the neuroma can range from 16 months to 50 years.[4]

Although amputation, or traumatic, neuroma is common in many parts of the body, reported cases in the orbit are rare. Henderson[5] did not come across such a tumor in his orbital survey. Duke-Elder and MacFaul[4] suggested that it rarely occurs in the orbit because the orbital nerves are small and there are usually no irritating factors. Blodi[6] reported an amputation neuroma in the orbit after enucleation for retinoblastoma in 1949. The neuroma was found in a specimen exenterated after recurrence of the retinoblastoma. The present case is unusual in that the neuroma formed in a nerve not known to have suffered trauma or amputation.

Orbital cartilage is normally found at the trochlea and occasionally at the caruncle.[7,8] Orbital chondromas are extremely rare. Dahlin,[9] in a review of 3,987 osseous and cartilaginous tumors, did not find any orbital chondromas. Henderson[10]

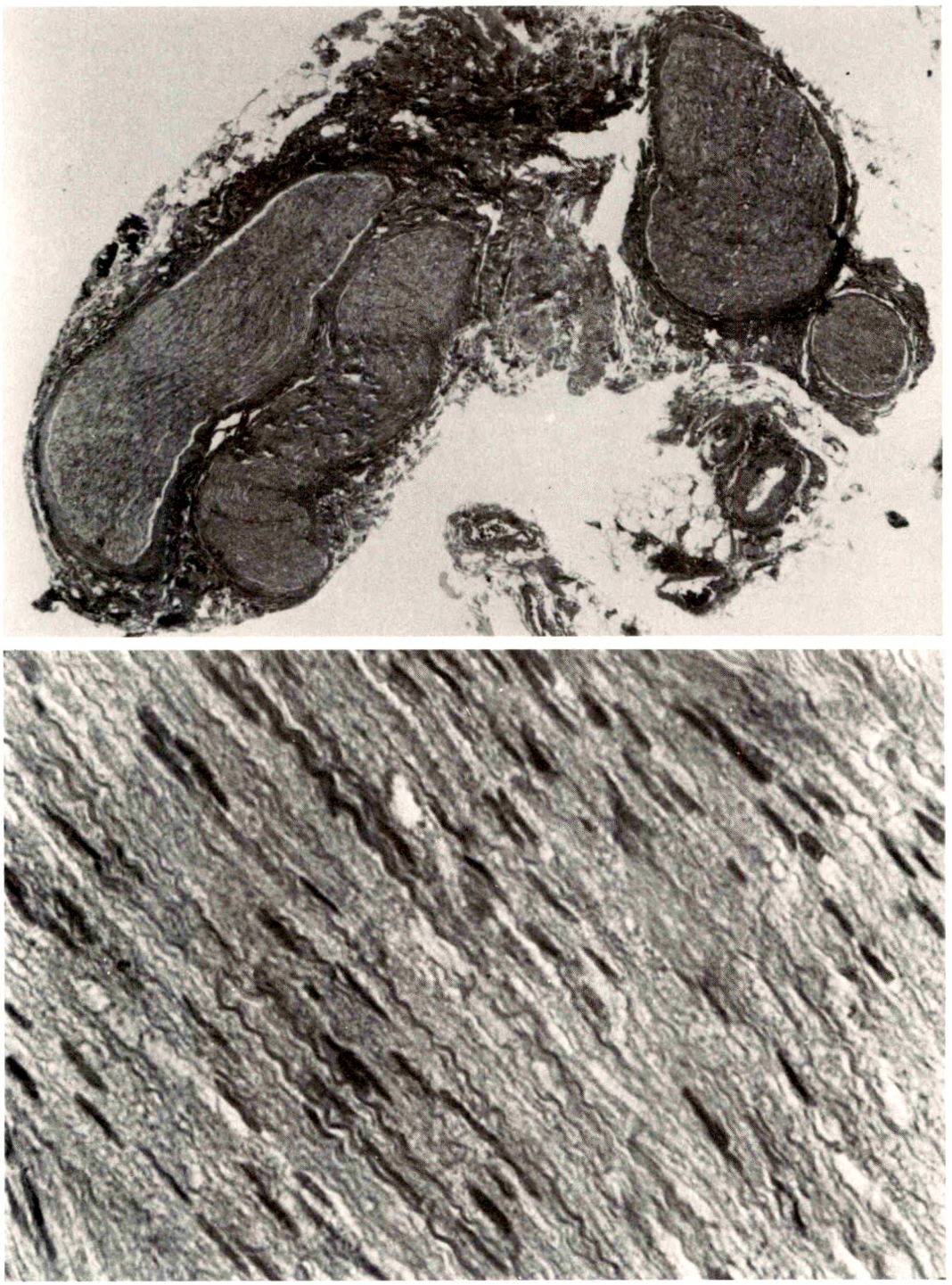


Fig. 2 (Sutula and Weiter). Histologic section of neuroma. Top, Low-power view shows axon cylinders encased in connective tissue (hematoxylin and eosin, × 25). Bottom, Higher-power view of axon cylinders (hematoxylin and eosin, × 200).

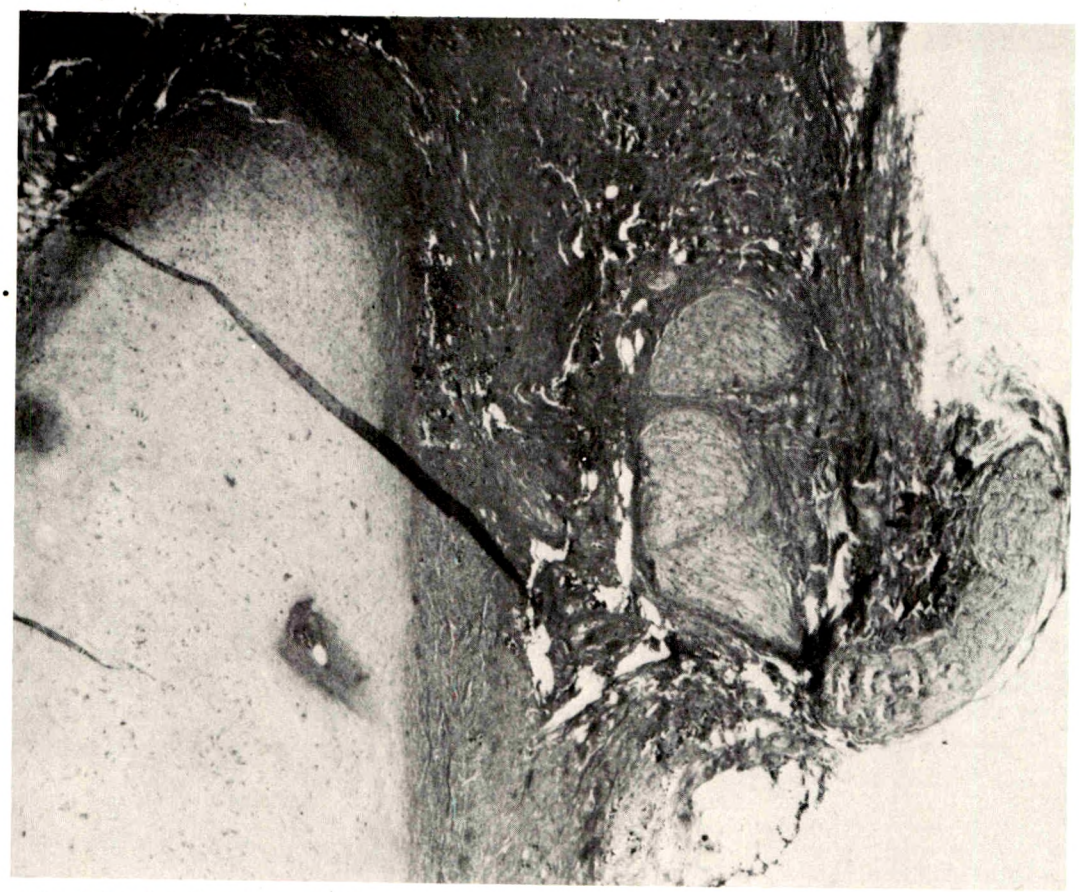


Fig. 3 (Sutula and Weiter). Histologic section shows neuroma in approximation to cartilaginous metaplasia (hematoxylin and eosin, × 25).

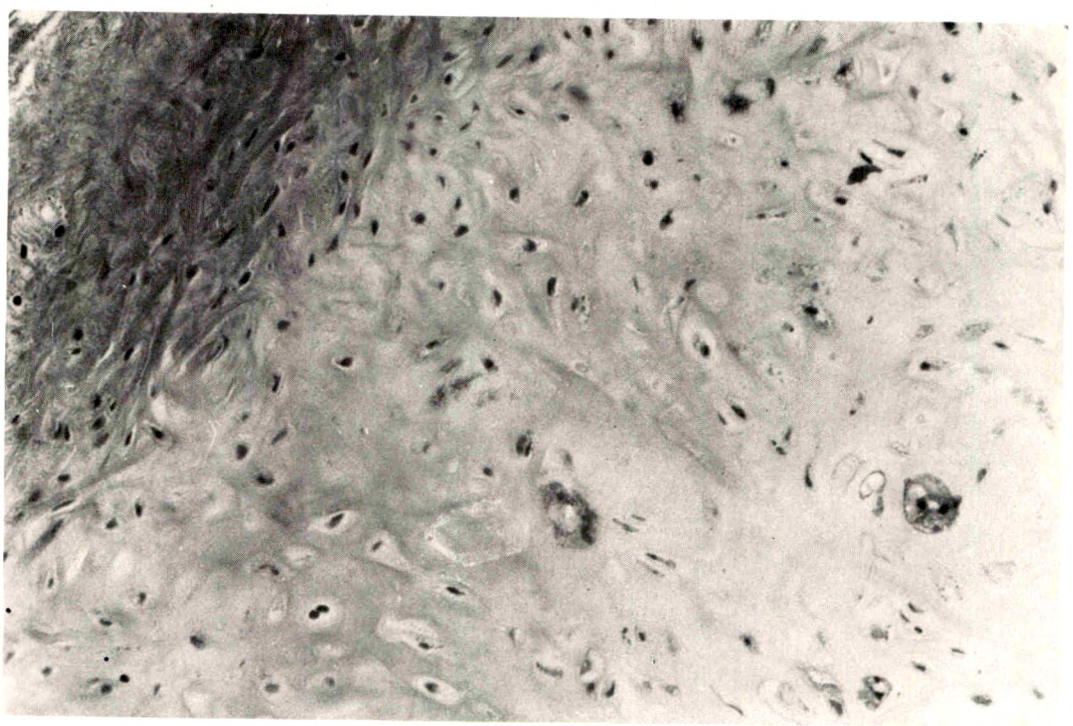


Fig. 4 (Sutula and Weiter). Histologic section of cartilaginous metaplasia showing disorderly cellular grouping (hematoxylin and eosin, × 200).

noted that these tumors are extremely rare in the orbit and that most cartilaginous growth occurring around the rim of the orbit is likely to be cartilaginous metaplasia incorporated into the fibromas. A neuroma contains fibrous connective tissue in addition to Schwann cells, perineurium, and endoneurium. We know that this tissue is capable of differentiating into many mesenchymal elements, including cartilage, under irritating influences.

This case is unique for several reasons. Both neuroma and cartilaginous metaplasia are rare in the orbit; an orbital manifestation with both tissue types has not been reported to our knowledge. Additionally, the neuroma arose from a nerve that was not traumatized by enucleation. The supratrochlear nerve, not the nasociliary nerve, was involved.

Summary

Total retinal detachment and neovascular glaucoma occurred after a penetrating injury to the left globe that left the eye blind and painful. A retrobulbar injection of alcohol and enucleation modified but did not relieve the orbital discomfort. At surgery, we discovered a neuroma with cartilaginous metaplasia involving the supratrochlear nerve. Excision of this mass produced relief of symptoms. This is, to our knowledge, the first reported case of a neuroma with cartilaginous metaplasia involving the supratrochlear nerve.

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INTRAOCULAR INVASION BY PAPILLARY SQUAMOUS CELL CARCINOMA OF THE CONJUNCTIVA

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Squamous cell carcinoma of the conjunctiva is usually a locally invasive tumor, and intraocular extension is rare.[1-3] In previously reported cases,[4-6] intraocular involvement generally led to enucleation of the globe. Experience with resection of certain iris and ciliary body tumors using the available microsurgical techniques has shown that local en bloc excision of the cornea, sclera, iris, and ciliary body are compatible with the preservation of good visual acuity.[7,8] We describe two patients with incompletely excised invasive papillary squamous cell carcinoma of the conjunctiva. We attempted local resections in these patients. Despite apparently adequate clearance of the tumor margins, as seen during surgery, the surgical resection margins were not cleared histopathologically, and we had to enucleate the eyes.

SUBJECTS AND METHODS

Two patients came here with biopsy-proven invasive papillary squamous cell carcinoma of the conjunctiva that had been incompletely excised. After a complete ocular examination, each patient underwent a second biopsy of the previously excised limbal area to confirm the presence of residual tumor before more extensive resection procedures.

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CASE REPORTS

Case 1—A 33-year-old woman had had a "growth" removed from the temporal corneoscleral limbus of her left eye in 1963. In November 1974, an ocular examination disclosed a red lesion on her left temporal corneoscleral limbus, which had been enlarging for two months. Follow-up examination in July 1975 showed that the lesion had encroached 0.5 mm onto the cornea. She underwent an excisional biopsy followed by radiation therapy. Histopathologic examination showed a papillary squamous cell carcinoma of the conjunctiva.

In December 1975, the patient came here because of redness and increased vascularization at the biopsy site. Visual acuity was 6/12 (20/40) in the left eye. Slit-lamp examination showed diffuse hyperemia and a raised, whitish vascularized lesion located at the temporal corneoscleral limbus. A fan-like midstromal corneal opacification with thickening and infiltrate extended from the corneoscleral limbus almost to the visual axis (Fig. 1). There were 2+ cells and flare in the anterior chamber. Gonioscopy showed that the inner cornea was displaced inwardly into the anterior chamber, but we saw no peripheral anterior synechiae. A superficial biopsy of the conjunctival lesion showed papillary squamous cell carcinoma. In an effort to retain the eye, we performed an en bloc

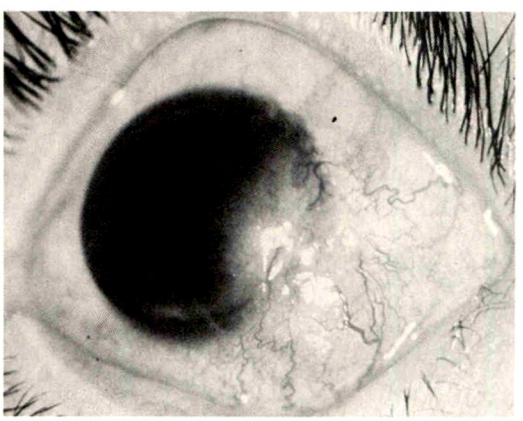


Fig. 1 (Li, Pettit, and Zakka). Case 1. Left eye shows recurrence of corneoscleral limbal lesion after excision and irradiation therapy. Note prominent vascularity and tumor infiltrate of the deep corneal stroma (December 1975).

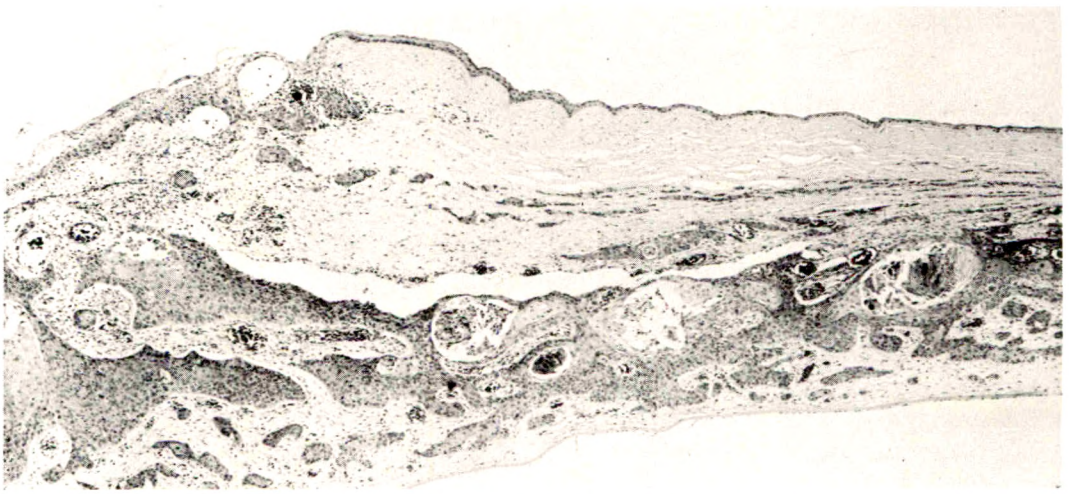


Fig. 2 (Li, Pettit, and Zakka). Case 1. Section of cornea shows extensive tumor invasion of the deep stromal lamellae and an intact Descemet's membrane (hematoxylin and eosin, ×100).

corneoscleral resection of the lesion and a patch graft. The corneoscleral button straddled the corneoscleral limbus and included more than 50% of the cornea.

Histopathologic examination of the corneoscleral button showed papillary squamous cell carcinoma infiltrating the corneal stromal lamellae (Fig. 2) and the trabecular meshwork (Fig. 3). Descemet's membrane remained intact. We enucleated the left eye because of probable intraocular invasion secondary to the trabecular meshwork involvement.

Histopathologic examination of the enucleated eye showed papillary squamous cell carcinoma with tumor cells in the donor trabecular meshwork, adjacent supraciliary space, iris, and ciliary body temporally (Fig. 4). Tumor cells were present in the iris stroma at the surgical iridectomy sites. There were no tumor cells in the nasal anterior chamber angle and trabecular meshwork. Special stains for mucin were negative in all sections.

Case 2—A 69-year-old woman had noted intermittent irritation of her right eye in 1975. Ocular examination disclosed a small white plaque at the apex of a pinguecula located at the nasal corneoscleral limbus. The plaque was easily "wiped off" with a sterile cotton applicator, but recurred on several occasions. In October 1977, an excisional biopsy of this recurrent lesion showed a pinguecula. The lesion again recurred, increased in size, and, in January 1978, was excised with a 2-mm conjunctival border. Histopathologic examination showed papillary squamous cell carcinoma of the conjunctiva, well differentiated with focal invasion of the deep margins.

The patient was referred here in June 1978 with persistent irritation of the right eye. Visual acuity was 6/6 (20/20). The eye had a mild circumciliary flush and the nasal corneoscleral limbus showed an elevated vascularized hypertrophic lesion. The underlying sclera was irregular with focal areas showing clusters of fine vascular tufts (Fig. 5). The anterior chamber contained 1+ flare and an occasional cell. Gonioscopy showed a band of peripheral anterior synechiae extending from the 2:30- to 4-o'clock meridian. Biopsy of the lesion confirmed the presence of focally invasive papillary squamous cell carcinoma of the conjunctiva. We decided to try to salvage the eye with an en bloc resection of the involved conjunctiva, sclera, cornea, iris, and ciliary body. In August 1978, we performed a resection extending from the 2- to 5-o'clock meridian. We repaired the defect with a freehand corneoscleral graft.

Histopathologic examination showed papillary squamous cell carcinoma extending into the full thickness of the sclera and into the iris and ciliary body. Peripheral anterior synechiae were present, and tumor cells extended posteriorly into the adjacent supraciliary space, ciliary body, and trabecular meshwork. The surgical margins at the 2- and 5-o'clock positions were involved with tumor cells. We enucleated the eye one week later.

Histopathologic examination of the enucleated eye showed papillary squamous cell carcinoma, well differentiated, extending from the ciliary body into the area of resection. Tumor cells were visible in the posterior aspect of the ciliary body (Fig. 6). There was no tumor seeding into the temporal anterior chamber angle. Special stains for mucin were negative in all sections.

Discussion

Most cases of squamous cell carcinoma of the conjunctiva have a relatively benign clinical course. The term carcinoma in situ, or intraepithelial epithelioma, denotes an intact basement membrane and the presence of tumor cells within

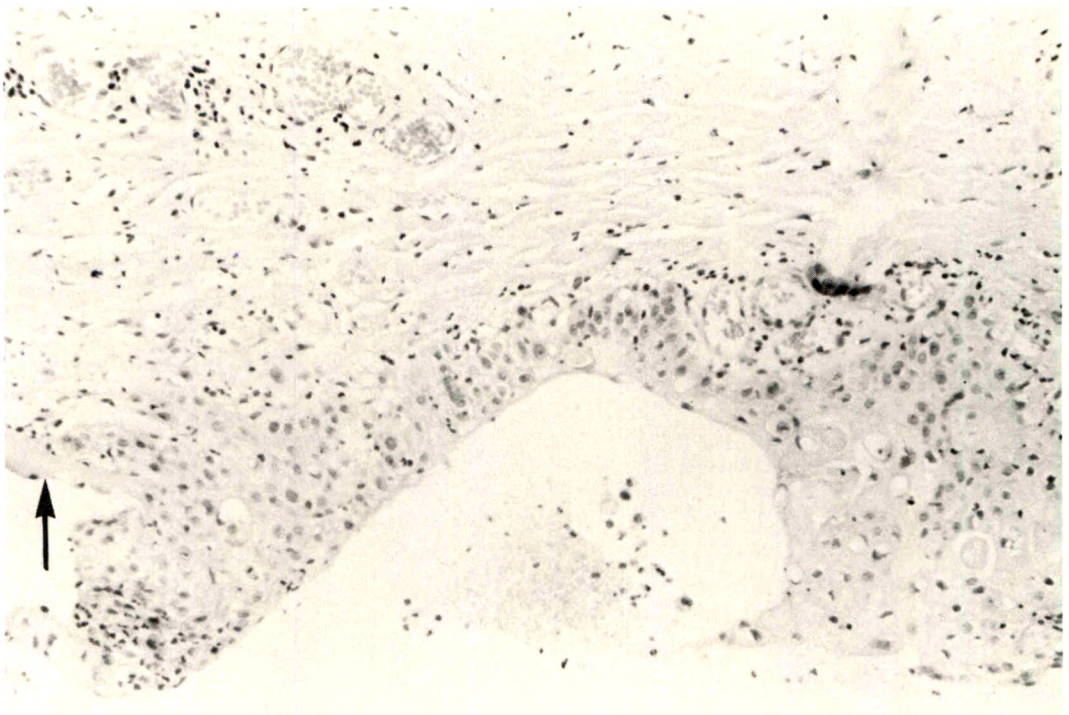


Fig. 3 (Li, Pettit, and Zakka). Case 1. Section of cornea shows conjunctival papillary squamous cell carcinoma infiltrating the trabecular meshwork. Note surrounding chronic inflammation and edge of Descemet's membrane (arrow) (hematoxylin and eosin, ×100).

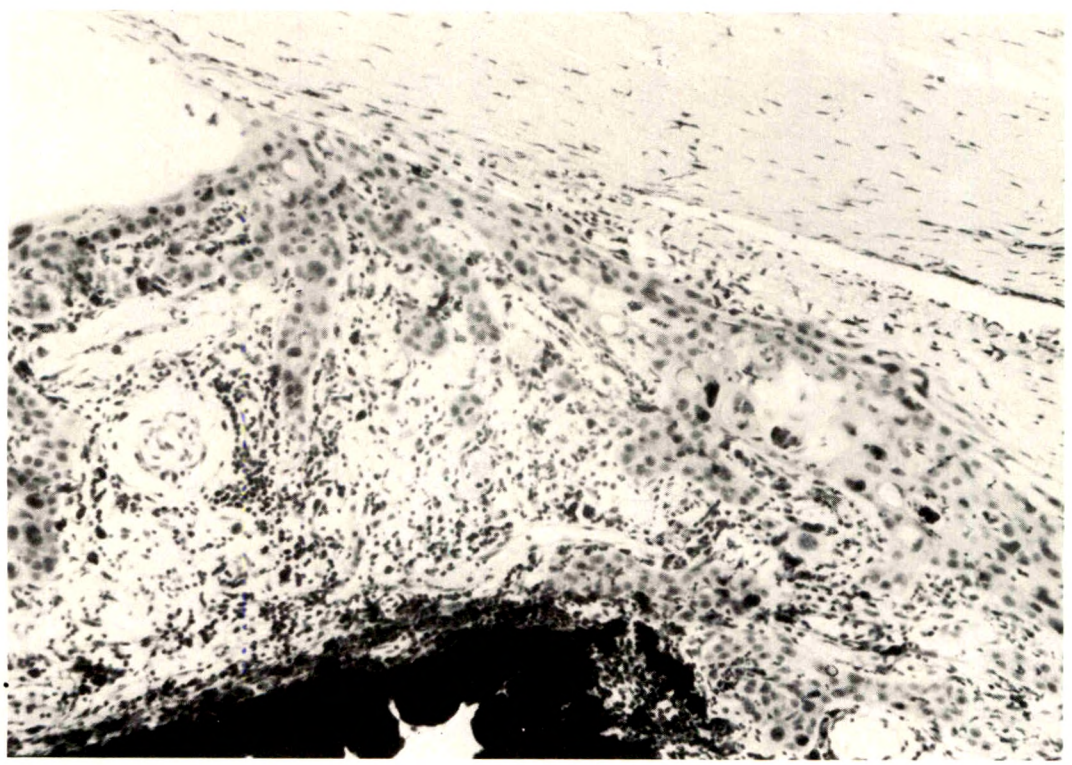


Fig. 4 (Li, Pettit, and Zakka). Case 1. Section of enucleated left eye through the area of the corneoscleral graft shows extensive infiltration of the iris and ciliary body with conjunctival papillary squamous cell carcinoma cells. The donor trabecular meshwork overlays tumor cells (hematoxylin and eosin, ×100).

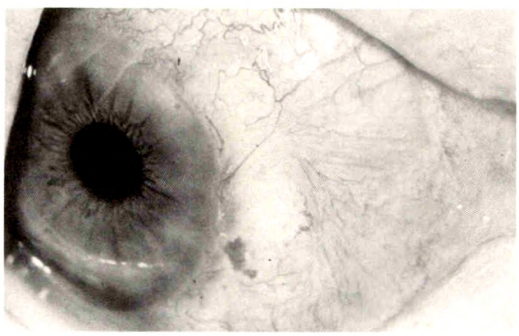


Fig. 5 (Li, Pettit, and Zakka). Case 2. Right eye shows mild circumciliary flush with an elevated vascular lesion located at the nasal corneoscleral limbus.

the epithelium. Of these conjunctival squamous cell carcinomas, 10% to 35% recur. Local reexcision with or without irradiation is usually adequate treatment.[3,5]

Invasive squamous cell carcinoma of the conjunctiva is the extension of tumor cells through the basement membrane. If excision is inadequate or irradiation is unsuccessful, these tumors may extend along ocular surfaces, involve adjacent adnexa, and, on occasion, invade the eye.[4-6]

In previously reported cases of squamous cell carcinoma of the conjunctiva with intraocular invasion, enucleation was performed.[4-6] Pathologic studies have demonstrated that the usual route of invasion is through the corneoscleral limbal region by way of numerous drainage channels located in the area.

Nicholson and Herschler[4] reported that cytological examination of aqueous humor for intraocular tumor cells is useful where intraocular invasion is suspected. A positive anterior chamber paracentesis indicates intraocular extension of tumor and implies that the eye cannot be salvaged by an additional local resection procedure and that an enucleation is indicated.

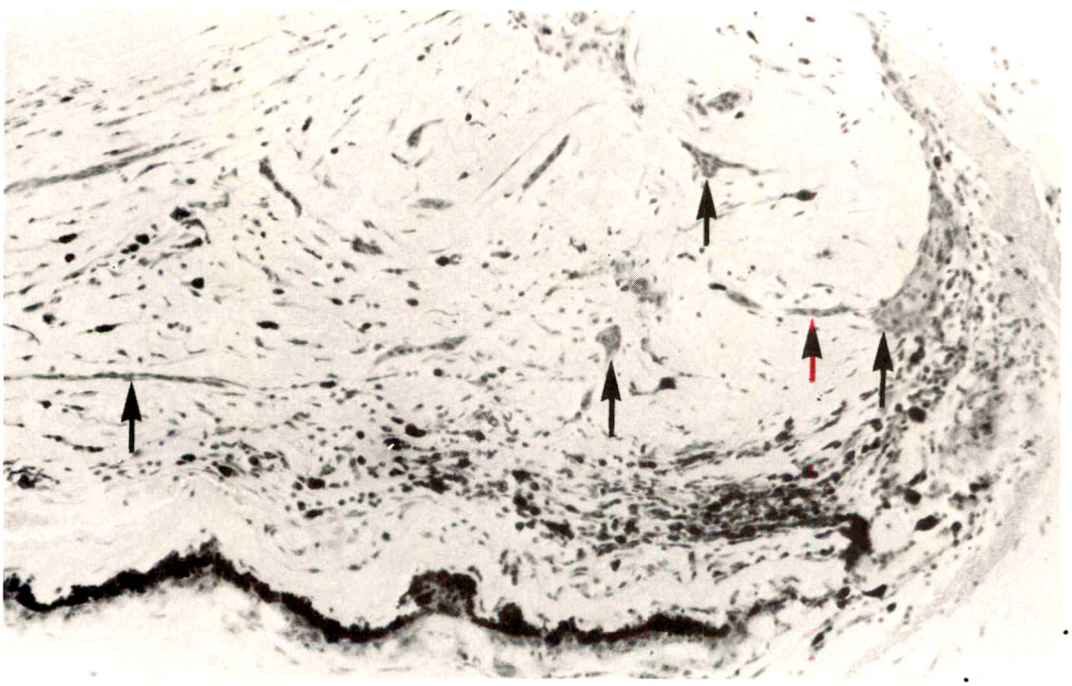


Fig. 6 (Li, Pettit, and Zakka). Case 2. Section of enucleated right eye shows nests and strands of conjunctival papillary squamous cell carcinoma cells infiltrating the edge of the corneoscleral graft and remnants of the ciliary body (arrows) (hematoxylin and eosin, ×150).

In our two patients, only minimal clinical signs were present to suggest intraocular involvement. Clinical examination of both patients showed mild anterior chamber reaction, which was thought to be inflammation rather than tumor invasion; in Case 1 the cornea showed localized midstromal opacification, and in Case 2 subtle peripheral anterior synechiae were present.

We attempted en bloc surgical resections in these two cases because the clinical signs were minimal and we suspected incomplete transmural extension or only localized intraocular involvement. We judged the amount of resection as adequate to eradicate the tumor as evaluated at surgery with the operating microscope. Histopathologic examination of the excised specimens showed tumor cells in the surgical margins, indicating more extensive involvement than anticipated. In our cases and in previous reports of papillary squamous cell carcinoma of the conjunctiva with intraocular extension, the original conjunctival lesions were incompletely excised and showed tumor involvement of the deep margins.[4-6] Our cases illustrate the potential aggressiveness of the tumor and suggest that when excision of a surface lesion is incomplete, prompt surgical reexcision may permit complete eradication of the tumor and may prevent intraocular extension.

We believe that in those cases where the tumor is localized, reexcision of the lesion should be attempted with the help of frozen sections to determine that the surgical margins are clear. In our limited experience, histopathologic examination of frozen sections has been helpful in one case of squamous cell carcinoma of the corneoscleral limbus where the initial surgery failed to clear the surgical margins. When many reexcisions of the conjunctival tumor show the deep surgical margins persistently involved with tumor cells on frozen sections, a wide en bloc resection of the conjunctival mass, corneosclera, iris, and ciliary body may be attempted. The value of this procedure is yet to be proved. In our two cases, we were unsuccessful in clearing the margins of the tumor, and enucleations were required. In view of the lethal potential of this tumor, the accepted treatment is enucleation whenever there is intraocular invasion.[4-6]

Summary

Two patients with incompletely excised invasive papillary squamous cell carcinoma of the corneoscleral limbal conjunctiva had subtle clinical signs suggesting incomplete transmural extension or localized intraocular tumor invasion. We attempted a wide en bloc resection procedure in each case. Histopathologic examination disclosed that the surgical margins were involved with tumor cells and the eyes were subsequently enucleated. The enucleated eyes disclosed more extensive microscopic intraocular involvement than had been anticipated.

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OCULAR METASTASES FROM SYSTEMIC MELANOMA

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Melanoma metastatic to the eye has been reported in fewer than 40 cases.[1-5] Most ocular metastases are symptomatic, with distinct tumors visible on ophthalmoscopic examination; some patients with melanoma, however, have ocular metastases that are small, diffuse infiltrates, difficult to diagnose.[5] We recently examined two patients with ocular metastases from widespread melanoma. Neither patient had distinct ocular tumors; one patient had numerous melanoma cells in the vitreous and the second patient had heterochromia with a brownish-colored hypopyon. The diagnosis of metastatic melanoma to the eye was confirmed in each case by cytologic examination of the appropriate ocular fluid. Metastatic melanoma of the eye can show a number of unusual features, and cytology can be a useful diagnostic adjunct.

CASE REPORTS

Case 1—This 72-year-old man was examined because of a two-month history of vitreous floaters in the right eye. Seven years before admission the patient had had a melanoma removed from the lateral aspect of his left arm. Results of an axillary node dissection at that time were negative; however, two years later the patient developed a subcutaneous nodule in the axilla that was histologically diagnosed as a recurrent melanoma. This axillary mass recurred and was excised a second time. Systemic examinations at that time gave otherwise normal results. The patient was initially treated with transfer factor; however, when visceral disease developed he received radiation and chemotherapy. The patient had an excellent response to this therapeutic regimen; at the time of his ocular symptoms, the visceral disease had regressed.

On ocular examination visual acuity was R.E.: 6/9 (20/30) and L.E.: 6/6 (20/20). Anterior segment examinations gave normal results bilaterally. The left eye was normal. Examination of the right vitreous disclosed clumps of brownish cells and strands (Fig. 1). There were no discrete tumors in the fundus, ciliary body, or iris of the right eye. There was depigmentation of the retinal pigment epithelium with some clumped brownish pigment, both superficially and beneath the retinal vessels (Fig. 2). We performed an uneventful pars plana vitreous aspiration. Cytologic examination of the vitreous showed many malignant cells on the Millipore filter preparation stained with a modification of the Papanicolaou method (Fig. 3).[6] These melanoma cells had nuclear enlargement, hyperchromatism, and increased nucleocytoplasmic ratios when compared with adjacent benign histiocytic cells. The pigmentation of these melanoma cells varied; most had fine, uniformly distributed dark-brown pigmentation. In contrast,

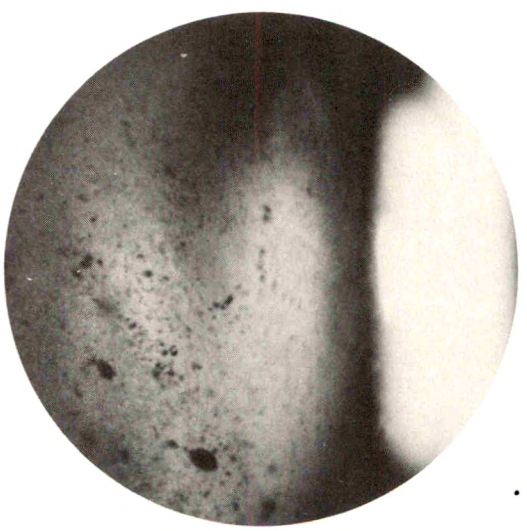


Fig. 1 (Char and associates). Case 1. Vitreous cavity is filled with brownish cells and strands.

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Fig. 2 (Char and associates). Case 1. General disturbance of the retinal pigment epithelium with clumped pigment superficially and beneath retinal vessels.

the pigmentation in benign macrophages was irregular in size and distribution.

Nuclear membranes of the melanoma cells were irregular with fine, shallow invaginations. There were no intranuclear cytoplasmic inclusions.

Case 2—This 27-year-old woman with metastatic melanoma, primarily involving cutaneous tissues, was referred because of headaches and decreased visual acuity in her left eye. The patient had had a melanoma resected from her right shoulder and a secondary axillary node dissection for recurrent disease. She had been treated for diffuse cutaneous metastases with transfer factor and BCG vaccine 1½ years before the ocular examination.

A brain scan done with computed axial tomography and cytologic examination of cerebrospinal fluid disclosed no unusual findings. Visual acuity was R.E.: 6/6 (20/20) and L.E.: 6/22 (20/70).

Clinically detectable metastatic disease was confined to the anterior segment of the left eye. The patient had prominent episcleral vessels, heterochromia with diffusely increased pigmentation of the left iris, a brown hypopyon with brownish keratitic precipitates, pigment on the anterior lens capsule, and increased pigmentation of the anterior chamber angle (Fig. 4 and 5). The intraocular pressure in the involved left eye was 35 mm Hg; the pressure in the right eye was 17 mm Hg. Results of an ultrasonographic evaluation of the left eye were unremarkable.

We performed an uneventful anterior chamber paracentesis in the left eye. A 0.25-ml sample of anterior chamber fluid was processed by the Millipore filtration method and stained with a modified Papanicolaou stain.[6] Malignant cells and benign lymphocytes and histiocytes were identified in the aqueous specimen. The melanoma cells had a moderate amount of cytoplasm and one or two nuclei. These nuclei were approximately twice as large as those in the nonmalignant lymphocytes. We found no nuclear inclusions in the melanoma cells; the nuclear outline varied from smooth to irregular. The cytoplasmic borders of the tumor cells were sharp, and occasional melanoma cells contained finely granular brown pigment consistent with melanin (Fig. 6).

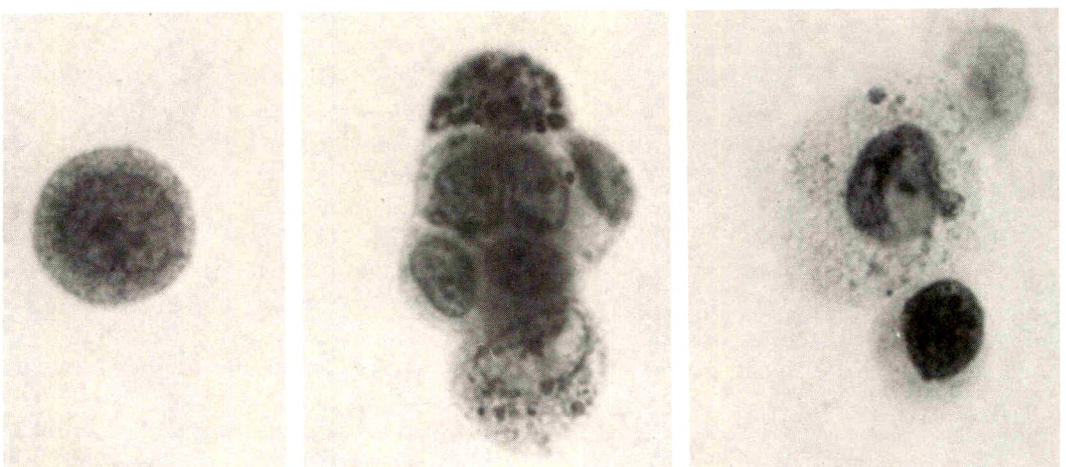


Fig. 3 (Char and associates). Case 1. Metastatic melanoma in the vitreous. Left, Typical malignant melanocyte with prominent nucleolus, irregular nuclear membrane, and small fine pigment granules evenly spaced throughout the cytoplasm. Middle, A group of malignant melanocytes with unusually large uniform coarse granules. Right, Benign macrophage (at top) with a bland nucleus, abundant cytoplasm, and variable size pigment granules. These features of the macrophage are in marked contrast to those of the malignant melanocyte (at bottom).

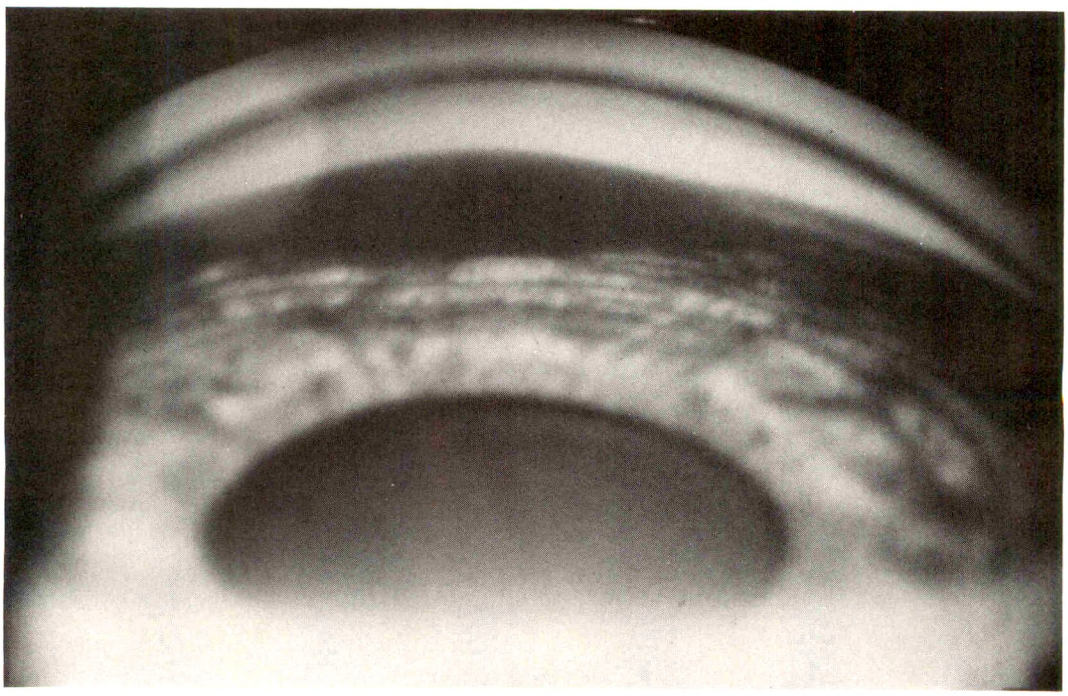


Fig. 4 (Char and associates). Case 2. Brown hypopyon with heterochromia.

Discussion

Patients with melanoma metastatic to the eye and adnexa usually have discrete tumors of the iris, ciliary body, choroid, retina, optic nerve, or orbit.[7-12] The unusual findings in our two cases were the absence of discrete tumors and the obvious involvement of only the anterior chamber in Case 1 and the vitreous in Case 2.

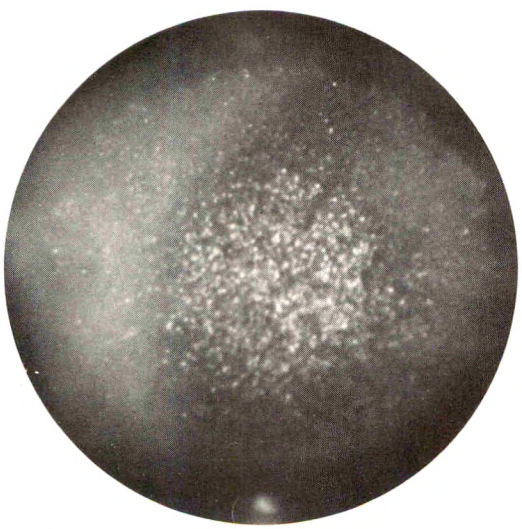


Fig. 5 (Char and associates). Case 2. Pigment on the corneal endothelium and anterior lens capsule.

Fishman, Tomaszewski, and Kuwabara[5] described 15 patients without ocular symptoms who died of widespread systemic melanoma. Five of these patients had small tumor foci in the choroid or retina. These authors concluded that small, asymptomatic ocular metastases were more common than previously reported. In our two patients, tumor cells were found in the ocular fluids without an obvious focal mass. We have occasionally observed a similar finding in ocular reticulum cell sarcoma (unpublished data).

Although there have been occasional reports of metastatic melanoma producing a brown or black hypopyon, we are unaware of a previous case of isolated metastases to the vitreous.[13] Newer

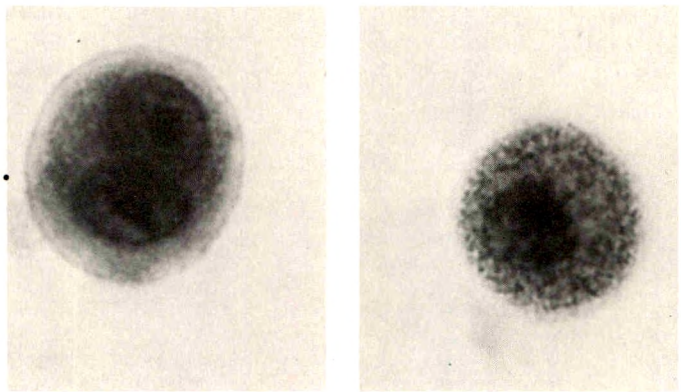


Fig. 6 (Char and associates). Case 2. Metastatic malignant melanoma in anterior chamber fluid. Left, Malignant melanoma cell is binucleated with large nucleoli and perinucleolar clearing. Right, Malignant melanoma cell has finely granular intracytoplasmic granules consistent with melanin.

chemotherapeutic approaches in the management of widespread melanoma may have altered the metastatic pattern of this disease, causing some unusual clinical presentations, such as those we observed.

Cytologic techniques have been used to diagnose ocular metastatic melanoma.[14] Although the cytologic features of systemic malignant melanoma are well known, only a few patients with ocular metastases have been studied.[14-18] Ocular fluids from eyes with metastatic melanoma may contain distinctive neoplastic melanocytes as well as inflammatory cells; we observed these findings in our two cases. The malignant melanocytes range from 10 to 300 μ in diameter,[7] usually have abundant cytoplasm, and vary in shape; occasionally, multinucleate and bizarre giant cells are found.[17-20] Malignant melanoma cells are usually found alone, but occasionally they occur in groups.

Several nuclear features are characteristic of melanoma cells.[17,20,21] Nuclei are eccentrically placed, the chromatin pattern is lacy, and there are prominent, large, central nucleoli. The nuclear membrane is often delicate, with shallow infoldings without chromatin condensation. Intranuclear cytoplasmic inclusions suggest melanoma and are a useful finding in clinically amelanotic tumors.[17,20] Almost all of the above findings were present in the cytologic examination of our two cases.

The cytoplasmic production of pigment, as a result of conversion of tyrosine to dihydroxyphenylalamine and its subsequent combination with protein, in a melanosome is the hallmark of a melanoma cell.[19] Melanic pigment must be differentiated from the other brownish pigments, hemosiderin and lipofuscin. Melanin particles are characteristically small, uniform in size, and evenly dispersed throughout the cytoplasm; these findings are not characteristic of either hemosiderin or lipofuscin.[21] Histochemical stains can also differentiate iron and lipofuscin from melanin.[22] Alternatively, the presence of melanin can be directly determined in cytologic material with a melanin bleaching process, dihydroxyphenylalamine reaction, or electron microscopy.[23-25]

A number of other metabolic, inflammatory, and neoplastic ocular lesions can show cells in either the vitreous or the aqueous fluids. Fine-needle biopsy for systemic tumors is a safe procedure with almost no risk of tumor dissemination.[26]

The usefulness and safety of intraocular fine-needle biopsy are unproven. Except in rare instances, we do not believe there are sufficient data on the specificity and sensitivity of this technique to advocate

its routine clinical use in the evaluation of patients with probable primary ocular tumors. In our two patients with documented systemic metastases and probable metastatic melanoma involving the ocular fluids, we did not consider the risk of fine-needle aspiration of the ocular fluids for cytologic examination significant.

Both of our patients with ocular metastases refused therapy. The treatment of melanoma metastatic to the eye is not well delineated. Some patients with ocular metastases have had a good response with chemotherapy; other patients receiving chemotherapy have experienced either the development or progression of ocular metastases during treatment.[27] In patients with metastatic melanoma and ocular involvement, cytologic diagnostic methods may be useful. In those patients not receiving treatment for metastatic melanoma, chemotherapy may eradicate the metastases. If the ocular metastases predominantly involve the posterior segment of the eye, irradiation may also be useful. The response rate of metastatic melanoma with conventional forms of external beam irradiation is approximately 30% to 50%.[28] As with other types of metastatic melanoma, the prognosis with ocular metastases is poor; fewer than 10% of patients survive more than eight months.

Summary

We studied two patients, a 72-year-old man and a 27-year-old woman, with metastatic melanoma documented by cytologic examination of the ocular fluids. Neither patient had a clinically detectable ocular mass. Fine-needle aspiration with cytologic examination may be useful in the diagnosis of metastatic ocular melanoma.

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CONJUNCTIVAL ULTRASTRUCTURE IN NIEMANN-PICK DISEASE TYPE C

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Crocker[1] described four phenotypes of Niemann-Pick disease (types A to D) in 1961. A presumed fifth (type E) and a presumed sixth (type F) phenotypes were described later.[2,3] The different types are actually separate diseases, differing in their clinical manifestations, age at onset, severity of the neurological involvement, and genetic background. All have in common a deficiency of sphingomyelinase enzyme or isoenzyme.

Types A (classical infantile form) and C (late infantile or juvenile form) are of interest to the ophthalmologist. Ocular changes have been described in these two types. The earliest, most common, and most obvious ocular manifestation is the cherry-red spot found in about one half of all cases of Niemann-Pick disease.[4] Corneal and lens involvement has also been described in type A.[5]

The classical infantile form (type A) is the most common and the most severe. Libert and Danis[6] studied the conjunctiva in this type of Niemann-Pick disease by light and electron microscopy and found characteristic lesions. These investigators recently described conjunctival biopsy findings in types A, B, and C.[7]

We studied the ultrastructure of the conjunctiva in Niemann-Pick disease type C. Our findings indicate that conjunctival biopsy may be helpful in distinguishing among the phenotypes of Niemann-Pick disease.

CASE REPORT

The patient was the product of an uneventful pregnancy and delivery. Until the age of 10 months her development was normal. However, after the age of 10 months, a slow but progressive psychomotor deterioration began. At the age of 5 years she was unable to sit or walk, and had severe mental retardation, hypotonia, and hepatosplenomegaly of 4 to 5 cm. At the age of 5½ years she died of pneumonia.

Laboratory studies—The specific activity of sphingomyelinase in fibroblast extracts was 17 nmoles/hr/mg of protein, about 24% of normal (controls, average 72 ± 7; range, 54 to 84). Bone marrow samples disclosed typical foamy histiocytes.

Family history—The parents were first cousins of Jewish Iranian origin. Both were healthy. Four other children died with a similar pattern of hepatosplenomegaly and mental deterioration. In two of them bone marrow and liver biopsies showed the foamy histiocytes typical of Niemann-Pick disease.

MATERIAL AND METHODS

We performed a conjunctival biopsy as described previously.[7] A small piece of conjunctiva was fixed in 3% glutaraldehyde in 0.1 M phosphate buffer for three to four hours and postfixed in 2% osmium tetroxide for one hour. Then the specimen was dehydrated in graded alcohols and propylene oxide, embedded in Araldite, stained with uranyl acetate and lead citrate, and examined by electron microscopy.

RESULTS

The cytoplasm of most conjunctival epithelial cells contained many inclusion bodies (Fig. 1). These were concentrated around the nucleus (Fig. 2), especially near the Golgi apparatus, which was unusually developed (Fig. 3). The inclusion bodies seemed to consist of numerous concentric, osmiophilic lamellae

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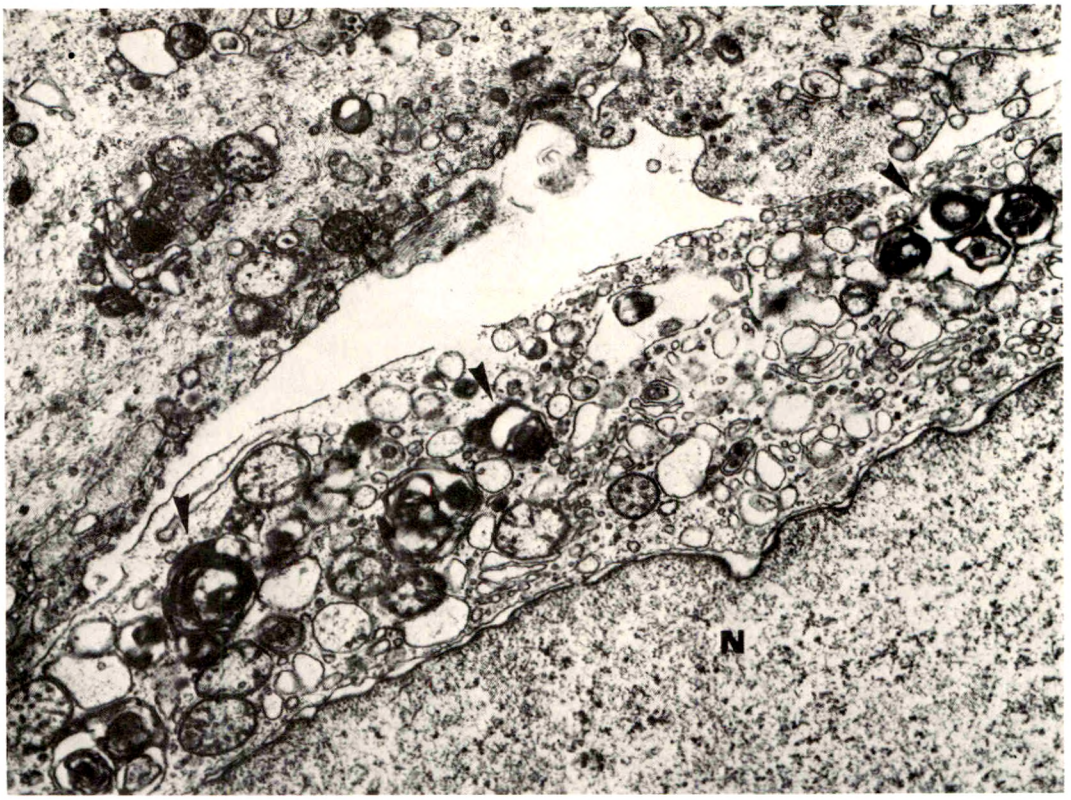


Fig. 1 (Merin, Livni, and Yatziv). There are many vacuoles containing lamellar cytoplasmic bodies (arrows) in epithelial cells of conjunctiva of patient with Niemann-Pick disease type C (N, nucleus) (× 16,200).

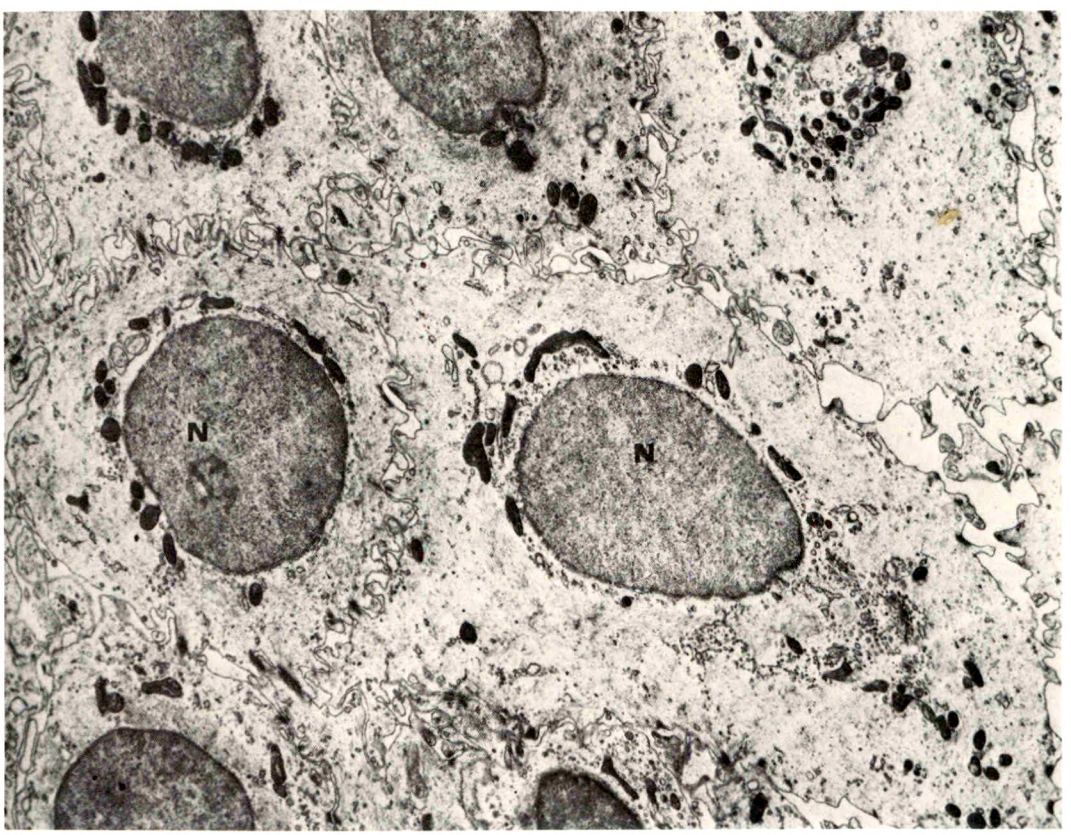


Fig. 2 (Merin, Livni, and Yatziv). Low-power view of the perinuclear arrangement of dense osmiophilic lamellar cytoplasmic bodies (N, nucleus) (× 6,100).

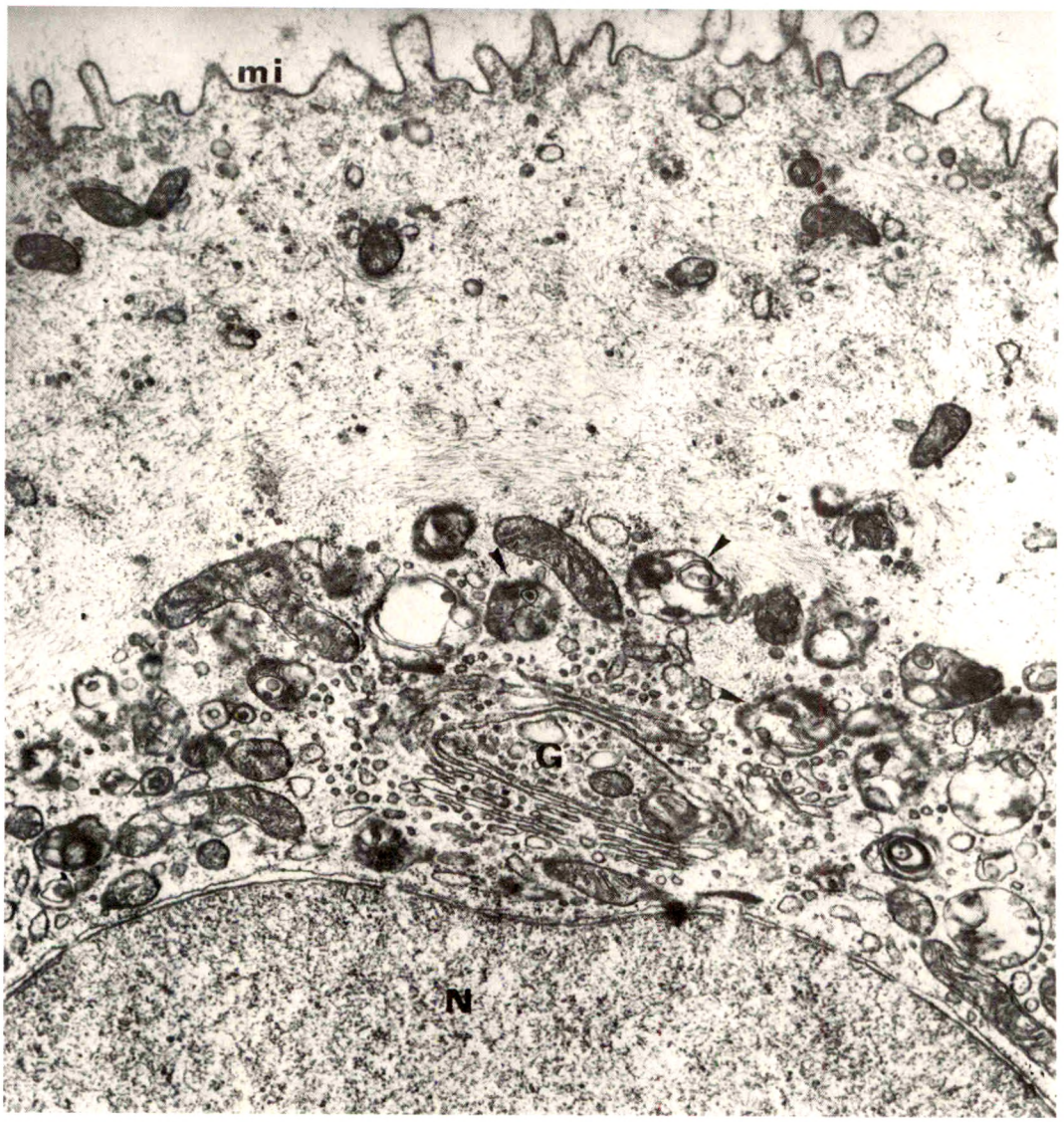


Fig. 3 (Merin, Livni, and Yatziv). A superficial epithelial cell with microvilli (mi) and many inclusion bodies (arrows) arranged around the nucleus (N) and adjacent to a well-developed Golgi apparatus (G) (× 20,000).

with large empty spaces between them (Fig. 4).

Stromal fibroblasts contained similar cytoplasmic lamellar bodies inside vacuoles, limited by a single membrane, and concentrated near the nucleus (Fig. 5).

Stromal capillaries showed similar involvement in the cytoplasm of both endothelial cells and pericytes (Fig. 6). The basement membrane of all examined capillaries was multilayered.

Discussion

Ocular abnormalities such as cherry-red spots, corneal cloudiness, and brown discoloration of the lens may be helpful in



Fig. 4 (Merin, Livni, and Yatziv). High-power view of the lamellar structure of the inclusion bodies in an epithelial cell (× 32,000).

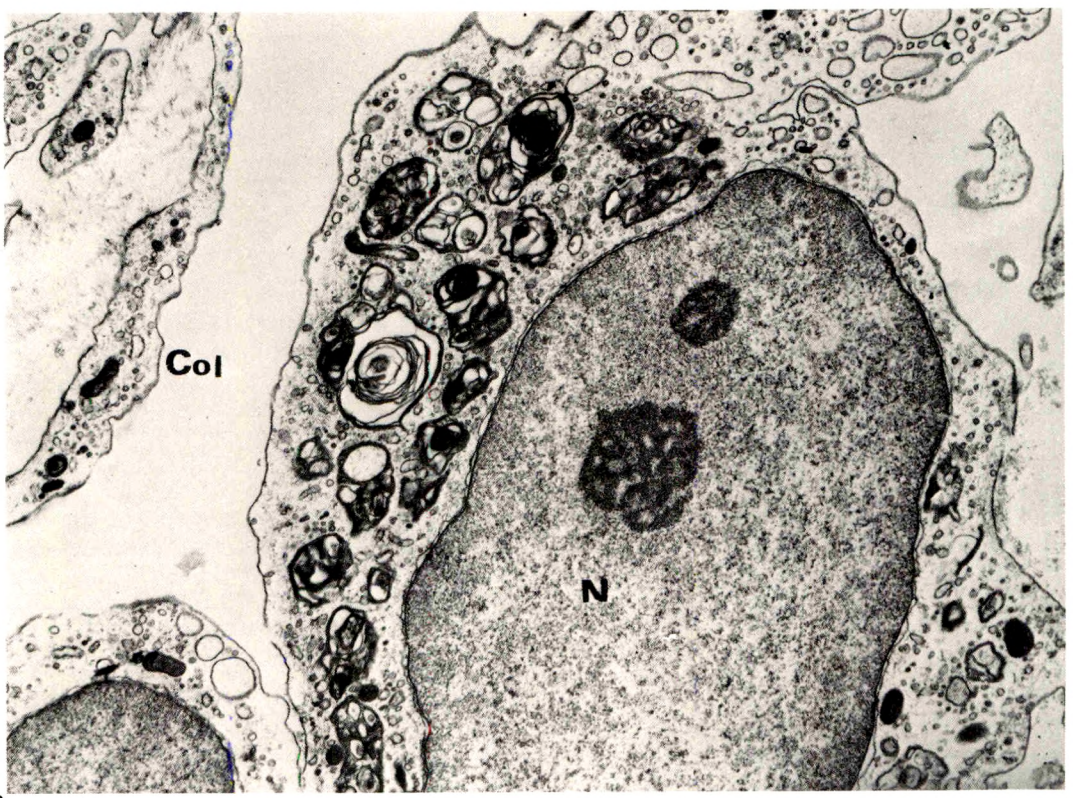


Fig. 5 (Merin, Livni, and Yatziv). Stromal fibroblast containing vacuoles with many lamellar bodies (N, nucleus; Col, collagen) (× 9,000).

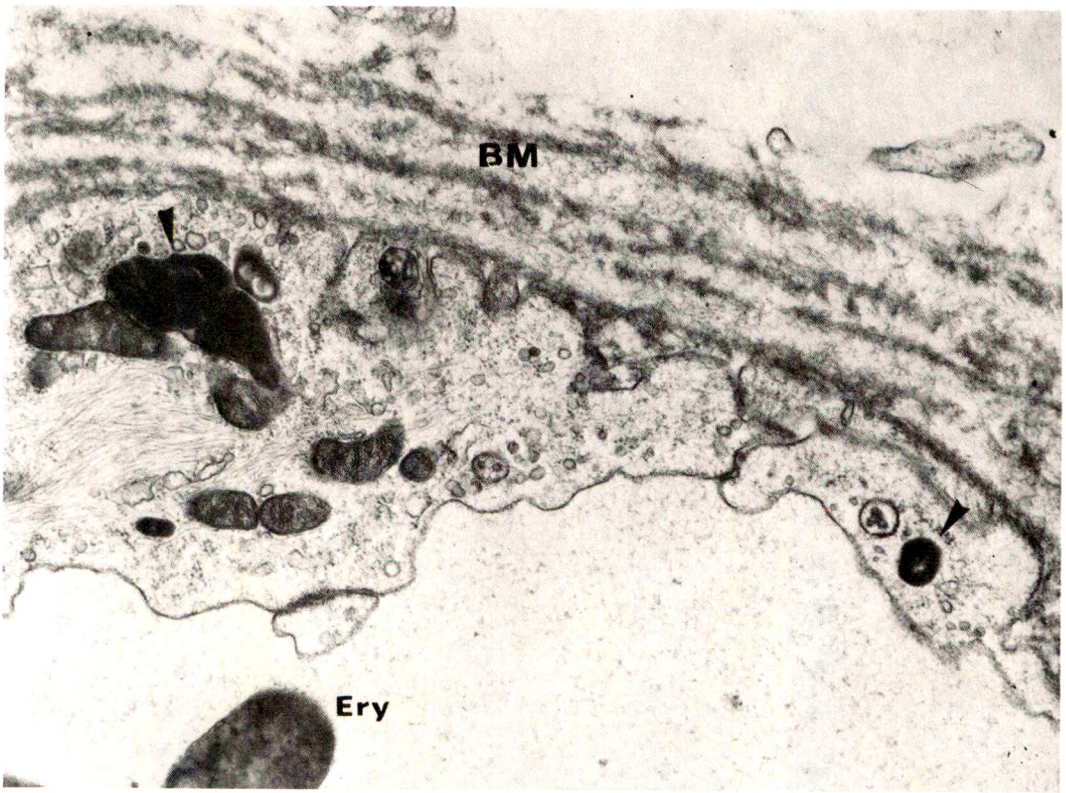


Fig. 6 (Merin, Livni, and Yatziv). Endothelial cell containing some lamellated inclusion bodies (arrows). The basement membrane (BM) of the vessel is multilayered (Ery, erythrocyte) (× 21,500).

confirming the diagnosis of Niemann-Pick disease type A and in the differential diagnosis of the four types (Table). However, even in type A these findings are initially absent as it takes time for the abnormal storage material to accumulate in the cells and manifest itself.

The globe and its inner structures are

TABLE

OCULAR FINDINGS, HISTOPATHOLOGY, AND ULTRASTRUCTURE IN FOUR TYPES OF NIEMANN-PICK DISEASE

Sign or Symptom	Niemann-Pick Disease			
	Type A	Type B	Type C	Type D
Finding				
Cherry-red spot	Frequent	None	Occasional	None
Corneal cloudiness	Present	None	None	None
Brown discoloration of lens	Present	None	None	None
Histopathology and ultrastructure				
Involved inner structures	Marked	Unknown	Slight or none	Unknown
Involved conjunctival epithelium	Slight or moderate	Slight	Moderate or marked	Unknown
Involved conjunctival stroma	Marked	Moderate	Marked	Unknown

severely involved in type A.[8,9] This involvement occurs very early, probably prenatally.[10] In type C, ocular structures have been reported as normal.[11] Ballooned retinal ganglion cells have been found despite a normal fundus and visual acuity.[12] Harzer and associates[13] described pleomorphic and oligomembranous inclusion bodies in neurons in another case of type C. The conjunctiva was not examined in most of these pathologic studies.

Libert and Danis[6] examined conjunctival biopsy material in four patients with type A and found lamellar cytoplasmic bodies including fibroblasts, pericytes, endothelial cells, and Schwann cells widely spread throughout the stromal cells. Epithelial cells were only slightly affected by a few vacuoles. The inclusion bodies were pleomorphic but essentially consisted of dark osmiophilic lamellae with wide electron-lucent spaces. Libert and Danis[7] compared these findings to those for types B and C. In type B, they found far fewer inclusions in all cell types. In type C fibroblasts were essentially involved. To our knowledge, there have been no reports of a multilayered basement membrane around the stromal capillaries in this disease, although it has been described in diabetes[14] and in old age.[15] Conjunctival ultrastructure in Niemann-Pick type C, as described here, resembles that in type A in that the cytoplasmic inclusion body is similar. This may be characteristic of Niemann-Pick disease. The dark lamella probably represents the sphingomyelin and the lucent area represents the cholesterol accumulation. However, in our patient who had type C, epithelial involvement was widespread. The combination of ocular findings and conjunctival ultrastructure (Table) may be helpful in the differential diagnosis of the different types of Niemann-Pick disease.

Summary

A 5-year-old girl with Niemann-Pick disease type C had normal eyes but the conjunctival ultrastructure was abnormal. Lamellar cytoplasmic bodies, characteristic of Niemann-Pick disease, were found in epithelial cells, stromal fibroblasts, endothelial cells, and pericytes of the stromal capillaries. The basement membrane of the capillaries was multilayered. The Golgi apparatus was unusually well developed. The combination of ocular findings and conjunctival ultrastructure may be helpful in the differential diagnosis of Niemann-Pick disease and its subtypes.

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OPHTHALMIC MINIATURE

For myself, I deprecate the alteration; above all things the meddling legislation, which is the order of the day. We, the medical profession, partake of the social character of these realms, which are not yet a republic. Public opinion is the great cement of our complicated relations. Public opinion, in the higher sense of the word, still favors our present medical regulations; still abhors the irregular practitioner in physic, as well as in divinity and law. Legislation, however necessary, up to a certain point, for all three, should never supersede totally the influence of public opinion.

Robert Hull,
Cursory Notes on *The Morbid Eye*,
London, Longman, Orme, Brown,
Green and Longman, 1840

IMMUNOASSAY OF HUMAN TEAR LYSOZYME

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Lysozyme, discovered by Fleming,[1] is widely distributed in nature. Mammalian sources include tears, saliva, serum, neutrophils, macrophages, nasal secretions, and urine.[2] Alteration in the lysozyme level in human tears has been observed in some ocular diseases,[3-10] so its measurement can be of considerable diagnostic value.

Our purpose was twofold: (1) to determine whether the immunodiffusion technique[11] could be used to estimate tear lysozyme levels; and (2) to investigate whether there is any alteration in tear lysozyme level in patients with trachoma.

SUBJECTS AND METHODS

We chose 114 healthy subjects (mean age, 32.6 years) with no ocular or systemic disease seen in the outpatient department between September 1979 and November 1979. They came here mainly for refraction. There were 56 males (mean age, 36.8 years) and 58 females (mean age, 28.6 years). The age distribution of the subjects is given in Table 1.

We also selected 30 patients with trachoma (mean age, 34.0 years) treated in the outpatient department during the same period. There were 13 males (mean age, 34.5 years) and 17 females (mean age, 33.7 years). The diagnosis of trachoma was made by slit-lamp examination. The clinical criteria used for the diagnosis were the presence of mature follicles in the upper tarsal conjunctiva, progressive pannus in the upper part of the cornea, and early signs of cicatrization of the follicles visible with the slit lamp.

We collected tear samples by a method described previously,[12] and stored them at -20°C until assayed. We quantified lysozyme by a single radial immunodiffusion technique originally described by Mancini, Carbonera, and Heremans.[11] We obtained monospecific rabbit anti-human-lysozyme-serum and the reference standard (Kontrollogen E) from the Behring Institute, West Germany. The manufacturer expresses the lysozyme content of Kontrollogen E as micrograms per milliliter by reference to standard curves prepared with crystalline egg-white lysozyme (EL).

Immunodiffusion plates—We melted 1% (wt/vol) Special Agar-Noble, buffered in 0.045 M sodium phosphate, pH 7.4 (containing 0.1 M sodium chloride and 0.1%, wt/vol, sodium azide) in a boiling water bath and then transferred it to a 56°C water bath. We added 0.1 ml of the antiserum to 3 ml of melted agar at 56°C and mixed thoroughly. We poured the agar-antiserum mixture evenly onto a clean glass plate (75 mm × 25 mm) and placed it on a horizontal surface, using a

TABLE 1

DISTRIBUTION OF TEAR LYSOZYME IN NORMAL SUBJECTS

Age (yrs)	No. of Subjects	Mean ± S.D. (mg/ml)	Coefficient of Variation (%)
≤15	20	0.8 ± 0.3	37.50
16-30	37	1.6 ± 0.7	43.75
31-45	28	1.5 ± 0.6	40.00
46-60	21	1.1 ± 0.5	45.45
≥61	8	1.0 ± 0.4	40.00

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leveling table and a spirit level. We removed air bubbles with a Pasteur pipette. We allowed the agar gel to set at room temperature (24°C). We did the lysozyme assay the same day we prepared the plates.

Selection of antiserum concentration—We ascertained the most appropriate antiserum concentration by testing a series of antiserum concentrations in agar gel, choosing the least amount of specific antiserum that gave easily readable ring precipitates. We found this to be 0.1 ml for 3 ml of melted agar.

Dilution of tear samples—As the lysozyme concentration in tears is high, we needed to dilute the tear samples to get a clearly defined ring precipitate. Using a qualitative gel diffusion technique,[13] we found a dilution of 1:25 was satisfactory for the purpose.

Lysozyme assay—We cut small antigen wells with absolutely vertical sides with a gel punch, making sure that no cracks developed at the edges. The wells were 2.4 mm in diameter and 12 mm apart. We removed the agar plugs with a suction device. We measured a 4-μl tear sample with a Hamilton syringe and diluted it with 96 μl of 0.045 M sodium phosphate buffer (pH 7.4) in a 10 mm × 75 mm test tube. We placed 5 μl of diluted tear sample in the antigen wells after mixing. We placed three dilutions of reference-standard known lysozyme concentrations on each plate.

We placed the loaded agar plates in a moist chamber at room temperature (24°C) until diffusion was complete, that is, when daily measurements showed that the precipitation ring failed to increase in size. We measured the diameter of each ring to the nearest 0.1 mm with a magnifying glass with a micrometer scale. We viewed the agar plate against a black background and illuminated it obliquely. We held the plate with the glass side toward the viewer and placed the scale firmly against the glass surface. We used the average of the two perpendicular readings as the measurement for each diffusion ring. When we constructed a standard curve by plotting the squares of the ring diameters against the concentration of the standard solutions, using 1-cm graph paper, we found that it was linear. We constructed a standard curve for each plate and determined the concentration of lysozyme in tear samples with reference to the standard curve. We expressed values as milligrams of lysozyme per milliliter of tears.

Results

It was essential to test the reproducibility of the method. We therefore repeated the assay of lysozyme four times in each of the tear samples from ten different subjects. Results of an analysis of variance are shown in Table 2. The mean sum of squares caused by variation between replicates was less than the mean sum of

TABLE 2

Analysis of variance

Source of Variation	df	Total Sum of Squares	Mean Sum of Squares
Between subjects	9	9.080	1.009
Between replicates	3	0.017	0.006
Error	27	0.243	0.009
Total	39	9.340	—

squares due to error. We concluded, therefore, that there was no significant difference between the replicates and that the test results were reproducible.

In normal subjects, the value of lysozyme was 1.3 ± 0.6 mg/ml. There was no significant difference ($P > .2$) between the level in males (1.4 ± 0.7 mg/ml) and that in females (1.3 ± 0.7 mg/ml). The lysozyme levels in different age groups are shown in Table 1. We found no correlation between age and lysozyme level ($r = -0.04$), although the level in the group aged 16 to 45 years is higher than those of the younger and older age groups.

In patients with trachoma, the level of lysozyme was 0.9 ± 0.5 mg/ml (males, 1.1 ± 0.4 mg/ml; females, 0.8 ± 0.5 mg/ml). There was no significant difference in lysozyme level between males and females in this group ($P > .2$). The level of tear lysozyme was significantly lower ($P < .001$) in patients with trachoma than in the normal subjects in the comparable age groups.

Discussion

Viscometric,[14] electrophoretic,[3,15] and turbidimetric[16] methods have been used to estimate lysozyme level in tears. These techniques were difficult to apply routinely. The bacterial culture used differed in sensitivity to lysozyme. The data obtained by these methods also varied widely and the values were not comparable.[17] Ronen and associates[18] described a spectrophotometric procedure, but the most commonly used method for routine determination of lysozyme is still the lysoplate method.[17] We found the immunoassay method for the estimation of lysozyme in human tears to be reproducible and sensitive for routine purposes.

Bonavida and Sapse[17] found the lysozyme level in tears to be 1.7 mg/ml when measured against human tear lysozyme in normal subjects. Ronen and associates[18] found the level to be 6.1 mg/ml when measured against hen egg lysozyme. However, lysozyme level by the immunoassy method is 1.3 mg/ml when measured against human serum lysozyme, lower than the above values. We know however, that the level of plasma insulin determined by the innumoassay method[19] is also low (21 μunits/ml) compared to the level obtained by other methods (diaphragm assay method, 40 to 80 μunits/ml; epididymal adipose tissue method, 135 to 680 μunits/ml).[20,21]

Many investigators have found that age has no effect on the tear lysozyme level.[8,10,14,22,23] Some found it to be independent of sex as well.[8,10,22] Mackie and Seal[24] noted a gradual decline of lysozyme level with advancing age. Bonavida and Sapse[17] found lower levels of lysozyme at the upper and lower extremes of age. Pietsch and Pearlman[25] observed that the level of lysozyme fell continuously after the age of 10 years. Mukai[9] found that lysozyme reached peak levels in normal human subjects aged 11 to 20 years and tended to decrease thereafter. We found no significant correlation between age and the level of tear lysozyme, although there was a suggestion that tear lysozyme level rises with age and starts falling after the age of 45 years. We also found that tear lysozyme level was unrelated to the sex of the subjects.

Tear lysozyme has been found to be absent or at low levels in some ocular diseases.[3-10] Our study showed that the level is also low in patients with trachoma compared to normal controls. Testing this method by detecting reduction in the lysozyme level in patients with trachoma proved its utility.

Summary

We used a single radial immunodiffusion technique to determine the lysozyme content of human tears. The level

in healthy subjects with this method was 1.3 mg/ml of tears. In patients with trachoma, the level was low, 0.9 mg/ml of tears. Detecting the reduction in lysozyme level in patients with trachoma proved the utility of this method.

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TECHNICAL ASPECTS OF HISTAMINE DETERMINATION IN HUMAN TEARS

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As the measurement of tear histamine level becomes more useful clinically,[1] it is important to evaluate the technical factors that may influence measurement.

We evaluated (1) the conditions of collection, (2) storage, (3) the assay itself, and (4) whether abnormal levels of tear histamine could be detected in any disease.

SUBJECTS AND METHODS

Conditions of collection—We collected tears by placing sponges (Weck-Cel) in the cul-de-sac of the conjunctiva. Once they expanded, we placed the sponges in plastic centrifuge tubes and expressed the fluid by compressing the sponges with a probe.

We used several 10-μl glass pipettes to collect tears from the lacrimal lakes and expressed the fluid into plastic centrifuge tubes.

We placed three Schirmer strips in a capped plastic test tube and weighed the combination of strips, test tube, and cap (Fig. 1). We then placed the strips one by one over the eyelid margin and allowed them to become saturated. At this point, we replaced them in the tube and weighed the tube, cap, and saturated strips again. The difference between the weight of dry strips and container and saturated strips and container was taken to be the weight of the fluid. We assumed a specific gravity of 1 for the tears. We converted milligrams of fluid directly to microliters of tears.

We collected tears on sponges to assess the affect of trauma. Two minutes of firm rubbing of the closed eye with the subject's finger followed the collection. We then repeated the collection procedure for assay.

In order to assess the relationship of histamine level to tear flow we collected tear samples on sponges. We then performed various other tests requiring further tear sampling and took a later sample to compare the histamine level at different points in the flow of reflex tears.

Storage—We divided the tears from the subjects into two portions and stored them for three days at 4°C. In one portion, we separated the aqueous phase from the cells. We then froze both portions (one without cells and the other with whatever cells were in the tear film) at −20°C until assay. In order to assay fresh versus frozen tears we divided the tear samples into two portions. One was frozen at −20°C for 15 minutes and then thawed; the other was not frozen. We assayed both portions on the day of collection.

In order to assess the influence of storage at refrigerator temperature we collected tears and divided the samples in two parts. One portion was stored for three days at 4°C and then assayed; the other was stored for 10 days at 4°C and then assayed.

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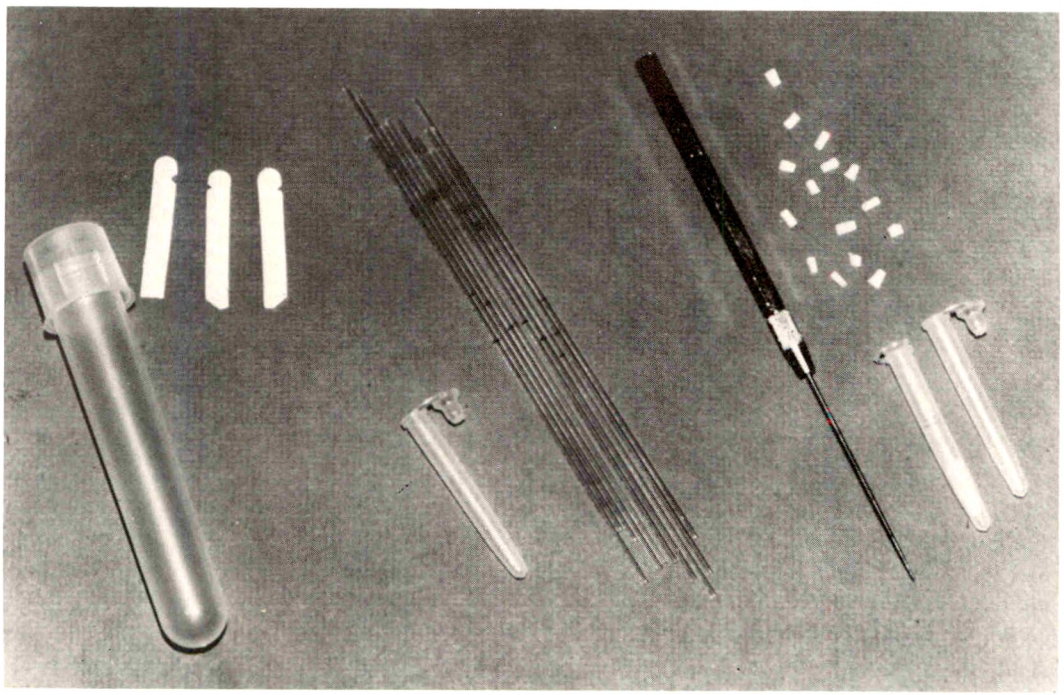


Fig. 1 (Allansmith and associates). Materials for three methods of tear collection. At left, three Schirmer strips fully wetted by tears are placed in a plastic tube. At center, 10-μl disposable glass pipettes collect tears which are then expressed into a plastic tube. At right, pieces of cellulose sponge collect tears; sponges are placed in centrifuge tubes (one has tear-filled sponges in it); a metal probe is used to recompress sponges.

Histamine analysis—We assayed all samples in duplicate by the radioisotope technique described by Beaven, Jacobsen, and Horakova.[2] To determine the error of the technique, we assayed five standards of reagent histamine (1.5, 3, 6, 12.5, and 25 ng/ml) ten times for each level of standard (Fig. 2).

To further investigate the influence of cells on the amount of histamine in tears, we collected samples from persons expected to have high histamine levels (those with vernal conjunctivitis) and those expected to have normal levels. We divided each sample into two parts and stored as already described.

Subjects—We collected tears from 104 subjects: 23 normal individuals, 17 with vernal conjunctivitis, 11 with hay fever comfirmed by a positive skin test (in whom tears were collected during their hay fever season), 16 with giant papillary conjunctivitis associated with soft contact lenses,[3] ten with giant papillary conjunctivitis associated with hard contact lenses, eight asymptomatic wearers of soft contact lenses, six asymptomatic wearers of hard contact lenses, and 13 patients with red eyes from other causes. The last group included four patients with atopic conjunctivitis or atopic keratoconjunctivitis, six with eyes that were red and itchy from an unknown cause, and three with house-dust allergy and itchy eyes.

Statistics—We compared histamine levels in the various groups by geometric means. We calculated the standard deviation range in logs and then converted them to arithmetical values. When handling the zero values for subjects in whom histamine was undetectable, we

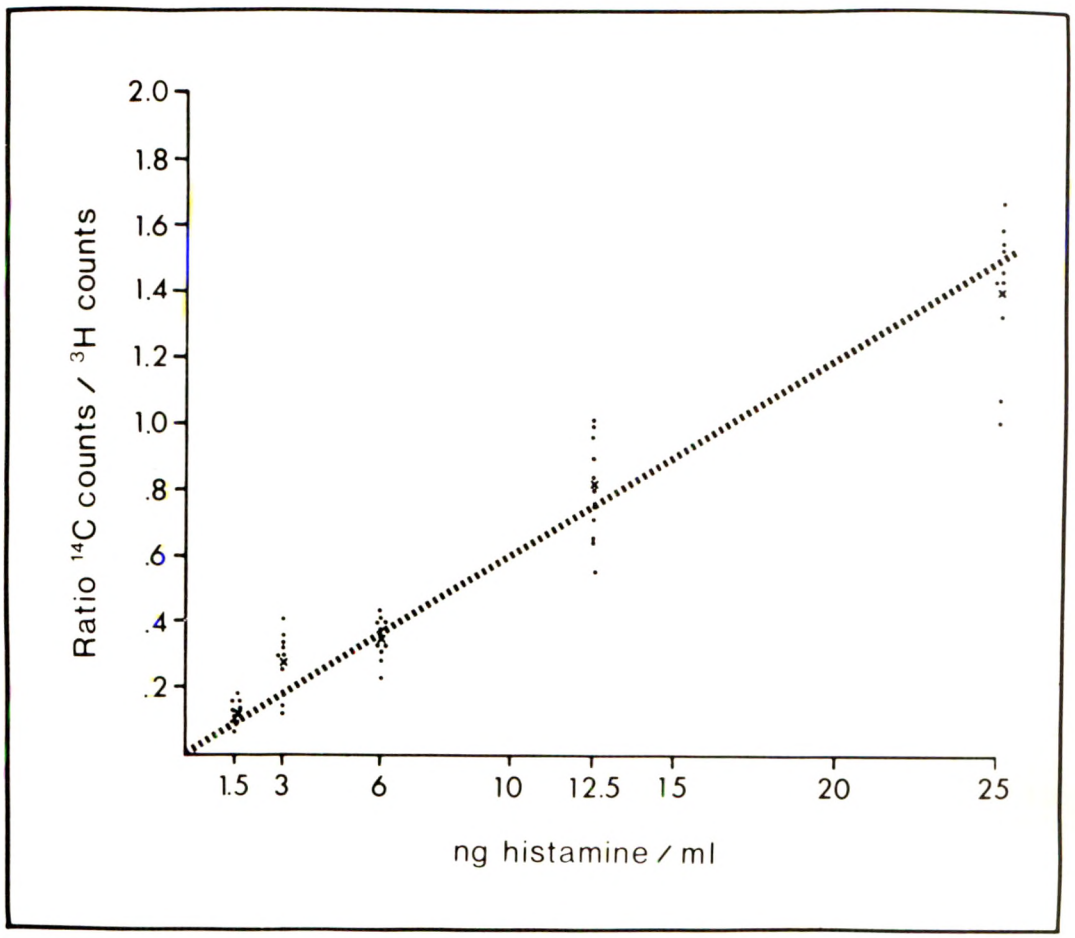


Fig. 2 (Allansmith and associates). Relationship of radioactive counts to nanograms of histamine per milliliter of tears. Note linearity of relationship for range shown. Each dot represents average of two determinations of one standard. The ten determinations at each standard point were performed on different days. Coefficient of variation was ±24%.

took the log of the histamine level plus one for each sample. We used Student's *t*-test for comparisons. We converted the logs of means back to their arithmetical numbers, subtracted one, and used this number as the mean for the group (Table).

Results

Conditions of collection—We collected reagent histamine on five different occasions with sponges, capillary pipettes, and Schirmer strips. The average histamine levels for the three methods were 14 ng/ml (S.D. range, 12–17 ng/ml) for sponges, 18 ng/ml (S.D. range, 16–20 ng/ml) for capillary pipettes, and 17 ng/ml (S.D. range, 16–18 ng/ml) for Schirmer strips. There was no significant difference among the groups ($P>.05$).

In five subjects, we collected tears with sponges and Schirmer strips. The average histamine levels were 9 ng/ml (S.D. range, 2–36 ng/ml) for sponges and 9 ng/ml (S.D. range, 4–22 ng/ml) for Schirmer strips. The differences were not significant ($P>.20$ by paired *t*-test).

TABLE

TEAR HISTAMINE LEVELS IN VARIOUS GROUPS OF SUBJECTS

Group	Histamine Level (ng/ml)			P
	No.	Mean	Range	
Normal	23	5	2-15	—
Vernal conjunctivitis	17	16	4-62	.001
Hay fever	11	4	0-16	NS
Giant papillary conjunctivitis				
Soft contact lenses	16	7	2-19	NS
Hard contact lenses	10	2	0-5	.02
Aysmptomatic				
Soft contact lenses	8	2	0-9	NS
Hard contact lenses	6	2	0-7	NS
Red eyes from other causes	13	2	0-3	.001

In five subjects, we collected tears before and after eye rubbing. The histamine levels were 8 ng/ml (S.D. range, 3–22 ng/ml) before rubbing and 9 ng/ml (S.D. range, 3–24 ng/ml) after rubbing ($P>.1$ by paired t-test).

Tear flow—In nine subjects, we collected the initial 100 μl of tears with Schirmer strips. After further tear collection for other assays, we collected another 100 μl of tears from the same nine persons. The early tear flow samples had an average histamine content of 10 ng/ml (S.D. range, 2–40 ng/ml); the later samples contained an average of 7 ng/ml (S.D. range, 1–27 ng/ml) ($P>.05$).

Storage—In five normal subjects, we collected tears and divided the samples into two portions. In one portion, we separated the aqueous phase from the cells by centrifugation. We froze both portions (one without tear film cells and the other with these cells) at −20°C until the day of assay. Histamine from fluid with cells measured 6 ng/ml (S.D. range, 4–11 ng/ml); in fluid without cells, histamine measured 7 ng/ml (S.D. range, 4–11 ng/ml) ($P>.05$).

To evaluate the effect of freezing on tear histamine levels, we collected samples from five subjects and divided them into two portions. We froze one portion at −20°C for 15 minutes and then thawed it; the other was not frozen. The average histamine content of the frozen samples was 3 ng/ml (S.D. range, 0–11 ng/ml), and 2 ng/ml (S.D. range, 0–7 ng/ml) for the unfrozen specimens ($P>.05$).

Next we investigated the influence of storage at refrigerator temperatures. We collected tears from five subjects and divided each sample in two. We stored one portion for three days at 4°C and then assayed it; we stored the other for ten days at 4°C and then assayed it. The average value for the material stored for three days was 4 ng/ml (S.D. range, 1–9 ng/ml); for the material stored for ten days, it was 3 ng/ml (S.D. range, 1–9 ng/ml) ($P>.05$).

Tear film cells—To determine the influence of the cell fraction on tear histamine level in individuals with normal and high histamine levels, we collected samples from 14 subjects with normal eyes and 12 patients with vernal conjunctivitis. We removed the cells and resuspended them to the original volume of tears. The average histamine content of tears from normal subjects was 1 ng/ml (S.D. range, 0–5 ng/ml). The cell fraction from this normal group had 0 ng/ml (S.D. range,

0–1 ng/ml). Patients with vernal conjunctivitis had an average of 27 ng/ml (S.D. range, 18–42 ng/ml). The cell fraction from this latter group was 6 ng/ml (S.D. range, 2–14 ng/ml).

After analyzing each of five standard points ten times, we found that the ratios of the counts of radioactive carbon were linearly related to the nanograms of histamine expected to be in the samples. The average coefficient of variation of the technique was 24%. The lower limit of sensitivity was 1.5 ng/ml for a 50-μl sample.

Subjects—Only the group with vernal conjunctivitis had increased tear histamine levels (Table) ($P<.001$). The 13 subjects with red eyes from causes other than vernal conjunctivitis had significantly decreased tear levels ($P<.001$) as did the ten with giant papillary conjunctivitis associated with hard contact lenses ($P<.02$).

Discussion

Histamine levels can be measured in the tears of most people. Various techniques of collecting tears can be used, none of which influences the level measured. Trauma, at least to the extent of vigorous eye rubbing, neither breaks up sufficient mast cells nor causes exudation of serum into tears at a rate that changes tear histamine levels. Whether the tears are stored for a number of days, whether they are frozen and thawed, or whether the cells are removed also do not influence the histamine level. The assay is a stable one with a variation of about ±24%.

It is possible to detect an elevation of tear histamine in some individuals. To date, this applies only to those with vernal conjunctivitis.[4] In a histologically similar condition, giant papillary conjunctivitis, tear histamine levels are not elevated.

Persons with red eyes from causes other than vernal conjunctivitis had consistently lower-than-normal tear histamine levels ($P<.01$). Previous work has shown that extremely red eyes leak serum.[5] It is also known that serum has extremely low histamine levels (0.012 ng/ml).[2] The reason for lower tear histamine in persons with red eyes is not known. Since our subjects' eyes were not as red as eyes in which serum leakage was shown,[5] it is unlikely that the leakage of fluid with low histamine content lowered tear histamine levels, but this remains to be studied. Possibly the low level of histamine in the tears of those with red eyes from causes other than vernal conjunctivitis was caused by high levels of histaminases in the tear film rather than by low histamine production.[6] This also remains to be studied. Wearers of hard contact lenses who had giant papillary conjunctivitis had significantly decreased levels of tear histamine ($P<.01$). The importance of this finding is unknown.

Summary

We evaluated various technical aspects of tear collection to assess their influence on the measurement of tear histamine levels. We included several methods of collecting tears, storage of tears with and without freezing, storage for three vs ten days, and removal vs nonremoval of cells before freezing. We assayed tear samples in various groups of people with inflamed eyes. None of the various means of collecting or storing the tear samples influenced the histamine measured. The measuring technique is a stable one that requires minimal treatment of samples before assay. To date, we have found that only patients with vernal conjunctivitis have elevated tear histamine levels. Any increase of tear histamine over 10 ng/ml (geometric mean plus 2 S.D.) or any significant decrease is probably related to the disease itself and not to factors

related to the technique used to measure tear histamine.

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Fifty years ago this month in THE JOURNAL:

Necessity of filtering scar for successful iridotasis or iridencleisis.

Question: In iridotasis or iridencleisis operations is it necessary to secure a filtering scar—i.e., a bleb beneath the conjunctiva—in order to obtain a permanent lowering of tension? Why, with the same technique, do some eyes have blebs and others not?

Answer: (from Dr. George S. Derby, Boston): In order to obtain a permanent lowering of the tension a filtering scar must be obtained. In some cases, however, with a minimal bleb or no demonstrable one, enough filtration appears to take place to regulate the tension in that particular case. The answer to the last question is beyond our certain knowledge.

Am. J. Ophthalmol. 13:1042, 1930

NOTES, CASES AND INSTRUMENTS

A FIBEROPTIC FIXATION TARGET FOR TONOGRAPHY

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Tonography is a useful adjunct to the supervision of glaucoma, but it is flawed by poor reproducibility and by irregularities in graphing that leave too much leeway for the interpreter.

One of the sources of error is wandering of the eye under the plunger. For the monocular patient, the conventional fixation target is useless. He may also suffer anxiety and, possibly, claustrophobia as all vision is shut out. A phoriatropic eye may drift in and out of binocularity as its position shifts under the plunger.

We devised a fiberoptic core (Fig. 1) to replace the standard Müller tonography plunger (Fig. 2). This core consists of approximately 60 fibers, each 0.45 μ in diameter. We calculated the lens strength and attached a protective lens to the footplate curvature. We easily performed the necessary recalibration of the 0 set and 7 set. Any convenient light source placed above the fiberoptic plunger will serve adequately.

SUMMARY

Substituting a fiberoptic core for the standard Müller tonography plunger provides a fixation target for tonography. The fiberoptic is particularly useful for testing patients who are difficult to examine with the conventional plunger (those with monocular vision, phoriatropia, and the like).

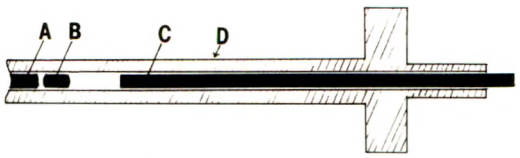


Fig. 1 (Lubkin and associates). Cross-section of experimental plunger. A, Protective lens; B, lens; C, fiberoptic bundle; D, conventional Müller plunger.

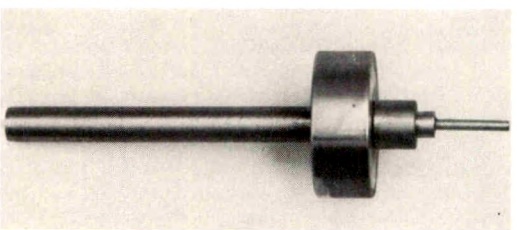


Fig. 2 (Lubkin and associates). External view of conventional Müller plunger.

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UNILATERAL RIEGER'S ANOMALY

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Rieger's anomaly is characterized principally by a prominent anteriorly displaced Schwalbe's ring, iris strands and

From the Department of Ophthalmology, University of Wisconsin Medical School, Madison, Wisconsin. This study was supported in part by grant 1 K04 EY00137 from the National Eye Institute.

Reprint requests to Paul L. Kaufman, M.D., Department of Ophthalmology, University of Wisconsin, Clinical Science Center, 600 Highland Ave., Madison, WI 53792.

bands spanning the iridocorneal angle and inserting into the prominent Schwalbe's ring, and hypoplasia of the anterior iris stroma. Corectopia, myopia, astigmatism, microcornea, and glaucoma may be present.[1-4] Although asymmetry has been noted, all previous reports have stressed the bilaterality of the condition. We studied a patient with unilateral Rieger's anomaly.

Case report

A 53-year-old diabetic man came here for elective repair of an abdominal incisional hernia. He had had adult-onset diabetes mellitus for at least 14 years and also had essential hypertension. He had undergone cholecystectomy for cholelithiasis 14 years previously. The patient stated that his family, consisting of both parents, two siblings, and seven children, had no history of ocular disease, no symptoms, and no unusual ocular appearance. A thorough physical examination disclosed no unusual findings except an incisional cholecystectomy hernia and the ocular abnormalities. Results of complete blood cell count, serum electrolytes study, urinalysis, chest X-ray, and electrocardiogram were normal.

Ocular examination—Manifest retinoscopy and refraction were R.E.: −2.25 +4.25 × 120 and L.E.: 6/6-3 +1.25 sphere. Best corrected visual acuity was R.E.: 6/6-3 (20/20–3) and L.E.: 6/6 (20/20). Best corrected near visual acuity, with a +3.00 sphere, was J1 at 33 cm in both eyes. Keratometry readings were R.E.: 39.25 × 180 ⌒ 45.00 × 90 and L.E.: 42.00 × 175 ⌒ 43.50 × 85. Axial globe length (A-mode ultrasound) was R.E.: 22.58 mm and L.E.: 22.68 mm. The eyelids and eyelashes were normal. The interpupillary distance was 67 mm. The medial intracanthal distance was 34 mm. The palpebral fissures measured at distance fixation horizontally were 28 mm in the right eye and 29 mm in the left eye; vertically, they were 7 mm in both eyes. Motility examination showed orthophoria at distance and near by cover testing, with full ductions and versions. The conjunctivas were normal. The horizontal diameter of the corneas from corneoscleral limbus to corneoscleral limbus was R.E.: 10.75 mm and L.E.: 11.70 mm.

On anterior segment biomicroscopy of the right eye, the cornea displayed a prominent, thickened, anteriorly displaced Schwalbe's ring (posterior embryotoxon). The cornea was otherwise normal, and the anterior chamber was deep and clear. The pupil was updrawn (corectopia) and slightly elongated vertically, measuring 2.5 mm horizontally by 3.0 mm vertically. The anterior iris surface was distinctly hypoplastic, especially superiorly from the 10- to 2-o'clock positions. In this sector, the collarette was absent and crypts and furrows were ill-defined (Figure). The radial fibrils of the deep posterior iris stroma gave the superior iris a distinctly grayish hue.

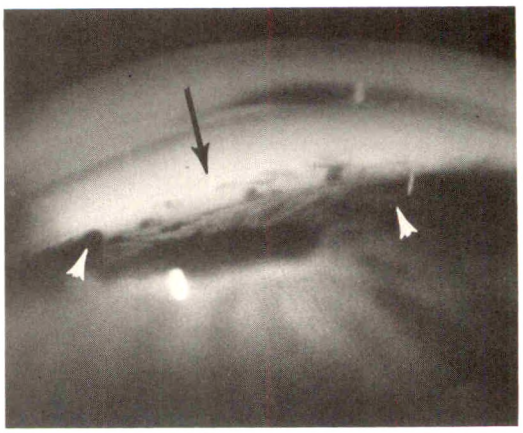


Figure (Bundy and associates). Affected right eye. Gonioscopy of the superior chamber angle demonstrated broad iris bands inserting into the widened trabecular zone and the thickened, anteriorly displaced Schwalbe's ring (arrow). Iris collarette is visible at arrowheads, but attenuated or absent between them.

Polycoria, retroillumination defects, and uveal ectropion were not present.

Gonioscopy disclosed a deep anterior chamber, distinctly deeper superiorly in the area of hypoplastic iris. Schwalbe's ring was thickened and displaced anteriorly, and the trabecular zone widened accordingly. Iris strands of varying thickness bridged the iridocorneal angle, inserting into the trabecular zone and Schwalbe's ring.

Biomicroscopic and gonioscopic findings in the left eye were within normal limits. The pupil was centrally located, round, and 3.5 mm in diameter. A few fine iris processes extended to the ciliary band or scleral spur inferiorly.

The pupillary light reflex was directly and consensually normal in both eyes. Intraocular pressure by Goldmann applanation was R.E.: 18 mm Hg and L.E.: 16 mm Hg. Tonographic values (5.5 g weight) were R.E.: P_0 = 18 mm Hg, C = 0.42 μl × min^{-1} × mm Hg^{-1} and L.E.: P_0 × 17 mm Hg, C = 0.45 μl × min^{-1} × mm Hg^{-1}. Goldmann visual fields in the right eye disclosed blind spot baring to I_2e object, but no paracentral or peripheral defects; the left eye was normal. Examination with the pupils dilated disclosed clear lenses without star-shaped capsular pigmentation, mild background diabetic retinopathy, normal optic nerve heads with round cups, and a 0.4 cup:disk ratio in both eyes.

Three of the patient's seven children received ocular and physical examinations and were entirely normal.

Discussion

The classic triad of Rieger's anomaly—prominent, anteriorly displaced Schwalbe's ring, broad iris strands insert-

ing into Schwalbe's ring, and anterior iris stromal hypoplasia—along with other associated ocular abnormalities (such as mild microcornea and marked astigmatism[1-4]) were clearly present in the right eye of our patient but not in the left. No facial, dental, or skeletal abnormalities were present, confirming the diagnosis of Rieger's anomaly rather than Rieger's syndrome.[3,4] Although asymmetric involvement has been described, this is, to our knowledge, the first truly unilateral case reported.

Summary

A 53-year-old man, to our knowledge the first person described with unilateral Rieger's anomaly, had a prominent anteriorly displaced Schwalbe's ring, broad iris strands inserting into Schwalbe's ring, anterior stromal iris hypoplasia, mild microcornea, and marked astigmatism in his right eye. His left eye was entirely normal.

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MEETINGS, CONFERENCES, SYMPOSIA

EDITED BY THOMAS CHALKLEY, M.D.

OCULAR MELANOMA TASK FORCE REPORT

In response to a resolution passed by the National Advisory Eye Council, the National Eye Institute convened a task force to evaluate the published reports on ocular melanoma and to recommend what research is needed to solve problems associated with this disease. The stimuli for this action were conflicting conclusions and recommendations appearing in published reports about the management of ocular melanoma and a controversy stemming from the interpretation of mortality data on patients with uveal melanomas who underwent enucleation compared with those who remained untreated.

Four months before the task force meeting, the participants were sent more than 700 abstracts from which to select relevant articles on the epidemiology, diagnosis, treatment, natural (untreated) course, and biology of ocular and cutaneous melanoma. Approximately 150 articles were chosen for critical review. The participants were asked to analyze the articles in terms of three questions:

What do we know?
What do we need to know?
How do we find out?

The task force met April 17 and 18, 1980, at the National Institutes of Health, Bethesda, Maryland, to discuss its findings. Its recommendations are summarized in the following sections.

Complete transcripts of the meeting and the bibliography reviewed by the task force participants may be obtained by writing to Bettie J. Graham, Ph.D., Room 6A-52, Building 31, National Eye Institute, National Institutes of Health, Bethesda, MD 20205.

EPIDEMIOLOGY

Uveal melanomas (choroidal, ciliary body, and iris) are the most common primary intraocular malignancies in adults and constitute over 80% of all eye malignancies. In the United States, the annual incidence rate is six cases per one million population, that is, approximately 1,500 new cases each year. Limited data are available on the risk factors associated with uveal melanomas. White populations are at a higher risk than members of other races: ocular melanomas occur eight times more frequently among white than among black Americans. The risk of uveal melanoma increases greatly with age: the median age at diagnosis is approximately 55 years and the peak incidence is between 60 and 69 years. International data on incidence and mortality rates for all ocular tumors show higher rates for males than for females.

Most clinical studies of uveal melanoma present comprehensive data on ocular findings, yet data on possible risk factors other than age, sex, and race are seldom recorded. Additionally, practically all studies lack adequate control groups, thus limiting the interpretation of descriptive data on uveal melanoma patients. To increase our understanding of the epidemiology of uveal melanomas we need to know:

What are the risk factors for uveal melanoma? For example, eye, skin, and hair color; nevi and other cutaneous findings; sunlight exposure; laterality of tumor by sex; previous ocular conditions; hormonal factors; sociodemographic factors; occupational or other exposure to chemical carcinogens; and family history.

What are the patterns of occurrence of

uveal melanoma over time, and in different geographic areas, especially where the risk of ocular tumors is known to be high?

Is there a correlation between the incidence of cutaneous and uveal melanomas? Do they share the same risk factors?

At least two types of epidemiological studies would address some of the questions posed. Analytic epidemiologic studies of the case-control type are needed to determine whether certain factors are associated with an increased risk of uveal melanoma. Such studies would also estimate the magnitude of risk involved. Descriptive epidemiologic studies would evaluate past and present patterns of incidence and mortality and provide baseline data for monitoring future trends. By analyzing existing data on ocular tumors and separating melanomas from all other histologic types, a trend in occurrence might be determined.

Diagnosis

Clinical diagnosis of ocular melanoma is remarkably accurate, considering that it is usually performed without the benefit of histopathologic material. Errors in diagnosis in ocular melanoma have become increasingly rare in eye centers where experienced physicians and ancillary diagnostic testing are available. Currently, the mainstay of diagnosis in ocular melanoma remains clinical examination including pupillary dilation and indirect ophthalmoscopy. Ancillary diagnostic tests, while nonspecific, can be extremely helpful in confirming the clinical diagnosis.

Ultrasound—Examination of the eye by ultrasound may improve the accuracy of diagnosis in eyes with opaque media and can add significantly to diagnostic accuracy in eyes with clear media. The combined use of A- and B-scan techniques is ideal, and A-scan techniques can often provide size data for serial measurements. Ultrasonography is extremely helpful in distinguishing between melanomas and some lesions which simulate melanomas, especially choroidal hemorrhage or hemangioma.

Serial fundus photography—This technique is useful in evaluating lesions which are located posteriorly and can be included in a single field of the fundus camera. Problems in reproducibility must be considered when evaluating serial photographs. Stereoscopic fundus photographs are useful in evaluating changes in small lesions, where minimal degrees of elevation are present. Newer wide-angle fundus cameras will greatly expand the utility of photography in following the course of the tumor.

Fluorescein angiography—This technique can help differentiate between melanomas and lesions which simulate melanomas, especially choroidal hemorrhage and hemangioma. Damage to the retinal pigment epithelium and neovascularization along the plane of Bruch's membrane, detectable by angiography, may be a guide to growth in choroidal tumors.

Radiologic examination—Standard X-ray and computed axial tomography have limited use in the diagnosis of most ocular melanomas, but can confirm the presence of osseous choristoma or help to assess extraocular or optic nerve extension of the tumor.

Radioactive phosphorus (^{32}P) uptake test—This is another ancillary test which is utilized by some clinicians and may not always be necessary in routine cases where diagnosis of ocular melanoma can be made with less complicated procedures.

Complete evaluation of the patient's health status is essential and provides baseline information for monitoring treatment or progression of disease. Patients with ocular melanoma should have a

complete physical examination and clinical laboratory studies, including routine blood work, chest X-ray, liver enzyme measurements, and, probably, radioisotope liver and spleen scans. These tests should be supervised and evaluated by physicians who are experts in the diagnosis and treatment of tumors.

At present, clinical observation and documentation of growth remain the most reliable ways to differentiate between benign nevi and melanomas. The diagnostic tool most needed in evaluating ocular melanoma is one which would differentiate between a nevus and a small melanoma and reliably measure tumor growth, in addition to being noninvasive. Techniques which are relatively new in ophthalmology, such as immunologic tests, are exciting areas for future development.

Pathology

Histologic examinations of ocular tumors can be useful to the clinician when the relationship between the histopathologic features of the tumor can be correlated with the natural course of the disease or the prognosis. In order to achieve this goal it is necessary to:

- Develop standard terminology to describe and compare such aspects of ocular tumors as uveal nevus, uveal melanoma, tumor size, and necrosis.
- Develop uniform methods of preparing tumor-bearing eyes for histologic, ultrastructural, and biochemical studies.
- Define how events in the natural course of the tumor are reflected in the histopathology of the tumor and how such findings correlate with prognosis.
- Improve the cellular criteria for categorizing tumors by the Callender classification and expand the Callender classification to an ultrastructural level to relate morphologic findings with tumor behavior and prognosis.

The above tasks can be accomplished by convening a panel of experienced ophthalmic pathologists and oncologic pathologists who could formulate terms and definitions that would be accepted and widely utilized by the ophthalmic community. The same or a similar panel, in a masked study, could review retrospective cases with defined histories in an attempt to relate significant histologic features to prognosis. As a control, eyes from patients with nonmetastasizing lesions should also be included in the survey. These studies should be complemented by correlating ultrastructural changes in tumor cells with malignant potential.

Natural course

Knowledge of the natural course of uveal melanomas is critical for designing treatment regimens, and such information also serves as the control for their evaluation. Most of our present information on ocular melanoma is inadequate and is derived from retrospective studies done after treatment, usually enucleation. While small tumors probably evolve into large tumors given sufficient time, and in so doing become more likely to produce metastatic disease, this concept is based on few actual observations. Since most melanomas are enucleated promptly, especially if they show signs of growth, very limited data are available regarding outcome without treatment. Additionally, very few cases have been followed with quantitative measurements over a sufficient number of years to provide any prospective data on the importance of growth rate to prognosis, and virtually nothing is known about the frequency of spontaneous regression in ocular melanoma, although it occurs in skin melanomas. The data needed to define the natural history of ocular melanomas include:

- Determing the natural course in large numbers of patients with lesions of varying size.

Determining which of the following clinically measurable factors are important in determining metastasizing potential and prognosis of uveal melanoma: size, location, degree of pigmentation, transillumination, surface alterations such as yellow pigment, drusen, retinal pigment, epithelial damage, neovascularization, collar-button extension, retinal detachment, vitreous or choroidal hemorrhage, secondary glaucoma, and visual field changes.

How does tumor growth rate and size correlate with prognosis?

Do nevi represent the only recognizable precursor lesions for choroidal melanomas or do some melanomas develop de novo?

Can the hormonal status of the individual or other factors influence the growth rate of uveal melanomas?

What is the frequency of spontaneous regression in ocular tumors?

How does the patient's immune state relate to prognosis?

Are there skin changes that correlate with prognosis in ocular melanoma, for example, vitiligo, or growth or change in pigmentation of skin nevi?

Melanomas of the ciliary body and iris, as well as malignant melanomas of the conjunctiva and eyelid, appear to be clinically different from choroidal melanomas; therefore, the natural course of each of these tumors should be evaluated separately.

A prospective study done before treatment of patients with uveal melanoma would provide needed information on the natural course of this disease. The study protocol and standardized data collection would be developed in collaboration with ophthalmologists, oncologists, and epidemiologists. Patients entering the study would receive a comprehensive evaluation to determine risk factors as well as local and systemic factors which might influence possible prognosis. After the initial evaluation, patients would be examined at regular intervals and complete follow-up data obtained. Considering the present variation in management, many patients with small-to-medium-sized lesions would probably be observed for a long period before treatment. In many cases, it would be possible to collect data on the epidemiology and natural course simultaneously.

Therapy

Effective intervention should alter the natural course of the primary lesion, or prevent metastases, or delay recurrence. The prognosis for local intraocular melanomas depends upon cell type, lesion size, stage of the disease, age, location in the uvea, mitotic activity, and, possibly, sex. The relative effectiveness of radiation therapy, such as rhodium 106 and proton beam, is not known, and the benefits of cryosurgery have not been properly evaluated. However, retrospective studies of patients with small melanomas treated by enucleation confirm their relatively favorable prognosis. The prognosis of patients with uveal melanoma with extrascleral extension is poor, and the cure rate is less than 50%. Exenteration of the orbit is probably the most commonly used procedure, although the precise technique (partial vs complete) varies among ophthalmic surgeons. In evaluating the data, it is often unclear whether cases of extrascleral extension have residual tumor in the orbit or extend near or to the line of transection. The pattern of metastatic disease in ocular melanoma is similar to that of cutaneous melanoma, with a single obvious exception: the lack of lymphatic drainage of the eye precludes the presence of lymph-borne metastases. Hepatic metastases tend to predominate in uveal melanoma, but the disease is capable of widespread metastatic dissemination and this complication should be suspected and explored in every patient. In any case, the disease is probably incurable at this stage. For tumors no longer confined

to the orbit, surgery, perhaps in combination with other therapy, is only palliative.

The interpretation of available data on the prognosis and efficacy of enucleation of medium and large tumors remains controversial. The recognition of metastasis before enucleation is very infrequent (1% to 3%) but no valid data exist. Additionally, the high mortality from metastases after surgery is not unique to uveal melanomas, but is characteristic of many other neoplasms.

Proper management of uveal melanomas requires knowledge about appropriate clinical technology and the basic biology of the tumor. Questions about appropriate clinical technology include:

- Is there a subgroup of primary ocular melanomas, based on size and rate of growth, for which observation alone is the optimal management choice?
- What is the effect of enucleation on the natural course of primary ocular melanoma?
- Can preoperative or postoperative radiation improve results when combined with enucleation?
- Can adjunctive therapy with immunotherapeutic agents such as thymosin, interferon, or transfer factor improve the outcome of treatment?
- Is there an optimal surgical procedure for orbital exenteration?
- Is antipigmentary therapy of any value in treating ocular melanoma?

A rigorous test of the efficacy of treatment on disease outcome is best accomplished by a randomized, controlled clinical trial. The feasibility of a clinical trial in this disease is greatly limited by insufficient information on the natural course of the disease; this is especially true for small melanomas without extraocular extension. However, since the prognosis of large melanomas or melanomas with extrascleral extensions is poor, patients with tumors in these two categories and their ophthalmic surgeons might ethically consider participating in a randomized controlled clinical study.

Biology of ocular melanomas

Understanding the biological and biochemical properties of uveal melanoma cells is a key to effective diagnosis and treatment. Our knowledge of ocular melanomas has been hampered by lack of cell lines or strains of human uveal melanomas and appropriate animal models. Some of the questions that need to be addressed include the following:

- What are the causative agents in ocular melanoma, for example, viruses, chemicals, environment?
- Are there receptors for hormones and vitamin A on melanoma tumor cells?
- Do melanoma tumor cells have specific enzyme requirements which explain their high incidence of liver metastasis?
- Can early ocular melanomas be induced to differentiate into nevi?
- Are some histopathologic types of tumor cells more hormonally sensitive or dependent than others?
- Are growth factors present within the melanoma tumors?
- Since there are variations within melanin pigments, are there specific pigments associated with melanomas?
- Are there tumor-specific antigens associated with the different cell types? If so, can monoclonal antibodies be produced against these antigens for immunodiagnostic purposes?
- Do patients develop cell-mediated or humoral antibodies to melanoma tumor cells? If so, how does the host's immune competency affect the behavior of the tumor and the development of metastasis?

To study the biology of tumor cells at the cellular and molecular level will require the establishment of tumor cell lines and strains, and the availability of properly prepared fresh human donor materials (eyes and sera). The development of appropriate animal models is also essential. Additionally, the latest technology should be used in solving problems associated with ocular melanoma.

In conclusion, the task force agreed that one of the first steps in achieving the desired goal of determining how to manage ocular melanomas properly is to pursue epidemiologic and natural course studies. This does not rule out a randomized controlled clinical trial of carefully chosen treatment(s) using a select group of patients with poor prognoses. Since the number of patients with ocular melanoma is relatively small, a collaborative effort between ophthalmologists caring for melanoma patients and scientists involved in ocular melanoma research is needed. Underlying these recommendations was the task force's belief in the need to share resources, the need for greater cooperation among ophthalmologists, and the need for increased communication among ophthalmologists, epidemiologists, and medical oncologists.

BETTIE J. GRAHAM
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THREE INTERNATIONAL MEETINGS ON SPECULAR MICROSCOPY

Three international meetings on specular microscopy of the human corneal endothelium occurred in the spring of 1980: the Workshop on Specular Microscopy of the Corneal Endothelium, hosted by F. Bigar in Zurich, Switzerland, on April 14 and 15; the Round Table on Specular Microscopy, chaired by R. Buckley at the VI European Congress of Ophthalmology in Brighton, England, on April 25; and the 1980 Specular Microscopy Symposium, organized by P. Binder preceding the Association for Research in Vision and Ophthalmology meeting in Orlando, Florida, on May 4.

These meetings provided information on the current state and future direction of specular microscopy.

Optical problems—In a historical survey, F. Bigar (Zurich) emphasized that A. Vogt's remarkably accurate drawings contained details now being observed with specular photomicrography. R. Laing (Boston) pointed out that the specular image is formed by light reflecting from each optical interface in the cornea combined with the light scattered back from the various tissue layers. The wider the slit used, the more cells can be photographed; the wider slit, however, increases the overlap of the reflected and scattered light, reducing the image contrast. A. Thaer (Zurich) sought to improve image contrast by reducing the light scatter with a narrow slit and reducing the reflections from the corneal surface with an objective lens or contact solution that has an index of refraction similar to that of the cornea. C. Koester (New York) stated that, since the endothelial specular image reflected only about 0.02% of the incident light, there

must be optical separation of the incident and reflected light for proper image contrast. He showed endothelial photographs taken with an experimental wide-field specular microscope with an oscillating mirror that scans the cornea 1,000 times per second, capturing over 2,000 cells per photograph in both contact and noncontact modes. Both Thayer and Koester demonstrated television systems capable of displaying a live endothelial image, recording it for future study, and relaying it to an image processor for quantitative computer analysis.

E. Sherrard (London) and A. Yamanaha (Kobe) demonstrated the Pocklington microscope. It takes wide-field photographs through a floating, dipping cone composed of materials with progressively decreasing indices of refraction applied to a soft contact lens on the cornea. Using a mosaic of photographs, Sherrard and R. Buckley (London) described posterior corneal rings that look like wrinkles in Descemet's membrane, form a circular pattern distinctive for each human cornea, do not appear in 13 of 14 other species examined, and provide a landmark for relocating the same endothelial area on subsequent examinations. J. Strobel (Giessen) compared Zeiss noncontact, the Bio-Optics, and Leitz contact specular microscopes. He preferred the Leitz because it has variable magnification and a dipping cone that does not increase intraocular pressure. (Recent Bio-Optics models have variable magnification and a nonapplanating objective.) N. Price and H. Cheng (Oxford) compared the Nikon noncontact with the Heyer-Schulte contact specular microscopes, and preferred the former because of the wider field, higher proportion of acceptable photographs, and greater ease of operation.

Both C. Roberts (New York) and R. Laing (Boston) compared contact and noncontact methods. Three currently available microscopes can be used in both modes: the Bio-Optics, the Leitz, and the Keeler-Pocklington. The contact method is easier and faster, since it holds the eye steady, allows a rapid survey of different areas, and has better resolution with higher magnification. However, those instruments which require applanation of the cornea may induce artifacts, often reduce patient tolerance, and make photography in the immediate postoperative period more difficult. The noncontact mode produces a longer field of view with less resolution and is better tolerated by patients, but is more difficult to use, because ocular microsaccads and surface irregularities alter the alignment of specular reflection. B. Holden (Kensington, Australia) emphasized that the angles of illumination and observation must be precisely controlled during noncontact specular microscopy for proper calculation of magnification.

Methods of measurement—G. Waring (Atlanta) found that measurements of cell density and mean individual cell area were equally precise with a rectangle, a planimeter, and a computerized digitizer. He emphasized that endothelial cell size varies greatly at any given age, so that cell density cannot be reliably predicted from the age of the patient. After the age of 20 years, endothelial cell size is log-normally distributed. A thorough description of the endothelial population includes mean cell size (density or mean cell area), spread of cell sizes (standard deviation), and the symmetry of the population (coefficient of skewness). S. Sperling (Aarhus) illustrated a theoretical model showing that counting cells in a fixed frame overestimates the number of cells. F. Bigar (Zurich) found that the endothelial cell density did not decrease linearly with increasing age, but fell rapidly in the first three decades of life and then leveled off slightly, with wide variation at each age. The density was similar in both eyes of one patient.

Using computerized measurement, G. Rao (Rochester, New York) found that although some individual endothelial cells in normal individuals have four to

eight sides (mean, 5.5 sides), 75% have six sides. He found no correlation between cell shape and cell size.

G. Waring (Atlanta), W. Bourne (Rochester, Minnesota), and P. Rejman (Zurich) all described systems of computerized image analysis of either negatives or prints using various types of image enhancement. The indistinct cell borders and the black or white spots within cells have thwarted these attempts, so that only tracings of cell outlines can be directly image-scanned at present.

Clinical and pathologic correlations—D. Maurice (Palo Alto) showed the first endothelial specular photomicrograph and also described a scanning microscope for wide-field photographs in vitro. He thought most black or white spots on specular photographs occur because of irregularities on the posterior endothelial surface.

C. Koester (New York) explained that the light spots on specular photomicrographs come from either increased reflection from a concave surface or a structure that is dense, while the black spots arise from structures that allow light to pass across the endothelial surface without reflection.

B. Schimmelpfennig (Zurich) used a perfusion chamber to observe white spots between cell borders during cell death; he thought these spots corresponded to breakdown of apical junctions.

W. Bourne (Rochester, Minnesota) studied corneas with early cornea guttata and no clinical edema and found that they were thicker, had larger endothelial cells, and had a greater endothelial permeability to fluorescein by fluorophotometry despite a normal endothelial pump rate, indicating defective barrier function. Bourne also demonstrated progressive endothelial cell loss for more than two years after penetrating keratoplasty, whereas cell density stabilized by one year after intraocular lens implantation. He found a 16% cell loss after both intracapsular and extracapsular cataract extraction with intraocular lens implantation. Both G. Rao (Rochester, New York) and Bourne observed a greater increase in corneal thickness after cataract extraction when the preoperative variation in individual endothelial cell size was great. R. Laing (Boston) described a method of measuring the autofluorescence of the flavoproteins in the wall of mitochondria and the pyridine nucleotides as an indicator of metabolic state and endothelial cell death.

C. Ohrloff (Bonn) demonstrated that there is less endothelial cell loss when phacoemulsification is performed in the posterior chamber rather than in the anterior chamber.

Both R. Laing (Boston) and P. Binder (San Diego) emphasized the dynamic nature of the endothelium and described the motion of structures and reflections within cells in vivo, and morphologic changes after surgical or hypoxic insult.

Both G. Rao (Rochester, New York) and Ruusuvara (Helsinki) found higher cell densities on donor corneas than on host corneas, suggesting that the host endothelial cells do not slide in to help heal the donor.

B. Holden (Kensington, Australia) and A. Vannas (Helsinki) described small black endothelial blebs that arose within a few minutes after the induction of epithelial surface hypoxia by a soft contact lens, eyelid closure, or nitrogen. These were unassociated with increased corneal thickness, varied in severity for different individuals, were unassociated with endothelial cell loss, and disappeared within minutes of normal oxygen restoration. These may correspond to the swollen mitochondria seen on transmission electron microscopy. J. Krachmer (Iowa City) observed another type of bleb that lasted for days in a variety of ocular disorders and demonstrated in a rabbit model that they corresponded to transient endothelial cell swelling.

In posterior polymorphous dystrophy, F. Polack (Gainesville) found focal

crater-like defects in the endothelium by scanning electron microscopy that correspond to the vesicular lesions seen clinically. In Fuchs' dystrophy, F. Weebers (Liege) observed increased numbers of nuclei around guttate excrescences in flat mounts. R. Laing (Boston) described the development of cornea guttata in Fuchs' dystrophy with their progressive disruption of the endothelial mosaic. In herpes zoster ophthalmicus, R. Sundmacher (Freiburg) distinguished between keratic precipitates that showed up as white spots with minimal endothelial disruption and direct endothelial involvement with its bleb-like pattern.

C. Roberts (New York) demonstrated streaming cells in keratoconus, focally absent cells in posterior polymorphous dystrophy, and sharp borders of keratic precipitates as opposed to blurred borders of cornea guttata. R. Buckley (London) demonstrated that Chandler's syndrome has profusely abnormal cells; posterior polymorphous dystrophy shows focally abnormal cells; keratic precipitates and exfoliative capsular material appear as white spots that overlap cell borders; and argyrosis appears as white sliver-shaped reflections. D. Maurice (Palo Alto) was unable to correlate his observations on specular photomicrographs with those from scanning electron microscopy.

R. Laing (Boston) presented a schematic classification of morphologic findings on specular photomicrographs but emphasized that we do not know the anatomic correlates of most of the dark spots, light spots, and cell boundary variations.

Summary—Worldwide interest in clinical specular microscopy of the corneal endothelium has produced new instruments that provide wider fields of view and better resolution in both the contact and noncontact modes. Future instrument designs may include a scanning slit to produce an even wider field, and on-line televised recording for instant replay and later computerized image-processing. Advances in quantitation of the endothelium have shown that cell densities do not correlate directly with age, and that with advancing age, some cells maintain a constant small size, while others become progressively larger. Investigators now seek the correlation for the various dark and light morphologic features of endothelial cells and recognizable patterns for known clinical entities. Current clinical applications include determination of the amount of endothelial damage following surgery, evaluation of donor corneal tissue, assessment of the toxic effects of medications and devices like soft contact lenses, and diagnosis of subtle endothelial disease. The major clinical challenge is to correlate the morphologic findings seen on the specular photomicrograph with endothelial function. Since no direct correlation between endothelial function and density has yet been found, merely measuring and counting endothelial cells has limited value. Investigators are now trying to measure endothelial function in the living cornea. Once the correlation between endothelial structure and function is obtained, clinicians will have a better basis for predicting the cornea's response to surgery and disease.

GEORGE O. WARING III

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THE XXIV INTERNATIONAL CONGRESS OF OPHTHALMOLOGY—UPDATE

The United States first hosted an International Congress of Ophthalmology September 12–14, 1876, as part of the national centennial celebration. Its President was Elkanah Williams, Professor of Ophthalmology at Miami, Ohio (now Cincinnati) School of Medicine. Of the 106 who attended the meeting, only nine were from abroad. (Williams was later named along with Helmholtz, Donders, and Stokes as an honorary member of the Ophthalmological Society of the United Kingdom, when it was founded by William Bowman in 1880.) The most noteworthy event of the meeting was the international acceptance of the diopter as a unit of measurement of all spherical and cylindrical lenses.

A meeting called An International Congress of Ophthalmology was held April 25–28, 1922, in Washington, D.C. This congress was not entirely international, inasmuch as the antagonisms arising from World War I still persisted. It was only after the signing of the Locarno Peace Pact in the autumn of 1925 that an International Council could be formed to decide on the time and location of the next congress.

The XVII Congress in 1954 held its opening ceremony in Montreal and its closing ceremony in New York. Some 70 countries were represented by 2,164 delegates and 662 associate members. This was an exciting meeting in which Meyer-Schwickerath described his results with photocoagulation. A panel implicated oxygen therapy in the premature infant as a cause of retrolental fibroplasia, and Norman Ashton referred to David and Goliath as he took on the prevailing views of Alan Woods concerning the origin of uveitis.

In 1982, the International Congress returns to the United States to meet simultaneously with the American Academy of Ophthalmology from October 31 to

November 5 in San Francisco. A. Edward Maumenee is President, Frank W. Newell and Bradley R. Straatsma are Vice Presidents, and Bruce Spivey is the Secretary-General. Plans for this XXIV International Congress began in 1975. The International Council of Ophthalmology awarded the meeting to the United States when it met in Tokyo in 1978.

The main themes of the Congress are advances and diagnostic techniques in genetics, with the discussions to be led by Harold Henkes, The Netherlands; Franz Fankhauser, Switzerland; Jules Francois, Belgium; Irene Maumenee, United States; Karl Ossoinig, United States; and advances in surgical techniques, the discussions to be led by Robert Machemer, United States; Jose Barraquer, Colombia; Jacques Charleux, France; and Saiichi Mishima, Japan. Abstracts of scientific papers and films must be received by Dec. 15, 1981. Forms for submission of abstracts are available from Frederick C. Blodi, M.D., International Congress of Ophthalmology Box 3030, San Francisco, CA 94119, U.S.A.

A number of satellite meetings have been arranged by Bradley R. Straatsma; these will meet before and after the Congress. The opening session will be held on Sunday, Oct. 31, 1982, with the award of the Gonin medal. There will be five simultaneous scientific sessions and 36 instruction courses held at the Moscone Convention Center and adjacent hotels and Merchandise Mart complex. The San Francisco Opera and the San Francisco Symphony will be performing at the time of the meeting. The official languages of the Congress will be English, French, Japanese, and Spanish.

Further information concerning the International Congress and its joint meeting with the Academy is available from Phillip Rollins, M.D., P.O. Box 3030, San Francisco, CA 94119.

FRANK W. NEWELL

CORRESPONDENCE

Letters to the Editor must be typed double-spaced on 8½ × 11-inch bond paper, with 1½-inch margins on all four sides, and limited in length to two manuscript pages.

Cecil Textbook of Medicine

Editor:

As the editor of the "Cecil Textbook of Medicine," I must comment on your review (Am. J. Ophthalmol. 90:119, 1980). It is an interesting review and makes a good point that textbooks of medicine should continue to show a high level of ophthalmic awareness. We hope we have achieved some measure of success in that venture by inclusion of the section on Diseases of the Eye, by Maurice Landers.

I was momentarily jolted by your statement that Horner's syndrome is not mentioned in connection with pulmonary lesions, or cotton-wool patches in the chapter on lupus erythematosus. The reportedly absent references do occur. Admittedly, the descriptions could be more extensive, but I was happy to note that the references were there after all.

JAMES B. WYNGAARDEN, M.D.
Durham, North Carolina

Mea maxima culpa—The Editor

Radiating Lights and Radial Keratotomy

Editor:

Radial keratotomy has been widely publicized in the lay press[1,2] and courses teaching the surgical techniques are readily available. The few scientific studies published[3,4] indicate some subjective visual complications such as photophobia,

fluctuating vision, and glare,[5] in addition to possible induced hyperopia, residual myopia, and astigmatism.

I recently examined an asymptomatic patient with a visual acuity of 6/6+ (20/20+) three weeks after he underwent radial keratotomy in one eye. On monocularly viewing a pen light in a minimally darkened room, the patient reported seeing streaks of light around the pen light with the surgically treated eye and none with the fellow eye. He counted eight lines radiating around the light source. This simple light test provides objective information (which the patient can document with a drawing) about what may be a manifestation of the light-scattering properties of the induced linear corneal scars.

We have yet to evaluate the significance of the radiating streaks of light after monocular and binocular radial keratotomy in relation to ordinary activities such as driving or flying at night.

STEVEN L. LEMPERT, M.D.
Pittsburgh, Pennsylvania

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Zeiss Gonioscopy Lens for Noncontact Biomicroscopic Ophthalmoscopy

Editor:

Many patients should undergo stereoscopic biomicroscopy of the posterior pole region but not gonioscopy. One can use a contact lens of the Zeiss or Goldmann type, or a noncontact Hruby-type lens. The contact lenses require topically applied anesthesia; the Goldmann lens also requires an exogenous coupling medium. The Hruby lenses available on the Haag-Streit and Zeiss slit lamps offer very limited flexibility in lens position and movement relative to the patient's eye and the biomicroscopic observation system. The hand-held clamp-mounted Zeiss four-mirror gonioscopy lens allows rapid, flexible, dynamic noncontact stereobiomicroscopic visualization of the posterior fundus and, in our opinion, in most situations eliminates the need for a separate slit-lamp-mounted Hruby lens system.

The central portion of the Zeiss gonioscopy lens, like the Hruby, is a high-minus lens, and thus permits biomicroscopic observation of the fundus whether or not the lens touches the cornea. The great positional flexibility of the hand-held Zeiss lens allows the various reflections from the lens of the slit-lamp illumi-

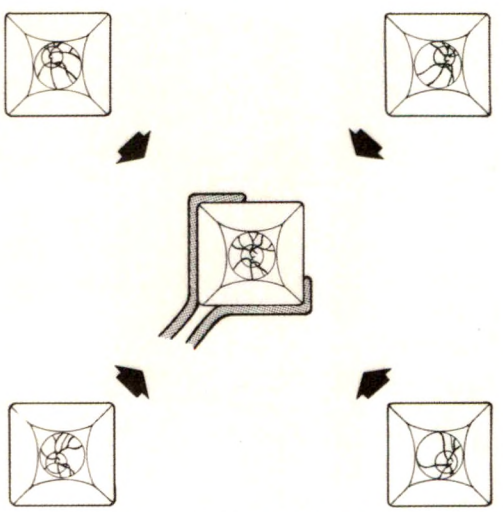


Figure (Kaufman, Bundy, and Neider). Prismatic effect of lens/eye optics centers object of regard. Arrows indicate direction of lens movement required to bring the optic disk from periphery to center of field.

nation system to be easily displaced away from the visual line, either by slight movement along the X–Y axis, or by tilting. This positional flexibility also permits the use of the prismatic effect of the lens to bring areas of the fundus outside the direct visual line into the field of view without altering the patient's eye position (Figure). The angle between the observation line and the fundus plane can be altered by tilting the lens, creating different viewing perspectives of specific fundus details. These manipulations are much more easily and rapidly performed with the hand-held Zeiss lens than with the slit-lamp-mounted Zeiss or Haag-Streit-type Hruby lens systems.

PAUL L. KAUFMAN, M.D.
WALTER E. BUNDY III, M.D.
MICHAEL W. NEIDER, B.A.
Madison, Wisconsin

Photohazards of Intraocular Lenses

Editor:

In his letter "Photohazards of intraocular lens impants in aphakia" (Am. J. Ophthalmol. 90:114, 1980), S. Zigman points out that polymethylmethacrylate does not absorb ultraviolet light. He therefore concludes that intraocular lenses pose a special hazard to aphakic eyes by failing to protect the retina.

Dr. Zigman failed to recognize that aphakic contact lenses do not absorb ultraviolet light either. Even tinted, hard contact lenses do not absorb ultraviolet in the wavelengths present in sunshine at ordinary altitudes. Aphakic glasses are no better unless they are made of special materials (such as UV 400 lenses), or are specially coated.

I agree that we should consider retinal protection in aphakic patients. The percentage of patients wearing truly protective glasses is very small at the present time (and there is no study to show that these patients do better in the long run than patients with ordinary aphakic corrections). With this exception, the statement that "these other devices are safer and now offer better retinal protection against radiant energy" is not true. A polymethylmethacrylate contact lens, for example, offers no more retinal protection than a polymethylmethacrylate intraocular lens.

ROBERT C. DREWS, M.D.
St. Louis, Missouri

Reply

Editor:

I welcome Dr. Drews' comments because they reemphasize my concern about altered eye/light interactions in humans with aphakia and the special visual aids they need because of this condition. Dr. Drews seems to agree that there may be additional hazards from the transmission of short-wavelength light to the posterior of the aphakic eye, and that polymethylmethacrylate intraocular lenses do allow this light to reach the retina quite readily, as do many other visual aids. No doubt, the filtering or lack of it by polymethylmethacrylate is the same, whether used for contact lenses or intraocular lenses, provided that the same raw materials are used to manufacture them and that the monomer and polymer in the different products are purified to the same degree.

I have found that many contact lenses and spectacle formulations and coatings do stop nearly all light with wavelengths shorter than 400 nm from entering the eye (and they also diminish chromatic abberation). Amber-tinted lenses are good absorbers of blue and ultraviolet light. Selected glass and plastic cataract spectacle lenses exhibited great variability in the absorption of light with wavelengths shorter than 400 nm. Some of

these lenses are fine absorbers of such light, but this is not by design. Guidelines are needed, based on this information, in order to provide proper light filtration for aphakic eyes.

Dr. Drews' comments that there are as yet no strong clinical findings indicating that the additional quantity of short-wavelength light reaching the retinas of aphakic human eyes has damaged them. This is a good point, but studies in humans to test this hypothesis would be difficult to perform safely. Nonetheless, current laser standards are based on experimental studies, and it would be unwise to ignore the data from animal studies that support the potential ocular hazards of short-wavelength light.

Our exchange of letters thus emphasizes strongly the need for careful laboratory investigation of the potential vitreoretinal hazards of short-wavelength light to individuals with aphakia, and also the importance of effective lenses to prevent them.

SEYMOUR ZIGMAN, PH.D.
Rochester, New York

Tissue Adhesives for Stromal Melting

Editor:

I read with great interest the article, "Tissue adhesive arrests stromal melting in the human cornea," by J. A. Fogle, K. R. Kenyon, and C. S. Foster (Am. J. Ophthalmol. 89:795, 1980).

Dr. Kenyon's original papers on the rabbit prompted me to start using cyanoacrylate adhesives in the treatment of collagenase ulcers in the dog. The brachycephalic, or short-nosed, breeds have a much higher incidence of collagenase ulcers than the longer-nosed breeds. It would be interesting to know the cause. Perhaps the amount of tearing in the short-nosed breeds with more exposed eyes is greater, causing more ulcers. I have used this adhesive for three or four years and have found that it effectively seals a melting descemetocele without the use of contact lenses. I have no experience whatsoever in the use of these lenses in combination with the adhesives. The adhesive alone seems to be very effective.

I place a drop or so on the descemetocele and re-examine the patient within 48 to 72 hours and re-apply the adhesive if necessary.

I thought you would be interested to know that veterinary ophthalmologists use the adhesives and have been very pleased with the results.

SETH A. KOCH, V.M.D.
Alexandria, Virginia

Radial Keratotomy

Editor:

In the last year, radial keratotomy has received widespread attention as a treatment for myopia. Studies to evaluate the benefits and risks of this surgical procedure in animals and humans are underway, and new research grant applications have been submitted to the National Eye Institute (NEI). These studies are needed to determine how effective radial keratotomy is in correcting myopia and to evaluate the safety of the procedure, including its short- and long-term effects.

The National Advisory Eye Council is the principal advisory group to the NEI. In order to discharge its responsibilities to the American public and to the scientific and health care community, the Council needs as much information as possible about the safety of radial keratotomy in humans. Consequently, we are urging all ophthalmologists to share whatever information they may have about eye problems that have resulted from this surgical procedure.

We would like to know if operative or postoperative complications, such as ocu-

lar rupture perforation, have occurred in individuals who have undergone radial keratotomy. If your patients have experienced, or if you have been consulted about, such problems, please write the Council immediately in care of its Executive Secretary, Ronald G. Geller, Ph.D., National Eye Institute, Building 31, Room 6A04, National Institutes of Health, Bethesda, MD 20205.

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BOOK REVIEWS

Complications in Ophthalmic Surgery. Edited by Stephen R. Waltman and Theodore Krupin. Philadelphia, J. B. Lippincott, 1980. Hardcover, 368 pages, index, 103 black and white figures. $37

This book contains 15 chapters by 16 authors, all associated with the department of ophthalmology at Washington University in St. Louis. The first two chapters deal with anesthesia and postoperative infection. The chapter on anesthesia discusses inhalation agents, intravenous agents, and local anesthetics in patients with a variety of ocular and systemic problems. The chapter on postoperative infection covers sources, characteristics, and management of infections. The next three chapters deal with cataracts: intracapsular and extracapsular extraction, intraocular lens implantation, and phacoemulsification. These are followed by chapters on keratoplasty, glaucoma, extraocular muscles, plastic surgery, lacrimal surgery, orbital surgery, retinal detachment, vitrectomy, photocoagulation and cryotherapy, and, finally, ocular trauma.

The book discusses more than complications in ophthalmic surgery. Each chapter contains a careful discussion of surgical or anesthetic technique. Although surgical technique is frequently a controversial subject, the methods described in the book are all respected procedures. In each case the discussion of complications is thorough and suggested methods for management are as up-to-date as possible in a book.

Many of the 103 illustrations are line drawings. Each chapter is followed by references and there is a good index. The variations in style usually encountered in a book with numerous authors is not troublesome.

This book should prove valuable to all residents and most practicing ophthalmologists. It brings together a wealth of techniques and strategies which are essential in the modern practice of ophthalmic surgery. I highly recommend this book to all ophthalmologists practicing surgery.

KARL J. FRITZ

Ocular Therapeutics. Edited by B. Dobli Srinivasan. New York, Masson Publishing USA, Inc., 1980. Hardcover, 231 pages, index, 67 black and white figures. $38.50

This text, written by several physicians, describes the clinical management of a variety of ocular disorders. It contains excellent presentations on herpes simplex, *Chlamydia* organisms, fungi, toxoplasmosis, toxocariasis, and bacterial

endophthalmitis. It includes a general review of corticosteroids and the diagnostic and therapeutic use of drugs in strabismus. Several good sections deal with toxic drug responses of the retina and optic nerve. One covers the treatment of glaucoma with echothiophate, timolol, and dipivalyl epinephrine. Additionally, there are a number of relatively brief reviews of other subjects.

The book is not sufficiently comprehensive to serve as a reference text on ocular therapy, but the subjects listed above are clearly and effectively reviewed. Perhaps future editions will be expanded to offer more complete coverage of available drugs and their use in ophthalmic therapy.

WILLIAM H. HAVENER

Microsurgery of Retinal Detachment. By Mireille Bonnet. Translated by Alexander J. Brucker. New York, Masson Publishing USA, Inc., 1980. Hardcover, 79 pages, references, index, 216 black and white figures. $24.50

This short book describing vitreoretinal surgery using the surgical microscope is divided into two major sections—instrumentation and surgical technique. The first section briefly catalogues and illustrates a number of available instruments as well as those preferred by the author.

The second section, illustrated in great detail, describes Bonnet's surgical techniques. The sequence of surgical steps is somewhat disrupted by a division into surgical "stages," some of which require simultaneous fundus observation and some of which do not. The topics covered are broad and some are treated too cursorily.

In other places the discussion is based upon a review of published reports rather than on Bonnet's own experience. This may be misleading; for example, the reader may assume that a carbon dioxide laser is more available for coagulating blood vessels in retinal fibrovascular tissue than it actually is.

The Goldmann three-mirror contact lens and slit-lamp examination of the fundus should be used before surgery for retinal detachments, but they are not necessary for every patient. The Goldmann contact lens examination has long been used as an alternative to indirect ophthalmoscopy. In patients with questionable retinal lesions, a Goldmann three-mirror contact lens examination, with or without scleral indentation, may be used preoperatively to evaluate the importance of the lesion.

Some of Bonnet's arguments with respect to the advantages of Goldmann contact lens surgery versus surgery using the indirect ophthalmoscope are debatable, however. One must carefully select the patients who will really benefit. For vitreous surgery the operating microscope is essential, whereas in most cases of retinal detachment surgery it is unnecessary. One great advantage of indirect ophthalmoscopy during surgery for retinal detachment is the panoramic view of the fundus that it provides. The use of methylcellulose 2% or other contact lens solutions, however, makes indirect ophthalmoscopy difficult at times, even after copious irrigation of the cornea.

In summary, this book provides an alternative approach to surgery for retinal detachment in some cases.

PETER H. MORSE

Diabetic Retinopathy. Sights and Sounds in Ophthalmology. Vol. 4. By Stuart L. Fine and Arnall Patz. St. Louis, C. V. Mosby Co., 1980. Hardcover, 61 pages, bibliography, index, three cassette tapes, 100 color slides. $175

This volume contains a comprehensive presentation of the Wilmer Retinal Vascu-

lar Service's clinical approach to diabetic retinopathy. The slides are generally well done and the entire set of 100 will be of considerable value to residents and practicing general ophthalmologists who seek an extensive, clinically oriented review of the current management of diabetic retinopathy. Those who devote much of their time to the care of patients with diabetic retinopathy will also find much of interest in this presentation, particularly the discussion of retreatment by photocoagulation of eyes with incomplete regression of new vessels after initial treatment and the section on other retinal vascular disorders. The case of systemic lupus erythematosus shown in slide No. 89 is particularly fascinating.

MATTHEW D. DAVIS

Lentes de Contacto. By E. Gil del Rio and P. Baronet. Barcelona, Editorial JIMS, 1980. Hardcover, 515 pages, index, 46 tables, 223 black and white figures, 67 color figures.

This book on contact lenses is beautifully made; the print is clear and well organized and the color plates are excellent. According to the authors, the purpose of the book was not only to inform readers about contact lenses but to emphasize corneal physiology and pathology related to contact lenses. Of the 28 chapters, six concern the anatomy and physiology of the eye. Nine chapters are dedicated to clinical procedures, including special methods of examining the cornea before contact lens adaptation.

The authors also describe the different materials used for the preparation of contact lenses, the prescription of the various types of contact lenses, and the methods used to design and adapt different types of lenses. Because of the importance of soft contact lenses today, they discuss the properties and characteristics of different types of soft contact lenses. These include the different complications caused by this type of lens and the alterations in the structure of the soft contact lenses seen in clinical practice. There is also a chapter on the use of permanent or extended-wear contact lenses; however, the authors' experience with this type of lens seems to be limited. They do not describe the cases where these types of lenses are indicated or the complications seen with the extended-wear contact lens. The chapters dealing with the anatomy and physiology of the eye, particularly of the cornea, are excellent and the authors have done a very good job of reviewing recently published data.

The chapters are profusely illustrated with diagrams and drawings that facilitate the assimilation of the material. This system and a good index help the reader locate a particular subject. The book has been based primarily on the experience of the authors and the references are limited. Unfortunately, the references in the text use only the date of publication to identify the source. The book lacks a list of references or bibliography.

Overall, this is a very good book. It presents the practical experience of the authors who have a conservative approach to this specialty. The book is recommended to all students of the art of contact-lens fitting.

FRANK M. POLACK

Surgery of Ptosis. By Sidney A. Fox. Baltimore, Williams & Wilkins Co., 1980. Hardcover, index, 119 black and white figures. $29.95

This well-written monograph discusses the history, theory, and practice of blepharoptosis surgery. The first chapter gives a brief history of the various techniques used throughout the years to correct blepharoptosis. In the following chapters he classifies types of blepharoptosis and

describes his examination techniques and reasons for choosing a particular operation. One important point is the author's belief that the surgical procedure should be based upon the action of the levator palpebrae superioris muscle and not on the amount of measured blepharoptosis. The arguments are valid and should produce excellent results. The techniques of levator palpebrae superioris muscle resection, both anterior and posterior approaches, are well illustrated and described in detail.

This is followed by a chapter on the Fasanella-Servat procedure and levator palpebrae superioris muscle aponeurosis tucking. The chapter on blepharoptosis with levator palpebrae superioris muscle action of less than 2 mm includes the frontalis sling procedure as well as older procedures such as the skin sling, the orbicularis oculi muscle sling, and the superior rectus muscle procedures. The portion of this chapter dealing with frontalis slings is well done. The discussion of the older techniques, however, particularly superior rectus muscle surgery should have been in the chapter on the history of blepharoptosis treatment rather than in the chapters dealing with surgical technique.

The chapter on complications of blepharoptosis surgery is very inclusive. Each complication is followed by a comment on how to avoid the complication and how to treat it if it does occur. The next two chapters deal with complicated blepharoptosis and pseudoblepharoptosis and present interesting procedures for treating these conditions.

The author discusses anatomy, physiology, and pathology at the end of his book rather than at the beginning. He does this to emphasize that physicians go too quickly to the surgical portion of a textbook, and completely skip over the portion on anatomy. If his unorthodox positioning of these chapters emphasizes the importance of anatomy, physiology, and pathology, this maneuver will have served its purpose.

One interesting point is Dr. Fox's disbelief in the theory that dehiscence of the levator palpebrae superioris muscle is a major cause of senile blepharoptosis. Since he holds this view, he considers aponeurosis repair of the levator palpebrae superioris muscle an unimportant procedure. Many surgeons have observed dehiscence, disinsertion, or degeneration of the levator palpebrae superioris muscle in the senile patient and have successfully cured blepharoptosis by aponeurosis repair. This procedure differs from Everbusch's tucking operation which in effect shortens the levator palpebrae superioris muscle without surgically excising it. The more modern use of the term tucking procedure refers to the repair of the dehiscence or disinsertion of the aponeurosis and therefore is not a direct descendant or a copy of Everbusch's procedure as the author contends. These are minor objections and should in no way discourage one from reading this excellent textbook.

This is a fine book, one that should be read by all residents in ophthalmology before performing blepharoptosis surgery. It should also be in the library of the surgeon who occasionally performs blepharoptosis operations because it is a concise, well-written review of the subject; it can help the surgeon to decide what procedure is appropriate and also gives a step-by-step description of how to perform the surgery.

FREDERICK A. MAUSOLF

ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

Acta Ophthalmologica

RESULTS OF PHOTOCOAGULATION IN DIABETIC RETINOPATHY AFTER LONG-TERM FOLLOW-UP. Zetterström, B. (Dept. Ophthalmol., Huddinge Univ. Hosp., Karolinska Inst., Stockholm, Sweden). Acta Ophthalmol. 58:361, 1980.

Seventy-three diabetics with retinopathy, in whom one eye was treated by photocoagulation, and the other was left untreated, were followed-up for nine to 11 years. Visual acuity, visual field, dark-adaptation curves, and intraocular pressure were recorded; biomicroscopy and photography of the fundus were carried out. Severe visual loss (<0.1) was noted in 4% of the treated and in 25% of the untreated eyes. Minor, subjectively unnoticed, scotomas were found at visual-field examination in 18 of 40 treated eyes. Dark-adaptation curves showed abnormal values both in the treated and in the untreated eyes, without any recordable difference. No contra-inducing long-term complications of the photocoagulation treatment could be demonstrated. (3 figures, 1 table, 8 references)—Author's abstract

Albrecht von Graefes Archiv für Klinische und Experimentelle Ophthalmologie

ANTERIOR PARS PLANA VITRECTOMY IN CILIARY AND IRIS BLOCK GLAUCOMA. Koerner, F. H. (Dept. Ophthalmol., Inselspital, Univ. Berne, Switzerland). Albrecht von Graefes Arch. Klin. Exp. Ophthalmol. 214:119, 1980.

Ciliary and iris block glaucoma in aphakic and phakic eyes is characterized by the accumulation of aqueous humor in the vitreous cavity causing an increase in vitreous volume, a flattening of the anterior chamber, and an elevation of intraocular pressure. As vitreous blocks the space between the ciliary body and the iris or lens, respectively, an anterior vitrectomy via the pars plana is the logical surgical approach. This operation was performed in one phakic and 15 aphakic eyes with so-called pupillary block. The anterior chamber retained a normal depth in each case. Postoperatively the vitreous was found to be completely removed from the posterior chamber and the epiciliary space, rendering a free communication between the ciliary body and the posterior and anterior chamber. Early surgery in aphakic eyes with a flat anterior chamber resulted in a prompt normalization of intraocular pressure, whereas eyes with a block duration of over four weeks were at high risk of developing a secondary outflow resistance probably due to chronic angle closure. (4 figures, 3 tables, 16 references)—Authors' abstract

A MODEL TO MEASURE THE VOLUME OF CHOROIDAL MELANOMAS. Guthoff, R. (Universitäts-Augenklinik Hamburg-Eppendorf, Martinistr. 52, D-2000 Hamburg 20, Bunderepublik Deutschland). Albrecht von Graefes Arch. Klin. Exp. Ophthalmol. 214:139, 1980.

The author used a contact B-scan and a grid to measure both the diameter and the height of melanomas of the choroid. He devised a formula into which he inserted the values obtained; this gave him the total volume of the tumor. The axial length of the eye and the refractive error have no influence on the final results. (6 figures, 1 table, 9 references)—David Shoch

Archives of Ophthalmology

MAYO CLINIC EXPERIENCE WITH CONJUNCTIVAL MELANOMAS. Liesegang, T. J., and Campbell, R. J. (Depts. Ophthalmol., and Surg. Pathol., Mayo Clinic, Rochester, Minn.). Arch. Ophthalmol. 98:1385, 1980.

One hundred twenty-six surgically excised pigmented lesions of the conjunctiva were evaluated and correlated with clinical data and follow-up information. Of these, 42 were classified as conjunctival melanomas, with 18 arising apparently de novo, 15 from acquired melanosis, and nine from nevi. The six tumor

deaths occurred only in patients with melanomas arising de novo or from acquired melanosis. Patients with tumor deaths usually demonstrated local recurrences long before metastases occurred. Moreover, several patients had local recurrences treated repeatedly for years without metastatic spread. (3 figures, 5 tables, 13 references)—Authors' abstract

COMBINED SURGERY AND CRYOTHERAPY FOR DIFFUSE MALIGNANT MELANOMA OF THE CONJUNCTIVA. Jakobiec, F. A., Brownstein, S., Wilkinson, R. D., Khalil, M., Cooper, W. C., and Shibata, H. R. (Algernon B. Reese Lab. of Edward S. Harkness Eye Inst., New York, N.Y.). Arch. Ophthalmol. 98:1390, 1980.

Two patients who had refused exenteration for widespread conjunctival melanoma were treated by a combination of cryotherapy and surgical excisions. Cryotherapy was delivered only to the extensive areas of flat intraepithelial melanocytic proliferation (precancerous melanosis), while the surgical excisions were performed on the focal nodules, representing localized invasive melanoma. In these two patients, the invasive nodules measured, respectively, 1.2 and 1.5 mm in greatest thickness, placing them in a low to borderline risk group for metastasis. Repeated cryoapplications were required to control the widespread flat intraepithelial disease. The conjunctiva tolerated these procedures well because the substantia propria is not sacrificed as it must be in surgical conjunctivectomy, allowing comparatively normal reepithelialization to occur from adjacent zones, after the treated epithelium containing the melanocytes sloughs. No evidence of invasive melanoma (cancerous melanosis) has developed in any of the cryotreated areas of intraepithelial disease (precancerous melanosis); sequential biopsies have established that the atypical melanocytes have disappeared from the epithelium. (24 figures, 15 references)—Authors' abstract

CHLORAMPHENICOL OPTIC NEUROPATHY. Godel, V., Nemet, P., and Lazar, M. (Dept. Ophthalmol., Ichilov Hosp., Tel Aviv Univ. Med. School, Tel Aviv, Israel). Arch. Ophthalmol. 98:1417, 1980.

Three patients with cystic fibrosis of the pancreas initially were seen because of optic neuropathy caused by a toxic reaction to chloramphenicol. The ocular symptoms were characterized by loss of visual acuity, central scotomas, red-green dyschromatopsia, and fundus changes. An increase in the latency of the occipital evoked potentials paralleled the visual impairment. A transient photopic decline in the red-light-elicited electroretinogram and the occurrence of Uhthoff's sign in the acute stage of the disease were observed. All visual impairment recovered partially or totally after the withdrawal of therapy with the drug. (9 figures, 16 references)—Authors' abstract

British Journal of Ophthalmology

THE ENVIRONMENT AND THE LENS. Taylor, H. R. (International Ctr. for Epidemiologic and Preventive Ophthalmol., Wilmer Inst., Johns Hopkins Hosp., Baltimore, Md.). Br. J. Ophthalmol. 64:303, 1980.

The association of various personal, occupational, and environmental factors with cataracts and pseudoexfoliation of the lens was examined in a group of Australian Aborigines. Cataracts occurred more commonly in the elderly but were also associated with high levels of ultraviolet radiation and sunlight and were more commonly seen in lower latitudes. Pseudoexfoliation was also seen more commonly in the elderly, especially among males. Pseudoexfoliation occurred particularly commonly in stockmen and was related to latitude and global radiation. It was positively associated with the occurrence of cataract and with climatic droplet keratopathy. It is suggested that senile cataracts are related to the amount of ultraviolet radiation and pseudoexfoliation to global radiation. (2 figures, 10 tables, 55 references)—Author's abstract

A NOTE ON A POSSIBLE RELATION BETWEEN REFRACTION AND A DISPOSITION FOR SENILE NUCLEAR CATARACT. Weale, R. (Dept. Vis. Sci., Inst. Ophthalmol., London). Br. J. Ophthalmol. 64:311, 1980.

A study was made of records of patients who have had at least one senile cataract removed and for whom refraction records were available. The risk of a cataract occurring was determined as a function of precataractous refraction. Emmetropia was found to provide a sharp borderline between the low risk (about 1:1000) of hypermetropes and an increasing risk (about 1:100) for moderate myopes for patients in their early 60s. (4 figures, 9 references)—Author's abstract

LONG-TERM FOLLOW-UP STUDY ON PREVENTION OF APHAKIC CYSTOID MACULAR OEDEMA BY TOPICAL INDOMETHACIN. Miyake, K., Sakamura, S., and Miura, H. (Shozankai Med. Foundation, Miyake Eye Clin. Hosp., Nagoya, Japan). Br. J. Ophthalmol. 64:324, 1980.

In addition to routine medications topical indomethacin was applied to one group of 112 eyes and an appropriate placebo was applied to another group of 106 eyes that underwent intracapsular cataract extraction. The investigation was conducted in a prospective double-blind fashion over a long period of time in order to demonstrate the effects of indomethacin in preventing the development of cystoid macular edema. Indomethacin drops were instilled once a day before surgery, twice on the day of surgery, and three times daily for two weeks postoperatively. The prevalence of cystoid macular edema as detected by fluorescein angiograms was as follows: in the early period (from one to two postoperative months), cystoid macular edema developed in 25% of the indomethacin-treated eyes and in 51% of the untreated eyes; in the middle period (from four to seven postoperative months), it developed in 11% of the former group and 21% in the latter group; in the late period (from one to one and a half postoperative years) it developed in 9% of the former group and 11% of the latter group. In both the early and middle periods the difference in prevalence was statistically significant, but in the late period there was no significant difference between the two groups. The onset of "indomethacin-masked cystoid macular edema" in several treated eyes may account for the insignificant difference in the late period. Prolonged biosynthesis of indomethacin-sensitive materials, including prostaglandins, may be suspected in some aphakic eyes. (3 figures, 3 tables, 17 references)—Authors' abstract

THE OCULOCARDIAC REFLEX AS A SURGICAL AID IN IDENTIFYING A SLIPPED OR "LOST" EXTRAOCULAR MUSCLE. Apt, L., and Isenberg, S. J. (Dept. Ophthalmol., Jules Stein Eye Inst., Univ. California, Los Angeles, Calif.). Br. J. Ophthalmol. 64:362, 1980.

The oculocardiac reflex during strabismus surgery has generally been regarded as a hazard capable of causing death. Six cases are presented which show a beneficial use of the oculocardiac reflex. Isolation of a previously slipped or "lost" extraocular muscle can be difficult. In this series identification of the tissue as muscle was substantiated by observing a positive oculocardiac reflex when traction was placed on the suspected tissue. Each of the six dislodged extraocular muscles was the medial rectus muscle. Three of the muscles had been resected and three either recessed or tenotomised. In one patient, despite six previous strabismus operations, including two strabotomies on a muscle that slipped, and in another patient, who had a lapse of six years since the last strabotomy, when the slipped muscle was isolated, the oculocardiac reflex could still be elicited. To avoid abolishing the oculocardiac reflex during surgery the anesthetist should be instructed to avoid the use of an intravenous parasympatholytic agent, such as atropine, at the time of induction and during the operation. (7 references)—Authors' abstract

Journal of the American Medical Association

ADVERSE EFFECTS OF CONTACT LENS WEAR DURING DECOMPRESSION. Simon, D. R., and Bradley, M. E. (Naval Medical Res. Inst., Bethesda, Md.). J.A.M.A. 244:1213, 1980.

Two divers wore three types of lenses during and after exposures in a hyperbaric chamber to depths equivalent to 45.5 M of seawater or altitudes of 11,277 M. With hard contact lenses there was a definite accumulation of air bubbles beneath the lenses and associated opacities in the cornea, apparently

from inability to disperse the nitrogen that accumulated under the contact lens. These problems did not occur if there was a single hole in the center of the lens through which the air could escape or if membrane lenses were worn. Hard unfenestrated contact lenses should not be worn either by deep-sea divers or in high-altitude flying. (2 figures, 2 references)—David Shoch

Journal of the Royal Society of Medicine

REGIONAL VARIATION IN CATARACT EXTRACTION RATES AND THEIR RELATIONSHIP WITH RESOURCE SUPPLY AND NEED. Sanderson, H. F. (Dept. Community Health, London School of Hygiene & Tropical Med., London). J. Royal Soc. Med. 73:492, 1980.

There are large variations in cataract extraction rates among different regions of England. It is obvious that one of the characteristics that determines the operation rate is the distribution of the aged in the population. However, even when the statistics are corrected by an age standardization method, there is still a high degree of difference in different areas in the country. The author tried to correlate this difference with various factors and found that the only real correlation appeared to be with the availability of ophthalmic beds. However, one cannot conclude from this that the bed supply determines the operation rate since in fact it may be the reverse in that the bed supply expands to meet the increasing number of surgical procedures. (3 figures, 4 tables, 14 references)—David Shoch

Klinische Monatsblaetter für Augenheilkunde

HOW BLURRED IS VISION IN MYOPIA?—AN ATTEMPT AT DEMONSTRATION BY MEANS OF PHOTOGRAPHY. (German) Kleberger, E. (Augenabteilung der Schlobpark-Klinik, Berlin). Klin. Monatsbl. Augenheilkd. 176:457, 1980.

The author took a series of photographs of eye charts and street scenes at different degrees of artificial camera myopia and at different *f* openings. He shows that in emmetropia the *f* opening makes very little difference but as the camera becomes more myopic there is an enormous difference between vision with an *f* opening of 22 and an *f* opening of 3. The pictures nicely illustrate why patients with myopia see more poorly at night when their pupils dilate. (7 figures)—David Shoch

THE EFFECTS OF TOPICAL STEROIDS IN EPIDEMIC KERATOCONJUNCTIVITIS. (German) Trauzettel-Klosinski, S., Sundmacher, R., and Wigand, R. (Univ.-Augenklinik Freiburg and Virologische Abteilung des Institutes für Hygiene und Mikrobiologie der Universität des Saarlandes). Klin. Monatsbl. Augenheilkd. 176:899, 1980.

Fifty-three patients with proven adenovirus type 8 infection were treated at random, in a prospective study, with either artificial tears or topical steroids over six weeks. Severe cases of nummular keratitis, as well as deep keratitis and slight iritis, were significantly less frequent with steroids. No difference existed with respect to the course of the conjunctivitis and the frequency of a superficial punctate keratitis. Furthermore, the overall frequency of nummular keratitis was equal in both groups, its manifestation merely being somewhat delayed in the steroid group. This may even be disadvantageous, as the natural course of the disease could be prolonged. A significant disadvantage of topical steroids, however, was the high incidence of permanent "dry eye" in the post-infection period. Therefore, as long as no effective antiviral treatment for adenovirus infection is available, steroids should only be administered to relieve symptoms in severe cases for a limited time. (6 figures, 2 tables, 20 references)—Authors' Abstract

New England Journal of Medicine

PREVENTION OF GLAUCOMA DURING HEMODIALYSIS BY MANNITOL AND ACETAZOLAMIDE. Jaeger, P., Morisod, L., Wauters, J. P., and Faggioni, R. (University Hosp., Lausanne, Switzerland). N. Engl. J. Med. 303:702, 1980.

Patients receiving long-term hemodialysis have a rather sudden drop in their serum osmolality. This rarely causes any problems in regard to intraocular pressure unless the patient has some interference with his outflow mechanism. The authors report the problems that occurred in a patient with a narrow angle whose intraocular pressure increased to 76 mm Hg. Since it was essential that he undergo dialysis, at the next session he was given mannitol intravenously which kept his intraocular pressure below 23 mm Hg. Additionally, he started taking 500 mg of acetazolamide per day. Since then he has had no further increases in pressure. (3 references)—David Shoch

ABDUCENS PALSY AFTER LUMBAR PUNCTURE. Insel, T. R., Kalin, N. H., Risch, S. C., Cohen, R. M., and Murphy, D. L. (National Inst. of Health Clin. Ctr., Bethesda, Md.). N. Engl. J. Med. 303:703, 1980.

A 60-year-old woman developed an abducens palsy after a lumbar puncture in which 30 ml of cerebrospinal fluid was withdrawn. The palsy persisted for six weeks. This problem has been reported on several occasions in the past and apparently results from a drop in spinal fluid pressure which allows a sagging of the intracranial contents with trauma to the sixth nerve. (3 references)—David Shoch

Ophthalmology

CRYOSURGERY FOR MALIGNANCIES OF THE EYELID. Fraunfelder, F. T., Zacarian, S. A., Limmer, B. L., and Wingfield, D. (Dept. Ophthalmol., Univ. Oregon, Portland, Ore.). Ophthalmol. 87:461, 1980.

The results of liquid nitrogen spray technique for 310 biopsy-proven eyelid malignancies are reviewed. The raw recurrence rate for basal cell carcinomas of the eyelids with a mean follow-up of almost three years is 4.2%, while the recurrence rate is 6% in 49 cases followed for five years. For all eyelid malignancies 10 mm or less in diameter, a 2.0% raw recurrence rate was obtained; however, in those lesions larger than 10 mm, this rose to 9.5%. With this form of therapy, basal cell carcinomas with infiltrative, nonelevated, indistinct borders had twice the recurrence rate as those tumors with elevated distinct borders. Twenty-one squamous cell carcinomas treated with cryosurgery with an average follow-up of 21.6 months have yet to show a recurrence. Complications with cryosurgery include permanent loss of eyelashes and depigmentation lasting from six months to over six years. Depending on the extent and location of the tumor, eyelid defects occurred. To date, five plastic reconstructive procedures have been required for ectropion or eyelid defects; however, no lacrimal outflow surgery has as yet been required. (8 tables, 14 references)—Authors' abstract

OBSERVATION OF SUSPECTED CHOROIDAL AND CILIARY BODY MELANOMAS FOR EVIDENCE OF GROWTH PRIOR TO ENUCLEATION. Gass, J. D. M. (Bascom Palmer Eye Inst., Univ. of Miami, Miami, Fla.). Ophthalmol. 87:523, 1980.

One hundred sixteen patients with suspected malignant melanoma of the choroid and ciliary body were observed for evidence of tumor growth prior to enucleation. All patients were followed for a minimum of five years after tumor detection. The tumor failed to grow in 69 patients, and none developed evidence of metastatic melanoma. The tumor grew in 47 patients, and 35 patients had enucleation. Five patients (four having enucleation) died of metastatic melanoma. Deaths caused by other primary malignancies (nine patients) and other causes (11 patients) outnumbered those caused by melanomas. The clinical accuracy of predicting growth of small and medium suspected melanomas was approximately 75%. Five patients died because of metastatic melanoma. This study demonstrated no evidence of growth prior to enucleation. (8 tables, 11 references)—Author's abstract

CATARACT EXTRACTION IN CASES OF CATARACT WITH VITREOUS ABNORMALITY. Machemer, R. (Duke University Eye Ctr., Durham, N.C.). Ophthalmol. 87:618, 1980.

Since good visibility of the deeper structures of the eye is a prerequisite for vitreous surgery, a lentectomy is often performed in combination with vitreous surgery even when the lens is only slightly cataractous. Clear

lenses may have to be removed when complete intraocular gas tamponade is utilized (treatment of giant tears 180 degrees and larger). A pars plana approach is recommended, and the lens is removed with the vitrectomy instruments as long as the nucleus is not too hard. Otherwise, phacoemulsifying instruments are needed. The techniques are described. The worst complication of posterior lentectomy is loss of lens material into the vitreous cavity. (5 figures, 1 table, 7 references)—Author's abstract

THE USE OF SODIUM HYALURONATE (HEALON) IN HUMAN ANTERIOR SEGMENT SURGERY. Pape, L. G., and Balazs, E. A. (Edward S. Harkness Eye Inst., Columbia Univ. New York, N.Y.). Ophthalmol. 87:699, 1980.

The safety and efficacy of sodium hyaluronate (Healon) was evaluated in a wide spectrum of anterior segment surgical procedures. Healon is not only safe, but actually facilitates the outcome of surgery. Healon placed intracamerally before cataract removal results in significantly decreased endothelial cell loss. In filtering procedures, use of intracameral and subconjunctival Healon promotes superior bleb formation while still maintaining chamber depth postoperatively. Corneal grafts performed over intracameral Healon receive maximal endothelial protection and manifest striking postoperative clarity. (6 figures, 4 references)—Authors' abstract

NEWS ITEMS

EDITED BY THOMAS CHALKLEY, M.D.

Send News Items double-spaced to News Editor, American Journal of Ophthalmology, Tribune Tower, Suite 1415, 435 N. Michigan Avenue, Chicago, Illinois 60611

For adequate publicity, notices of postgraduate courses, meetings, and lectures must be received at least four months before the date of occurrence.

RESEARCH TO PREVENT BLINDNESS: SCHOLARS AWARDS

Research to Prevent Blindness, Inc., (RPB) awarded the annual $25,000 RPB-William and Mary Greve International Research Scholars Award to Harry Quigley, assistant professor of ophthalmology at Johns Hopkins School of Medicine, Baltimore. The funds will advance his contributions to knowledge of how optic nerve damage occurs in glaucoma.

Kenneth Kenyon, assistant professor of ophthalmology at Harvard Medical School, Boston, is recipient of the $10,000 RPB-James S. Adams Research Scholars Award for his contributions to the clinical and experimental study of corneal diseases and disorders of the outer surface of the eye.

Frederick A. Jakobiec, professor of ophthalmology and pathology at Cornell University Medical College, New York City, is recipient of the $10,000 RPB-Robert E. McCormick Research Scholars Award. Dr. Jakobiec is combining computerized ultrasound techniques with scanning and electron microscopy to develop insight into the biological behavior of eye tumors.

Dominic Man-Kit Lam, associate professor of ophthalmology at the Baylor College of Medicine, Houston, Texas, has been named an RPB-Olga Keith Wiess Research Scholar. The award provides $10,000 for his ongoing research into the chemical events that transmit signals from cell to cell across the retina.

Keith Green, professor of ophthalmology at the Medical College of Georgia, is the recipient of a $7,500 RPB Manpower award to assist his studies in corneal physiology.

FOURTH INTERNATIONAL OCULOPLASTIC SOCIETY CONGRESS

The Fourth International Oculoplastic Society Congress will be held Feb. 14–21, 1981, at Margarita Island, Venezuela. The fee is $150. For further information, write Ms. P. Tamkin, c/o Dr. P. Guibor, 630 Park Ave., New York, NY 10021.

MANHATTAN EYE, EAR AND THROAT: RETINAL DETACHMENT SURGERY—STRATEGY AND TACTICS

A course in retinal detachment surgery will be sponsored by the Manhattan Eye, Ear and Throat Hospital, March 13 and 14, 1981. For further information, write James S. Schutz, M.D., Manhattan Eye, Ear and Throat Hospital, 210 E. 64th St., New York, NY 10021.

RUDOLPH ELLENDER MEDICAL FOUNDATION: 21ST ANNUAL COURSE IN CONTACT LENS FITTING

The Rudolph Ellender Medical Foundation will present its 21st annual instructional course in contact lens fitting, April 3–5, 1981, in New Orleans. For further information, write Joseph A. Baldone, M.D., Rudolph Ellender Medical Foundation, Inc., 136 S. Roman St., New Orleans, LA 70112.

FLORIDA MIDWINTER SEMINAR: PRACTICAL ADVANCES IN GENERAL OPHTHALMOLOGY

A seminar on Practical Advances in General Ophthalmology will be held Feb. 1–4, 1981, in Miami Beach, Florida. Registration fee is $225 for practitioners and $100 for residents. For further information, write Florida Midwinter Seminar, 405 N.E. 144th St., Miami, FL 33161.

UNIVERSITY OF MISSISSIPPI MEDICAL CENTER: THIRD ANNUAL COURSE ON STRABISMUS

The third annual course on strabismus will be held Dec. 12 and 13, 1980, at the University of Mississippi Medical Center in Jackson. For further information, write Dr. Raul E. Valenzuela, University of Mississippi Medical Center, 2500 N. State St., Jackson, MS 39216.

TENTH ANNUAL UNIVERSITY OF TENNESSEE RESIDENTS DAY

The Tenth Annual University of Tennessee Residents Day will be held at the University of Tennessee College of Medicine March 6, 1981. For further information, write Roger L. Hiatt, M.D., University of Tennessee College of Medicine, Dept. of Ophthalmology, 956 Court Ave., Memphis, TN 38163.

CLEVELAND CLINIC FOUNDATION: 19TH ANNUAL POSTGRADUATE SEMINAR

The Department of Ophthalmology at the Cleveland Clinic Foundation will sponsor the 19th annual

postgraduate seminar Dec. 4 and 5, 1980. For further information, write the Education Department, Cleveland Clinic Foundation, 9500 Euclid Ave., Cleveland, OH 44106.

American Academy of Facial Plastic and Reconstructive Surgery: Cosmetic Surgery of the Aging Eye

A course on cosmetic surgery of the aging eye will be held June 22 and 23, 1981, in New York City. The fee is $300. For further information, write Morris Feldstein, M.D., Page and William Black Post-Graduate School of Medicine, Mount Sinai School of Medicine, One Gustave L. Levy Place, New York, NY 10029.

Handicapped Physicians

I am a multiply-handicapped doctor interested in contacting other handicapped physicians in order to obtain some approximation of the size and characteristics of this population in the United States and Canada. There is currently no organization to provide statistical data on the career patterns of such individuals or to what extent various handicaps affect the physician's ability to remain in active practice. Should any physician become handicapped, such information would be of utmost value in determining the feasibility of remaining in any chosen field and would also provide some indication of the career opportunities available to this unique population.

All physicians, active or inactive, with any type of physical handicap are asked to write F. Zondlo, M.D., St. Paul-Ramsey Medical Education and Research Foundation, 640 Jackson St., St. Paul, MN 55101.

Personal

Arthur H. Keeney

Arthur H. Keeney has announced his retirement from the Deanship of the University of Louisville School of Medicine effective Sept. 1, 1980. The trustees of the University have announced his new designation as Dean Emeritus of the School of Medicine and Distinguished Professor of Ophthalmology.

National Retinitis Pigmentosa Foundation Grants

The National Retinitis Pigmentosa (RP) Foundation, headquartered in Baltimore, has awarded new research grants totaling $816,000 according to Alan Laties, chairman of the RP Scientific Advisory Board.

The goals of the new grants, according to Dr. Laties, are to help established scientists with outstanding credentials in the field of retinal degenerative diseases expand the scope of research. Retinitis pigmentosa and allied retinal degenerations, including Usher's syndrome and macular degeneration, affect over 400,000 persons in the United States and an estimated three million persons worldwide. There is no known treatment or cure for these disorders.

The centers receiving RP Foundation research awards this year are as follows: the Berman-Gund Laboratory for the Study of Retinal Degenerations at Harvard University, Boston ($111,542); Wilmer Eye Center, Johns Hopkins Hospital, Baltimore ($79,252); Jules Stein Institute, University of California at Los Angeles ($55,000); College of Physicians and Surgeons, Columbia University, New York ($65,451); University of Illinois Eye and Ear Infirmary, Chicago ($30,430); Institute of Ophthalmology, University of London and Moorfields Eye Hospital in London ($95,000); New York University Medical Center, School of Medicine, New York ($84,100); Medical College of Virginia, Virginia Commonwealth University, Richmond ($54,923); and Cullen Eye Institute, Baylor College of Medicine and Hermann Eye Center, University of Texas Medical School, Houston ($73,000).

The National Retinitis Pigmentosa Foundation is a nonprofit organization, established in 1971 to organize and develop research to discover the cause, treatment, and prevention of retinal degenerative diseases. Retinitis pigmentosa is a degenerative disease characterized by a loss of night vision, progressing to loss of peripheral vision, and a gradual loss of vision. Usher's syndrome, the combination of RP and deafness, is the largest cause of combination deafness and blindness in the country. Another retinal degenerative disease, macular degeneration is the largest cause of blindness in individuals over 65 years of age.

For further information on RP, write to the National RP Foundation, 8331 Mindale Circle, Baltimore, MD 21207.

NEW RESEARCH PROJECT GRANTS AWARDED BY THE NATIONAL RETINITIS PIGMENTOSA FOUNDATION
July 1, 1980, to June 30, 1981

Institution	Principal Investigator	Amount Awarded
College of Physicians and Surgeons Columbia University, New York City	Peter Gouras, M.D.	$ 65,451
Cullen Eye Institute, Baylor College of Medicine Hermann Eye Center University of Texas Medical School Houston, Texas	Joe G. Hollyfield, Ph.D. Charles A. Garcia, M.D.	73,000

Institution	Principal Investigator	Amount Awarded
Institute of Ophthalmology, University of London Moorfields Eye Hospital London, England	Geoffrey Arden, M.D., Ph.D. Alan Bird, M.D.	$ 95,000
University of Illinois, Eye and Ear Infirmary Chicago, Illinois	Gerald Fishman, M.D.	30,430
Medical College of Virginia, Virginia Commonwealth University Richmond, Virginia	Joann Boughman, Ph.D. Walter E. Nance, M.D., Ph.D.	54,923
New York University Medical Center School of Medicine New York, New York	Ronald Carr, M.D.	84,100
The Wilmer Eye Center, Johns Hopkins Hospital Baltimore, Maryland	Daniel Finkelstein, M.D.	79,252
Jules Stein Institute University of California Los Angeles, California	Michael Hall, Ph.D.	55,000
Berman-Gund Laboratory for the Study of Retinal Degenerations Massachusetts Eye and Ear Infirmary Harvard Medical School Boston, Massachusetts	Eliot Berson, M.D.	111,542

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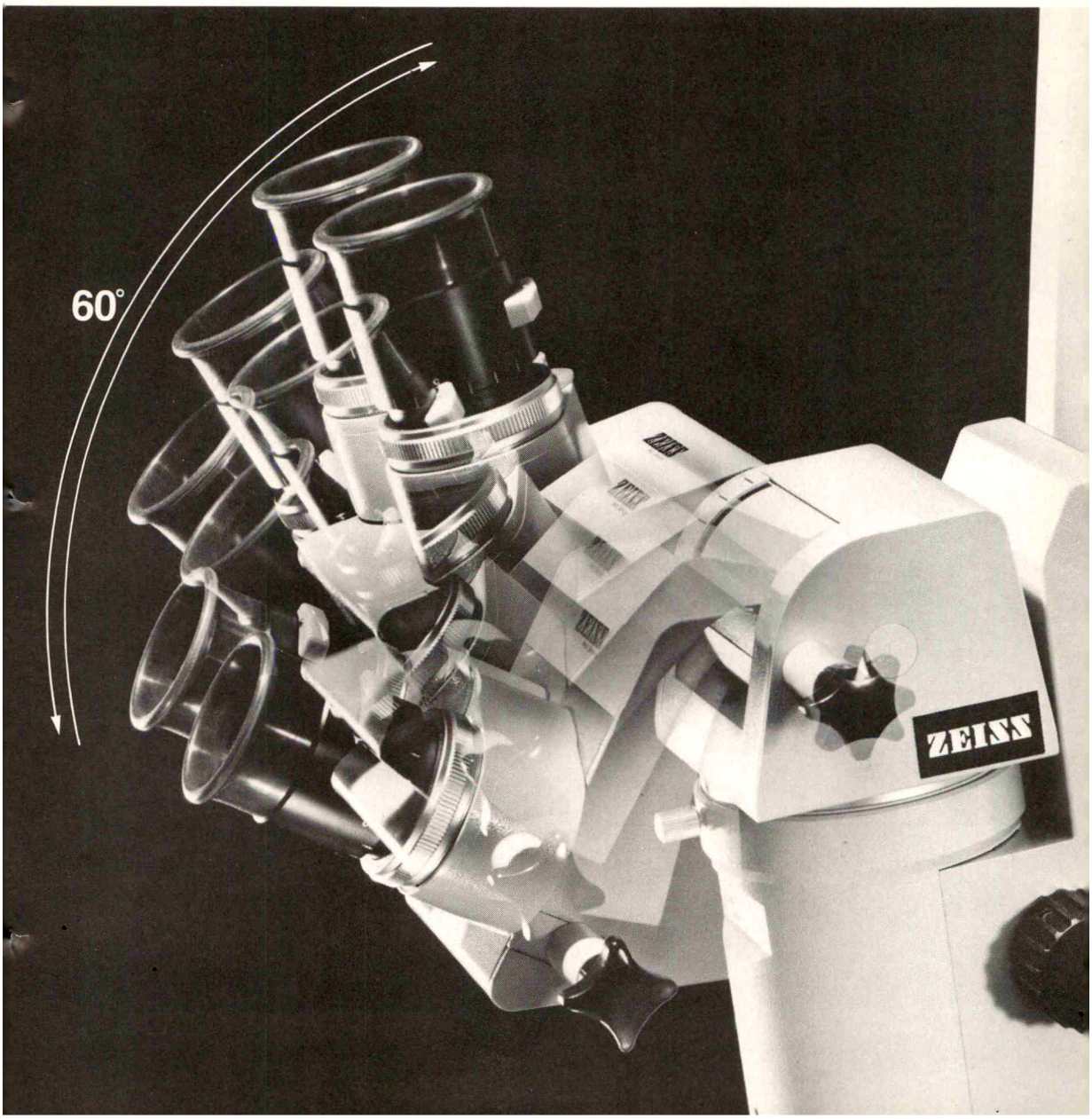
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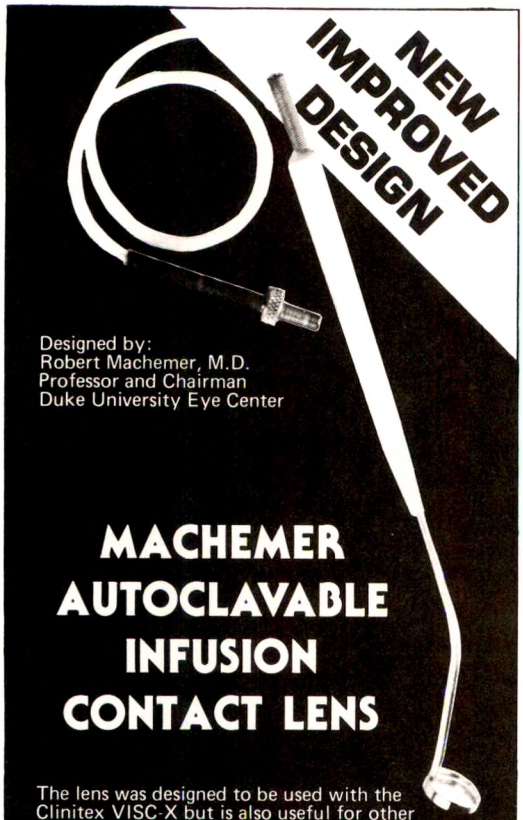
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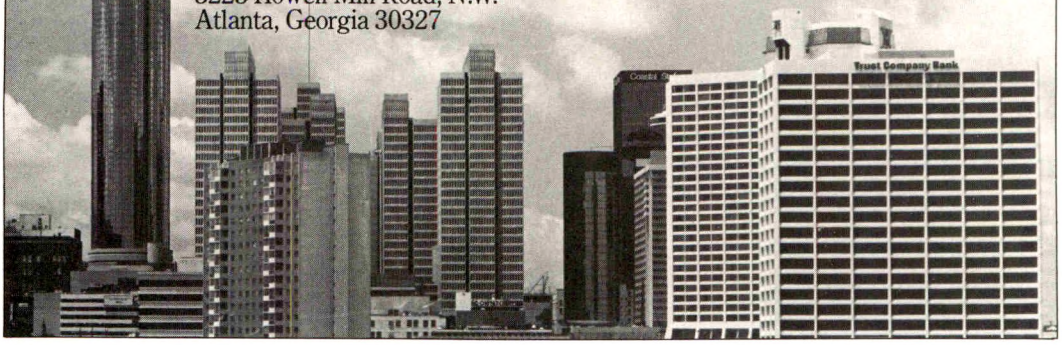
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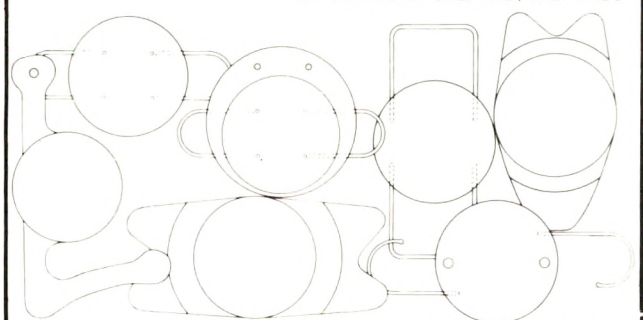
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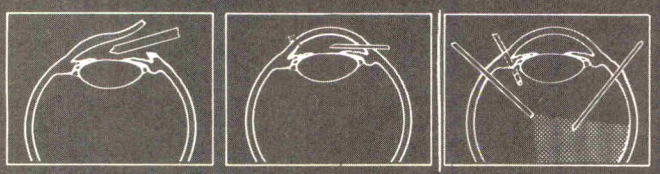
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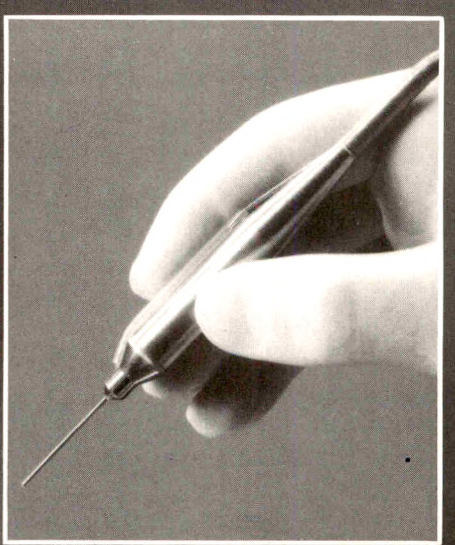
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STRABISMUS (4 hours)	Robert D. Reinecke, M.D., Professor and Chairman, Department of Ophthalmology, Albany Medical College of Union University
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GLAUCOMA AND METABOLIC DISEASES (4 hours)	H. Saul Sugar, M.D., Clinical Professor, Department of Ophthalmology, Wayne State University School of Medicine; Consultant, Sinai Hospital of Detroit; Consultant Glaucoma, Henry Ford Hospital
PHARMACOLOGY AND THERAPEUTICS (4 hours)	Joel S. Mindel, M.D., Ph.D., Assistant Professor, Department of Ophthalmology, Mt. Sinai School of Medicine
MEDICAL OPHTHALMOLOGY (4 hours)	Paul Henkind, M.D., Ph.D. Professor and Chairman, Department of Ophthalmology, Montefiore Hospital and Medical Center

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TOPICS

- REFRACTIVE SURGERY (Friday, March 13)
  Radial Keratotomy (Theory, Animal Studies, Technique, Human Results, Proposed Collaborative Study), Keratophakia, Keratomileusis, Epikeratophakia, Intraocular Lenses
- PEDIATRIC ANTERIOR SEGMENT SURGERY (Saturday, March 14)
  New Approaches to Congenital Cataracts, Subluxated Lenses, Secondary Membranes, Anterior Vitrectomy, Management of Pediatric Ocular Trauma
- UPDATE ON RETROLENTAL FIBROPLASIA (Sunday, March 15)
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11:15 - 12:15	Lens, Aqueous Biochemistry	Leo T. Chylack, Jr.
1:15 - 3:15	Glaucoma	Richard J. Simmons, Bernard Schwartz
3:30 - 5:30	Pharmacology and Therapeutics	Bernard Schwartz
6:00 - 8:00	Cocktail Party	

Sunday, December 7, 1980

7:45 - 9:45	Plastic Surgery	Arthur S. Grove, Jr.
10:00 - 12:00	Lacrimal Surgery, Orbital Disease	Arthur S. Grove, Jr.
12:45 - 2:45	Corneal, External Disease	Claes H. Dohlman, Kenneth R. Kenyon
3:00 - 5:00	Microbiology, Immunology	Mathea R. Allansmith

Friday, December 12, 1980

7:30 - 9:30	Neuro-Ophthalmology	Simmons Lessell
9:45 - 11:30	Neuro-Ophthalmology	Simmons Lessell
12:30 - 1:45	Emergency Medicine and Anesthesia	John V. Donlon, Jr.
1:45 - 3:15	Strabismus	Lloyd Wilcox
3:30 - 5:30	Strabismus	Lloyd Wilcox

Saturday, December 13, 1980

7:45 - 8:30	Embryology	Film
8:30 - 9:45	Developmental Anomalies	Richard M. Robb
10:00 - 11:00	Pediatric Inflammation	William P. Boger III
11:00 - 12:00	Genetics	Robert A. Petersen
12:45 - 2:45	Pathology	Daniel M. Albert
3:00 - 5:00	Pathology	Daniel M. Albert

Sunday, December 14, 1980

7:30 - 8:45	Contact Lenses, Optics	David Miller
9:00 - 11:00	Retinal Electrophysiology, psychophysics	Eliot L. Berson
11:00 - 11:45	Retina, Vitreous I	John Weiter
12:45 - 3:15	Retina, Vitreous II	Edward A. Ryan
3:30 - 5:00	Retina, Vitreous III	John Weiter

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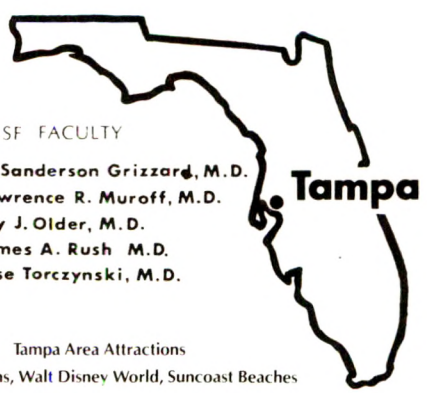
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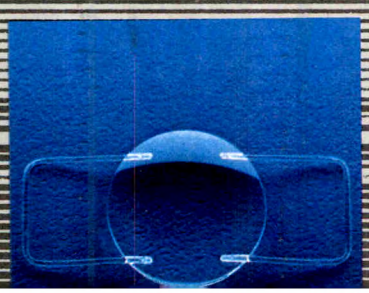
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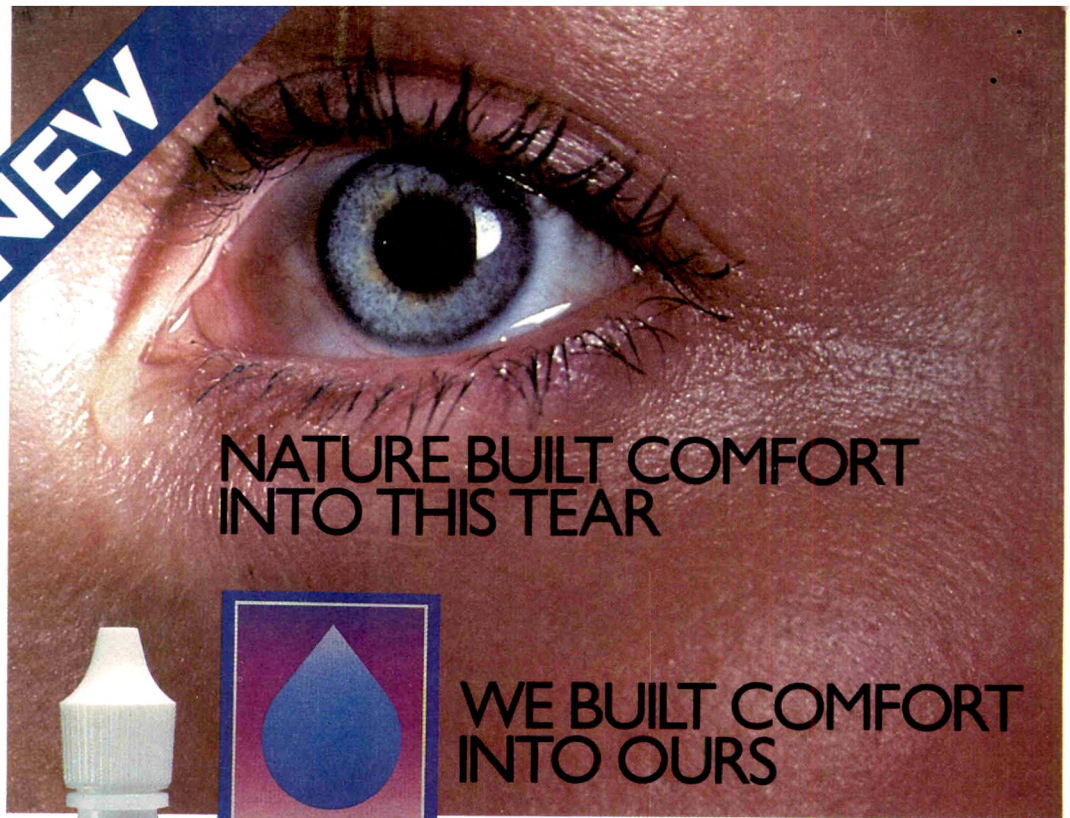
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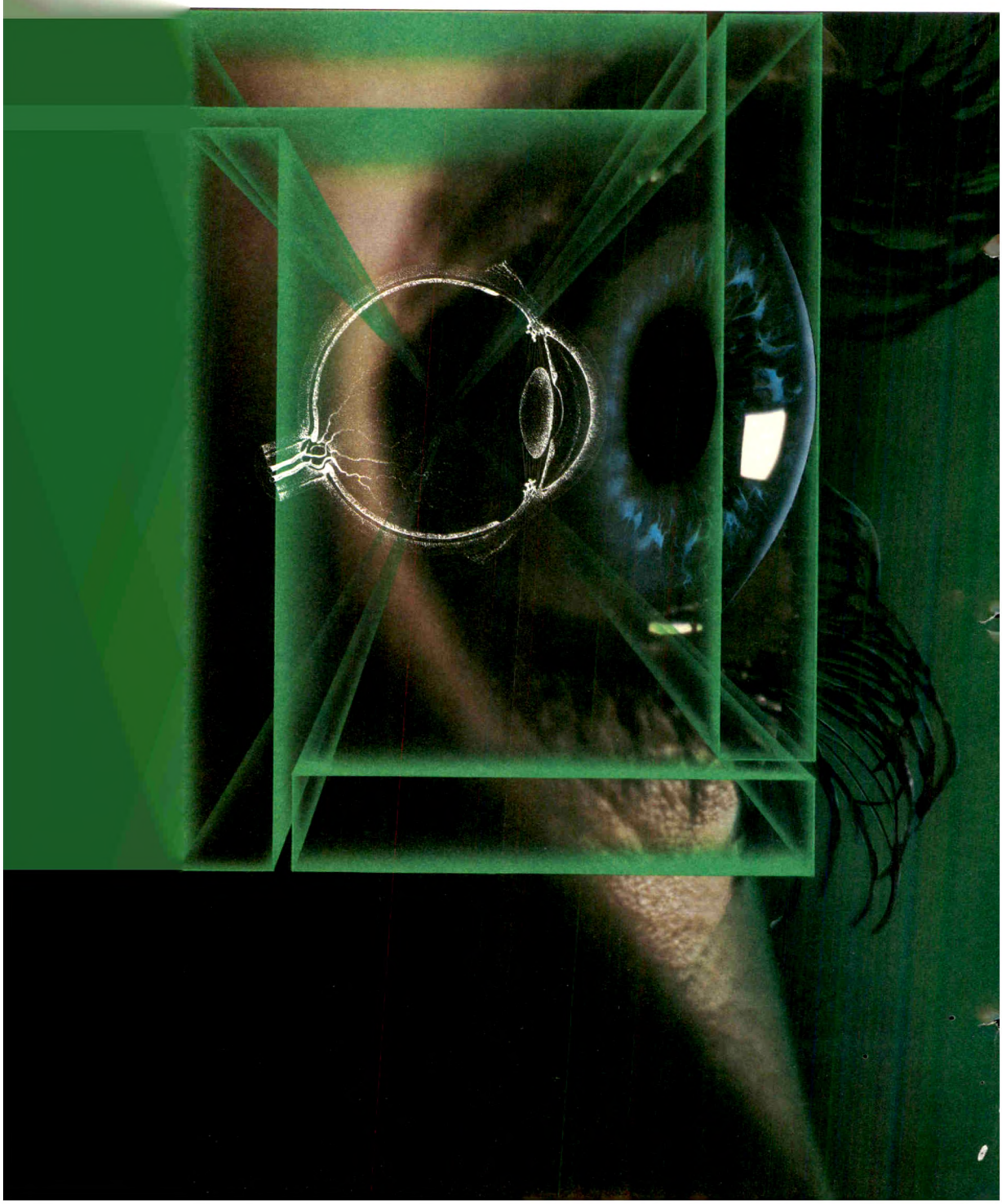
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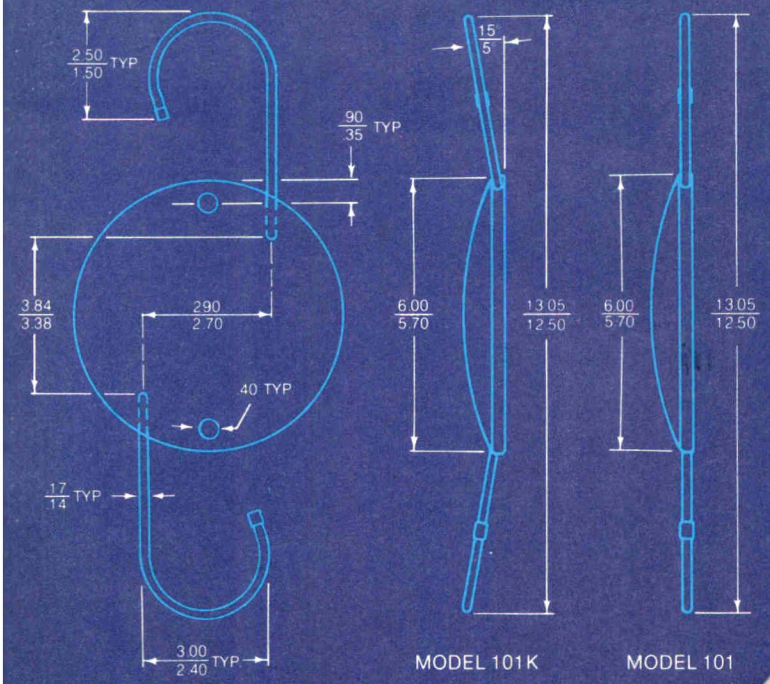
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1. Data on file at Alcon Laboratories.
2. Zimmerman, T. J., Ophthalmology Audio Digest, Vol. 17, No. 1

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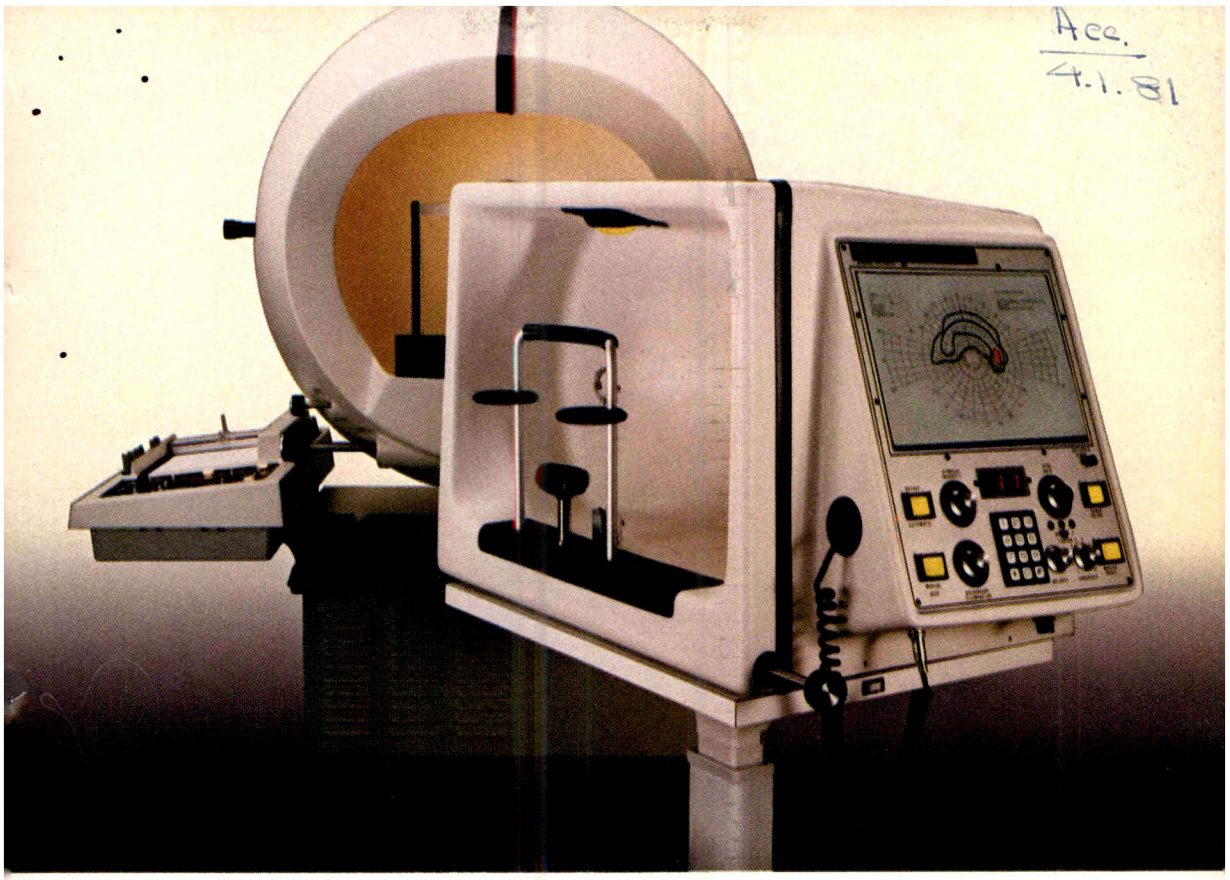
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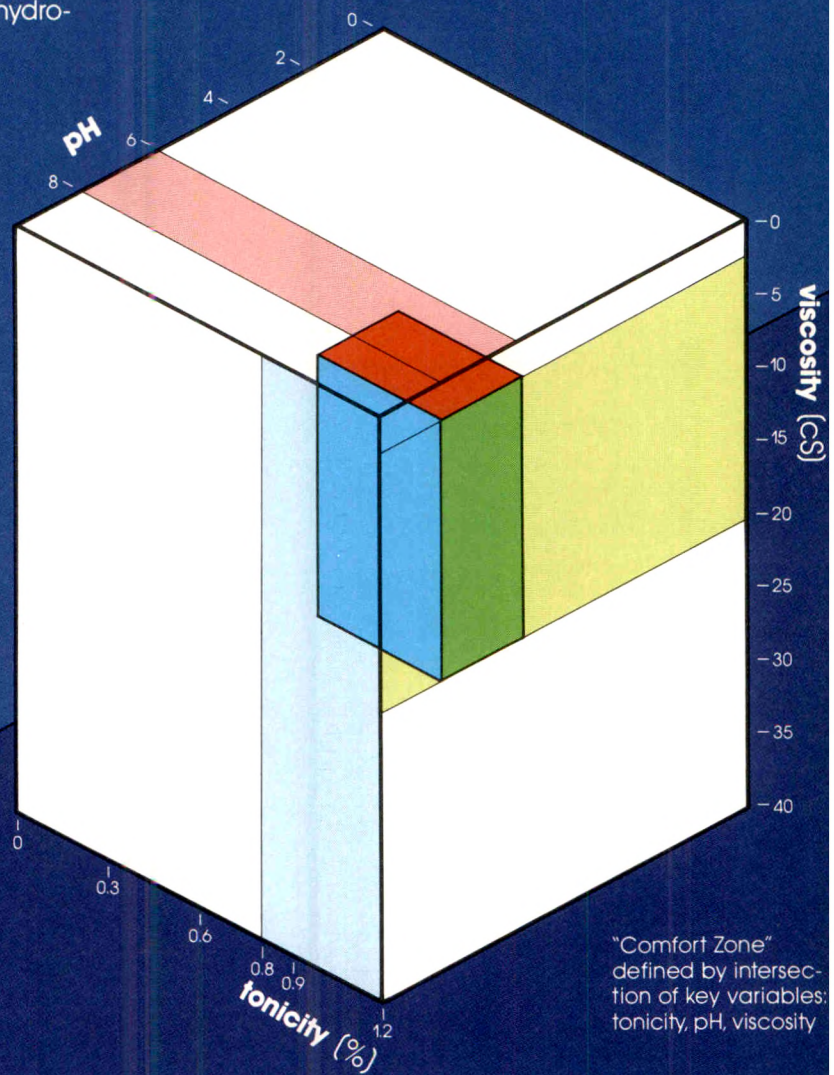
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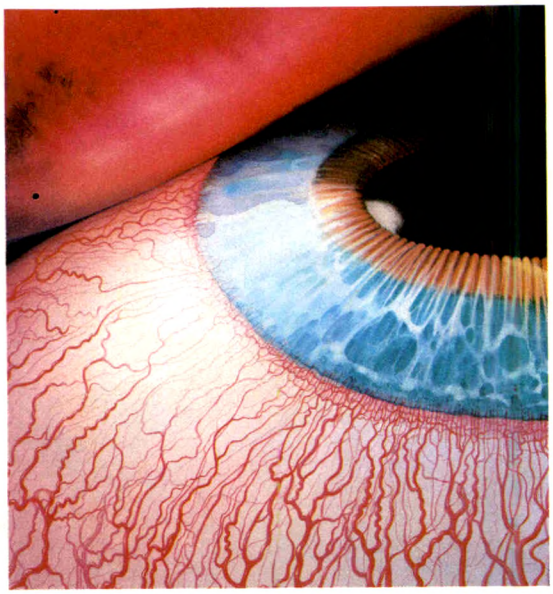
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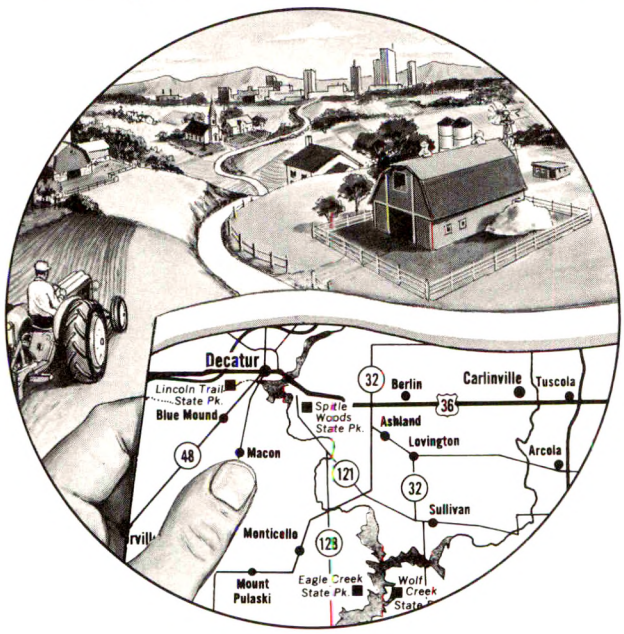
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[1]Leibowitz HM, Kupferman A. Bioavailability and therapeutic effectiveness of topically administered corticosteroids. Trans Am Acad Ophthalmol Otolaryngol 1975;79(1): OP78-OP88.

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Address _____

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Phone _____ / _____
Area Code

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Please fill in all spaces. These are the seven measurements we need.

Patient: _____

OPHTHALMOMETER

R _____ X _____ / _____ X _____

L _____ X _____ / _____ X _____

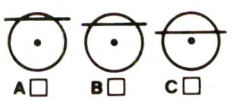
SPECTACLE RX

R _____ X _____ Near Add _____

L _____ X _____ Near Add _____

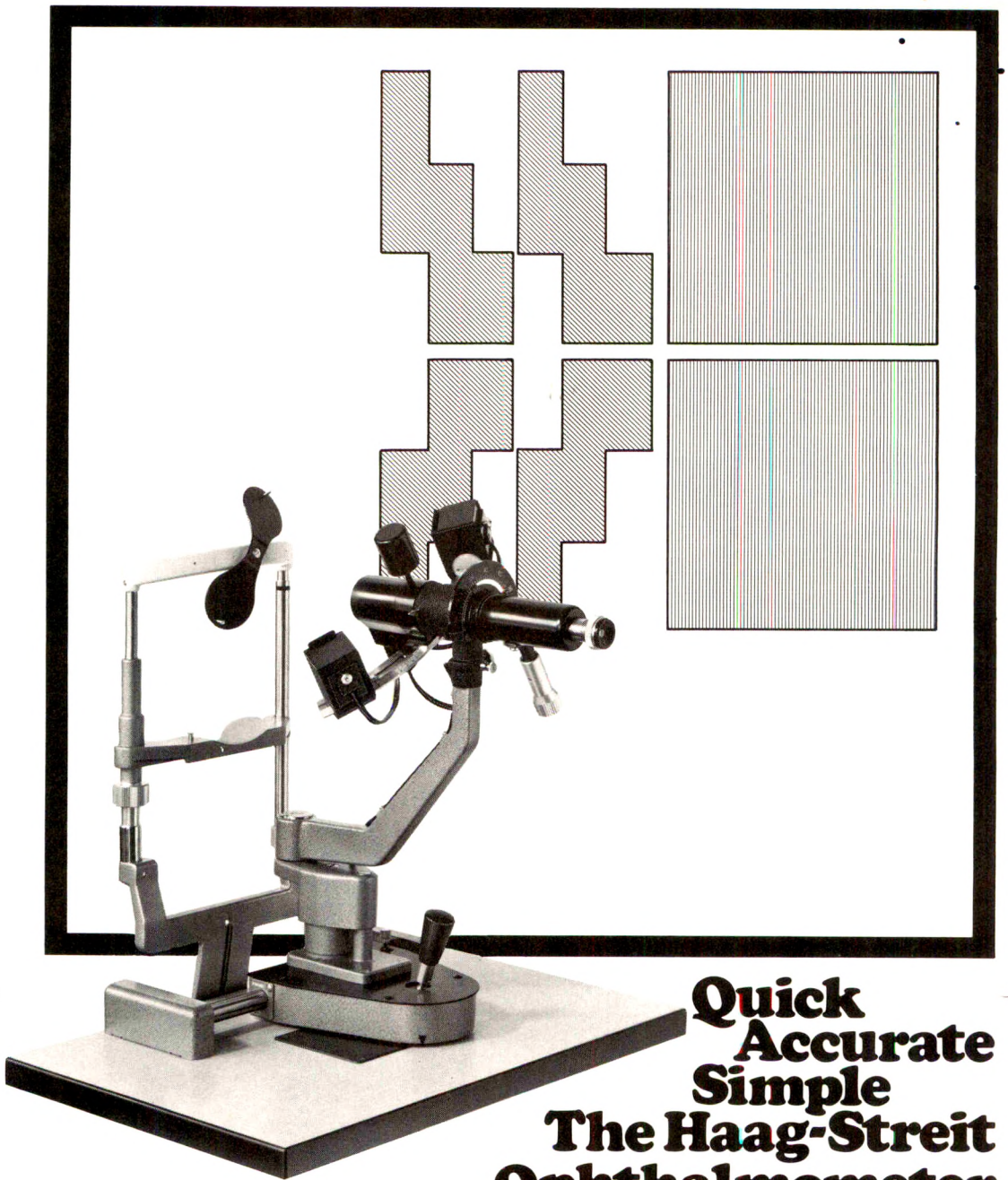
Palpebral Fissure _____

Upper Lid Position



MM Size of Cornea _____ MM
Spectacle Vertex Distance _____ MM
Pupil Diameter _____ MM
Color: Grey & Blue.

The Presbiflex® Lens is U. S. patent pending.



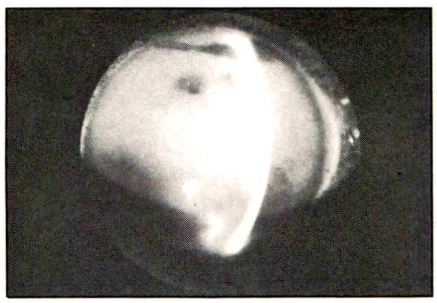
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THE LANCASTER COURSE IN OPHTHALMIC HISTOPATHOLOGY



Edited by **William C. Frayer,** MD,
Professor of Ophthalmology,
University of Pennsylvania School of Medicine,
Philadelphia, Pennsylvania.

This outstanding collection of 35mm Veri Color slides with accompanying text provides an invaluable teaching aid for the introduction of ophthalmic histopathology. It was produced by members of the faculty of the nationally recognized Lancaster Course, a core program in ophthalmology training held for six weeks each summer at Colby College, Waterville, Maine.

For this superb educational tool, the authors have abstracted the most salient sections of the course, with appropriate slides, to present in concise form the core on ophthalmic histopathology. The textual material is specially formulated for easy and relevant use with 666 full-color 35mm slides.

The Lancaster Course in Ophthalmic Histopathology is an excellent primer for the ophthalmology resident taking board exams. The practicing ophthalmologist will find the set an excellent referral for clinical cases or a helpful refresher before taking continuing education courses.

271 pages of text. Illustd. 666 35mm full-color slides. Ready October 1980.

Contents and Contributors

Unit 1—OCULAR INFLAMMATION—Barbara W. Streeten, MD, 30 slides
Unit 2—SUPPURATIVE AND NONGRANULOMATOUS OCULAR INFLAMMATION—Barbara W. Streeten, MD, 41 slides
Unit 3—GRANULOMATOUS INFLAMMATION OF THE EYE—Daniel M. Albert, MD, 31 slides
Unit 4—CONGENITAL ANOMALIES AND THE PHAKOMATOSES—William C. Frayer, MD, 45 slides
Unit 5—DISEASES OF THE EYELIDS—Barbara W. Streeten, MD, 47 slides
Unit 6—THE ORBIT—Myron Yanoff, MD, 60 slides
Unit 7—DISEASES OFTHE CONJUNCTIVA, CORNEA, AND SCLERA; OCULAR TRAUMA: ACCIDENTAL AND SURGICAL—Morton E. Smith, MD, 120 slides
Unit 8—PATHOLOGY OF THE VITREOUS—W. Richard Green, MD, 29 slides
Unit 9—PATHOLOGY OF THE RETINA—W. Richard Green, MD, 104 slides
Unit 10—PATHOLOGY OF THE CRYSTALLINE LENS—William C. Frayer, MD, 27 slides
Unit 11—DISEASES OF THE OPTIC NERVE—William C. Frayer, MD, 41 slides
Unit 12—HYPOTONY AND GLAUCOMA—Myron Yanoff, MD, 30 slides
Unit 13—RETINOBLASTOMA AND PSEUDOGLIOMA—Daniel M. Albert, MD, and Delia N. Sang, MD, 30 slides
Unit 14—MELANOMAS OF THE EYE—Daniel M. Albert, MD, and Carmen A. Puliafito, MD, 31 slides

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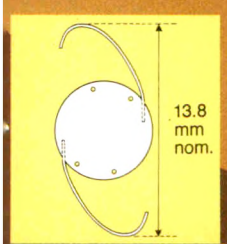
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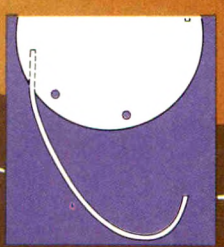
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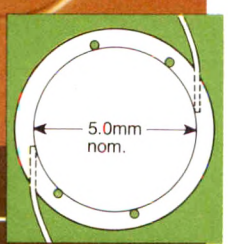
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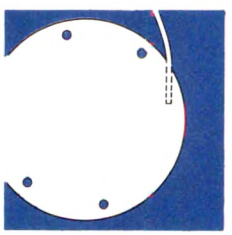
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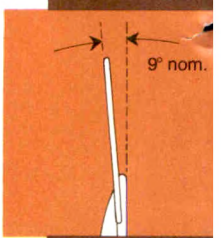
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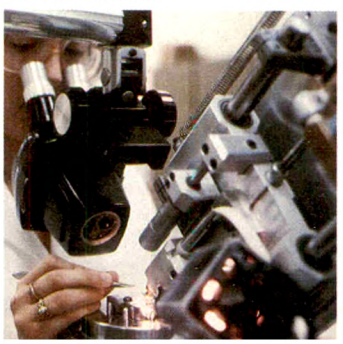
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CONTRAINDICATIONS: These products are contraindicated in acute purulent conjunctivitis and blepharitis; tuberculous, fungal or viral lesions of the skin or eye, including herpes simplex, dendritic keratitis, vaccinia or varicella; and conditions involving the posterior segment of the eye. They are also contraindicated in those individuals who have shown hypersensitivity to any of their components.

PRECAUTIONS: Extended ophthalmic use of topical steroid therapy may result in glaucoma with possible damage of the optic nerves, posterior subcapsular cataract formation, or may aid in the establishment of secondary ocular infections from fungi or viruses liberated from ocular tissues. It is advisable that intraocular pressure be checked frequently. In those diseases causing thinning of the cornea, perforation has been known to occur with the use of topical steroids. As with any antibiotic preparation, prolonged use may result in the overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

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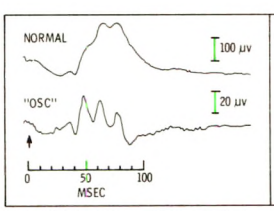
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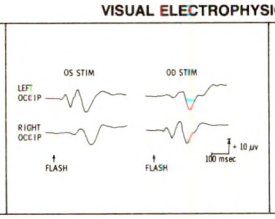
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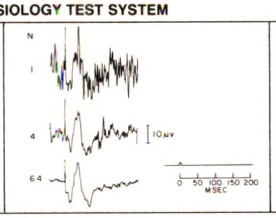
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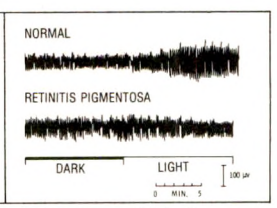
1. *ERG from normal dark-adapted eye. Above: "Normal" mode; Below: "OSC" mode, gain increased. In "OSC" mode the response is filtered to emphasize the oscillatory potential.*



2. *Visual evoked responses recorded from right and left occiputs (dual channel system). The patient has an optic chiasm lesion.*



3. *ERG recorded from lower eyelid with averaging. "N"s at left of traces indicate the number of responses averaged.*



4. *Above: normal EOG with light-peak /dark-trough ratio of 2.4. Below: abnormal EOG in retinitis pigmentosa with light-peak / dark-trough ratio of 1.0. (EOG mode, 0.5 mm chart speed).*

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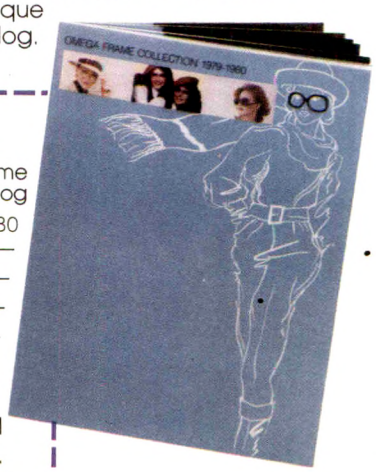


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PRACTICE SEMINAR
(9:00 a.m. - 12:00 noon Thursday)

Scientific Program begins at 2:00 p.m. Thursday

Thursday Afternoon (January 29)

Symposium: EXTENDED WEAR OF CONTACT LENSES IN APHAKIA ..... Jack Hartstein, M.D., *Moderator*
Patient Selection ..... Joseph A. Baldone, M.D.
Cooper Lenses ..... Richard G. Lembach, M.D.
Hydrocurve Lenses ..... Perry S. Binder, M.D.
Sauflon Lenses ..... Anthony B. Nesburn, M.D.
Silicone Lenses ..... Robert T. Blackhurst, M.D.
CSI Lenses ..... H. Dwight Cavanagh, M.D.
Complications of Extended Wear Lenses ..... G. Peter Halberg, M.D.
Panel Discussion

THE USE OF EXTENDED WEAR LENSES IN MYOPIA ..... Louis A. Wilson, M.D.
Symposium: THE USE OF EXTENDED WEAR LENSES FOR THERAPEUTIC PURPOSES ..... Richard H. Keates, M.D., *Moderator*
Cooper Lenses ..... Richard G. Lembach, M.D.
Bausch & Lomb Lenses ..... John Brodrick, M.D.
Sauflon Lenses ..... Bruce Bodner, M.D.
Softsight Therapeutic Lenses ..... Henry Gelender, M.D.
PRACTICAL WORKSHOPS ..... *Topics to be announced*

Friday Morning (January 30)

Symposium: TORIC SOFT LENSES ..... Barry A. Maltzman, M.D., *Moderator*
Lens Availability and Design ..... Fitting Techniques and Wear Expectations (Dura-Soft—Hydrocurve)
Fitting Techniques and Wearing Expectations (Hydromore–Miracon–Hydron)...Pitfalls to Avoid in Fitting Toric Lenses
Panel Discussion - Questions and Answers. ***Participants:*** Herschell H. Boyd, M.D., Paul R. Honan, Jr., M.D., Bernard J. Slatt, M.D., Harold A. Stein, M.D.
Symposium: HYGIENIC CARE OF SOFT LENSES ..... Ellis Gruber, M.D., *Moderator*
The Role of Saline and Enzymatic Cleaners in the Care of Soft Lenses ..... Mark B. Abelson, M.D.
The Role of Surfactant Cleaners in the Care of Soft Lenses ..... R. Linsy Farris, M.D.
Hot or Cold Care Systems? ..... Ellis Gruber, M.D.
OPHTHALMIC DEVICES: A Review of Three Years of FDA Involvement ..... Max Talbott, Ph.D.
Symposium: CONTACT LENSES IN INFANTS AND CHILDREN ..... Harold P. Koller, M.D., *Moderator*
Indications and Available Materials for Contact Lenses in Infants and Children ..... Judith E. Gurland, M.D.
Pathophysiology of Contact Lens Therapy in Infants and Children ..... I. Matthew Rabinowicz, M.D.
Contact Lens Fitting Techniques Unique to the Pediatric Population ..... Harold P. Koller, M.D.
Indications and Complications of Intraocular Lenses vs. Contact Lenses in Infants and Children ..... Jerry W. Maida, M.D.
Results of Extended and Daily Wear Contact Lenses in Infants and Children ..... Harold P. Koller, M.D.
DISTINGUISHED VISITING LECTURER ..... **Thomas F. Spring, M.D.**

Spouse Luncheon at Don the Beachcomber Compliments of CLAO

Friday Afternoon (January 30)

CONTACT LENS COURSES - Section I ..... R. Linsy Farris, M.D., *Director, Course Program*
1. Current Concepts in the Correction of Myopia—Extended Wear Lens, Orthokeratology and Refractive Corneal Surgery ..... Perry S. Binder, M.D.
2. Management of Corneal & Contact Lens Problems ..... G. Peter Halberg, M.D., Robert A. D'Amico, M.D.

3. How to Select the Best Soft Lens for Your Patient in 1981 ..... Ellis Gruber, M.D.
4. Astigmatism Correction with Hard & Soft Contact Lenses ..... Paul R. Honan, M.D., Herschell H. Boyd, M.D.
5. Specialized Techniques of Lens Fitting ..... Joseph W. Soper
6. Hard Lens Modification—Basic Lens Evaluation and Modification Techniques ..... Paul R. Honan, Jr., M.D., Marvin A. Garland, M.D.
7. Fitting of CAB and Silicone Lenses for Daily and Extended Wear .. Jay I. Lippman, M.D., Daniel Sigband, M.D.
8. Contact Lens Fitting in Infants & Children ..... Harold P. Koller, M.D.
9. Introduction to Refractive Keratoplasty ..... Miles H. Friedlander, M.D., Herbert E. Kaufman, M.D.
10. Contact Lens—Lacrimal System Interaction ..... David Lamberts, M.D., Frank J. Holly, Ph.D.
11. Extended Wear of Contact Lenses ..... Rao N. Gullapalli, M.D.
12. Contact Lens Equipment and Solutions ..... Ronald H. Akashi, M.D.

INTRAOCULAR LENS COURSES—Section I ..... Herve M. Byron, M.D., *Director, Course Program*

13. Intracapsular Extractions with Implants—I ..... Stephen Obstbaum, M.D., Marvin Kwitko, M.D.
14. Extracapsular Extractions with Implants—I ..... William Harris, M.D., Robert F. Azar, M.D.
15. Techniques for the Advance Implant Surgeon—I ..... Richard P. Kratz, M.D., Charles Kelman, M.D.
16. Special Instrumentation & Therapeutic Agents ..... Buol K. Heslin, M.D., David J. McIntyre, M.D.

CONTACT LENS COURSES—Section II ..... R. Linsy Farris, M.D., *Director, Course Program*

1. Extended Contact Lens Wear ..... Anthony B. Nesburn, M.D.
2. Update on Therapeutic Contact Lenses ..... Edward L. Shaw, M.D.
3. Keratoconus ..... Frank B. Hoefle, M.D.
4. Soft Lens Techniques ..... Joseph W. Soper
5. Bifocal & Multifocal Contact Lenses for the Presbyope and Aphake ..... Frank J. Weinstock, M.D., Abraham Schlossman, M.D.
6. The Beginning Training & Utilization of a Contact Lens Technician ..... Robert J. Crossen, M.D.
7. Starting from Scratch—Contact Lens Fitting ..... Paul R. Honan, M.D., Herschell H. Boyd, M.D.
8. Hard Lens Modification—Advanced Techniques ..... Marvin A. Garland, M.D., Paul R. Honan, M.D.
9. Soft Toric Lenses ..... Barry A. Maltzman, M.D.
10. Fitting Contact Lenses by Piggy Back Technique ..... Joseph A. Baldone, M.D.
11. Contact Lens Fitting in Difficult Cases ..... Donald J. Doughman, M.D.
12. Ocular Allergy Problems in Contact Lens Wearers ..... Mark B. Abelson, M.D., Ira J. Udell, M.D.

INTRAOCULAR LENS COURSES—Section II ..... Herve M. Byron, M.D., *Director, Course Program*

13. Intracapsular Extraction with Implants—II ..... Stephen Obstbaum, M.D., Marvin Kwitko, M.D.
14. Extracapsular Extraction with Implants—II ..... William Harris, M.D., Robert F. Azar, M.D.
15. Techniques for the Advanced Implant Surgeon—II ..... Richard Kratz, M.D., Charles Kelman, M.D.
16. Keratorefractive Surgery ..... James V. Aquavella, M.D., Dennis D. Shepard, M.D.

Wine and Cheese Tasting Party Compliments of Barnes-Hind Pharmaceuticals, Inc.

Saturday Morning (January 31)

Symposium: INTERACTION OF THE CORNEAL EPITHELIUM, TEAR FILM, AND THE CONTACT LENS ..... James V. Aquavella, M.D., *Moderator*

Stimulation and Tear Flow ..... Jules L. Baum, M.D.
The Contact Lens Surface ..... Rao N. Gullapalli, M.D.
Immunological Aspects ..... Mark B. Abelson, M.D.
Tear Film ph ..... Michael A. Lemp, M.D.
Tear Film Tonicity ..... R. Linsy Farris, M.D.
Summary and Discussion ..... James V. Aquavella, M.D.

Symposium: PROTECTION OF ENDOTHELIUM DURING IOL SURGERY .. Herbert E. Kaufman, M.D., *Moderator*

OVERVIEW—Problem of Cell Loss During Surgery ..... Herbert E. Kaufman, M.D.
Quantitation of Cell Loss and Its Prevention by Altering Surgical Technique ..... William M. Bourne, M.D.
The Use of Viscose Agents to Minimize Surgical Cell Loss ..... David Miller, M.D.
Lens Coatings and Other Non-Viscose Type Approaches to the Prevention of Cell Loss . Herbert E. Kaufman, M.D.

ISSUES FACING OPHTHALMOLOGY ..... George Edward Garcia, M.D., *President American Association of Ophthalmology*

CONRAD BERENS LECTURER ..... **John A. Dyer, M.D.**
OPHTHALMIC TECHNICIANS COURSE ..... Topics to be announced

Saturday Afternoon (January 31)

THEODORE E. OBRIG MEMORIAL LECTURER ..... To Be Announced

Symposium: WHAT'S NEW AND INTERESTING IN LENS IMPLANTATION ..... Harold A. Stein, M.D., *Moderator*

Patient Selection—Extended Wear of IOL? ..... Harold A. Stein, M.D.
Instrumentation in Surgery ..... Aziz J. Anis, M.D., F.A.C.S.
Newer Intraocular Lens Designs ..... Ronald W. Barnet, M.D.
Sutures for Cataracts, Implant Surgery and Astigmatism Control ..... J. Elliott Blaydes, Jr., M.D.
Statistics on Implant Results ..... James V. Aquavella, M.D.
Some Mechanical Considerations ..... C. William Simcoe, M.D.
Preventing Complications with IOL's ..... Stanley C. Becker, M.D.
Managing Complications ..... Richard P. Kratz, M.D.

Symposium: IS EXTRACAPSULAR CATARACT SURGERY WORTH IT? .... C. William Simcoe, M.D., *Moderator*
Three panelists (Richard P. Kratz, M.D., Richard H. Keates, M.D., and K. Buol Heslin, M.D.) will each give a 10 minute presentation of their personal experiences regarding: Prognosis for a Successful Repair of Retinal Detachment; Cystoid Macular Edema; Quality of Vision. A Panel Discussion and Questions from the Audience will follow.

CATARACT SURGICAL TECHNIQUES (Movies) ..... Robert F. Azar, M.D., *Moderator*
Recent Advances in Cataract Surgery ..... Leeds E. Katzen, M.D.
Cataract Surgery in Infants and Juveniles ..... J. Elliott Blaydes, M.D.
Extracapsular Technique (Manual) ..... Charles W. Simcoe, M.D.
Phacoemulsification Technique ..... Robert M. Sinskey, M.D.
Extracapuslar Technique (Mechanical) ..... William S. Harris, M.D.
Intracapsular Technique ..... Marvin L. Kwitko, M.D.

International Party Sponsored by Alcon/BP

Sunday Morning (February 1)

SURGICAL CORRECTION OF REFRACTIVE ERRORS
Jack Hartstein, M.D., *Chairman*

Symposium: SURGICAL CORRECTION OF ASTIGMATISM ..... Richard P. Kratz, M.D., *Moderator*
Prevention: Cataract Wound Closure ..... Clifford Terry, M.D.
Comparison of Keratometers ..... Dennis Shepard, M.D.
Correction: Reconstruction of Previous Incision .....Thomas Cravy, M.D.
Corneal Tuck ..... Robert F. Azar, M.D.

Symposium: SURGICAL CORRECTION OF MYOPIA—PRO's AND CON's ..... Walter J. Stark, M.D., *Moderator*
History of Surgical Correction of Myopia and Early Clinical Results ..... Leeds Katzen, M.D.
Surgical Correction of Myopia—Studies in Laboratory Animals and Human Cadaver Eyes ..... Ronald Smith, M.D.
Complications of Radial Keratotomy ..... J. James Rowsey, M.D.
Update of Clinical Experience with Surgical Correction of Myopia ..... Leo D. Bores, M.D.
A Prospective, Multicenter, Clinical Trial of Radial Keratotomy for Myopia ..... George Waring, M.D.
Summary and Conclusions ..... Walter J. Stark, M.D.

Symposium: SURGICAL CORRECTION OF APHAKIA ..... Perry S. Binder, M.D., *Moderator*
Keratophakia and Hypermetropic Keratomileusis ..... Casimir A. Swinger, M.D.
Hypermetropic Keratomileusis ..... Jorge Krumeich, M.D.
Keratophakia & Hyperopia Keratomileusis Using Precarved Frozen Material ..... Miles H. Friedlander, M.D.
Epikeratophakia ..... Herbert E. Kaufman, M.D.
Panel Discussion—Summary and Conclusions

Contact & Intraocular Lens Presentations—Thursday afternoon, Friday morning and Saturday morning

PRACTICE SEMINAR
Thursday, January 29, 1981 8:50 a.m. - 12:45 p.m.
Herschell H. Boyd, M.D., *Chairman*

Symposium: Time to Buy a Computer? Phillip C. Hessburg, M.D., *Moderator*
Participants: Richard H. Keates, M.D., Phillip C. Hessburg, M.D. *Questions from Audience*

Symposium: Office Management of Extended Wear Lenses Herve M. Byron, M.D., *Moderator*

Optical Dispensing by the Ophthalmologist? ..... R. Hugh Minor, M.D.
Office Surgery for Cataracts ..... David P. Morrison, M.D.
Office Surgery for Cataracts ..... Douglas E. Williamson, M.D.
Merits of a Subjective Refractor ..... Robert F. Azar, M.D.
Merits of a Subjective Refractor ..... Herschell H. Boyd, M.D.
Oil and Gas as a Diversion ..... Charles R. Moore, M.D.
Stop Your P.S. Corporation! ..... Paul R. Honan, Jr., M.D.
Security for the 80's ..... Herschell H. Boyd, M.D.

Questions from Audience

See next page for Ophthalmic Technicians Program, Special Notes and Registration Form

OPHTHALMIC TECHNICIAN PROGRAM
Saturday, January 31, 1981

Update of Contact Lenses
for Ophthalmic Assistants, Contact Lens Technicians and Residents

Director: Harold A. Stein, M.D. **Program Coordinator:** Penny Cook, F.O.C.L.A.
Faculty: L. Dean Clements, Doris Gaston, C.O.T., Ellis Gruber, M.D., Keith Harrison, F.O.C.L.A., Maija Kaufman, R.N., C.O.T., John Morgan, M.D., Bernard J. Slatt, M.D., Kenneth Swanson, C.O.T.
Instructional Objectives:
This course is designed for the resident, ophthalmic assistant and contact lens technician who has some experience in contact lens fitting. It is designed to review existing material available in the contact lens field and bring new material concerning current items of interest in the contact lens world.

Topics: Physiology Related to Contact . . . New Thrusts in Cornea and Tear Research . . . Clinical Selection of Patients . . . Practical Tips on Dispensing Contact Lenses . . . Office Cleaning of Soft Lenses . . . Staining Patterns with Hard Lenses . . . Selecting the Correct Lens . . . Toric Soft Lenses . . . Systems for Fitting Hard Lenses . . . Silicone Lenses . . . Newer Lens Materials and Designs . . . Gas Permeable Lenses . . . Extended Wear Lenses . . . Therapeutic and Coloured Lenses . . . Problems with Soft and Hard Contacts

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An optional Practice Management Seminar will be conducted on Thursday, January 29, 1981 immediately preceding the scientific session. There is no registration fee for the Practice Management Seminar; the registration fee for the Scientific Session is the total fee for all sessions.

Registration for the Scientific Session will take place on Wednesday, January 28 from 12:00 noon to 5:00 p.m. and Thursday, January 29 from 7:30 a.m. to 5:30 p.m. Use the registration form below to pre-register and save time at the meeting.

Special One Day Registration—Anyone who wishes to attend the meeting for only one day may do so for the nominal registration fee of $50.00 which includes participation in that day's sessions and the evening function.

Refund Policy. Full refund if cancellation is received by January 1, 1981. A $25.00 service charge per person for cancellations from January 2, 1981 to January 28, 1981. No refunds for cancellations received after January 29, 1981.

Spouse Luncheon (Friday)—Compliments of the Contact Lens Association of Ophthalmologists, Inc.

A special room rate has been arranged with the Sahara Hotel. For room reservations only, write to the Sahara Hotel, Las Vegas, Nevada 89114.

PRE-REGISTRATION FORM—CLAO MID-WINTER NATIONAL MEETING
January 29 - February 1, 1981 Sahara Hotel Las Vegas, Nevada

Name _____ M.D. _____ Other _____

Address _____

City, State, Zip Code _____ Telephone _____

☐ Physician Member of CLAO—US$100.00 ☐ Physician Non-Member—US$250.00

☐ Resident Physician in Ophthalmology—US$50.00

☐ Technician* employed by Ophthalmologist (Physician Member of CLAO). Must be identified by letter by the employing ophthalmologist—US$75.00

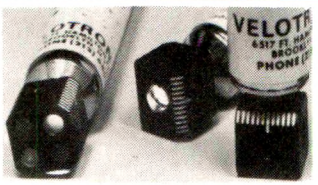
☐ Technician* employed by Ophthalmologist (Non-Member of CLAO). Must be identified by letter by the employing ophthalmologist—US$175.00

☐ ALL OTHERS—US$275.00 ☐ ONE DAY REGISTRATION—US$50.00 Please specify day: _____

Please make check payable to: CLAO—Mid Winter Meeting and forward to R. Linsy Farris, M.D.
Treasurer—CLAO
2620 Jena Street
New Orleans, LA 70115

*Technicians. This fee includes the entire meeting (3-1/2 days), in addition to the Ophthalmic Assistants and Technicians Course.

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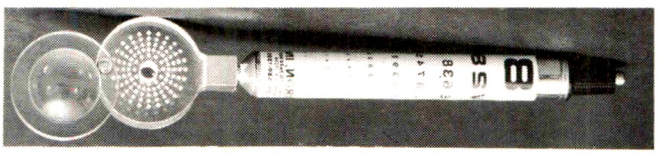
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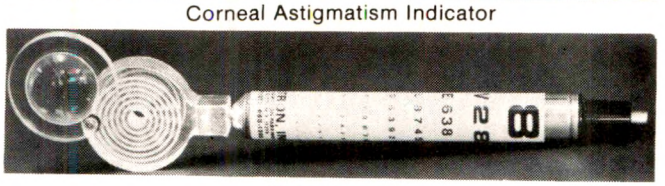
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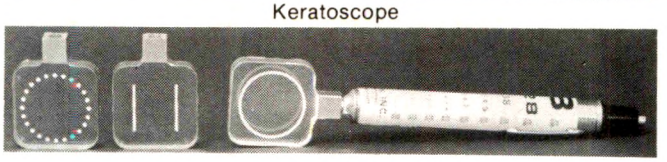
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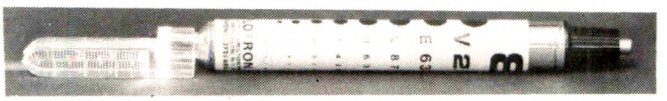
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Keratoscope



Corneal Scanners



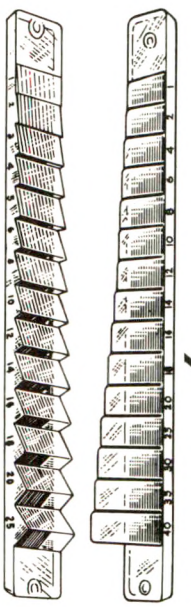
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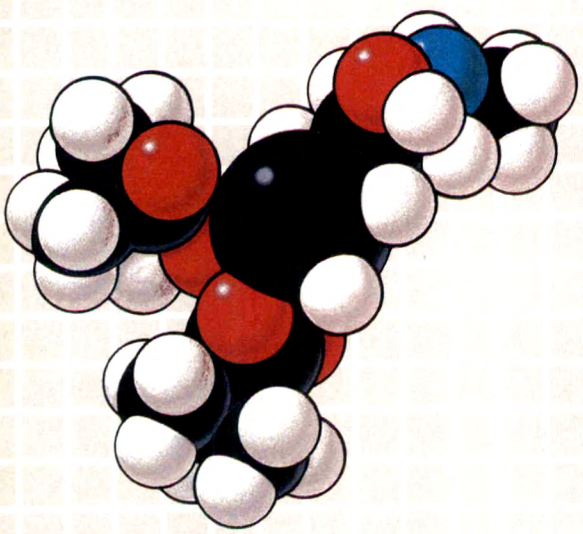
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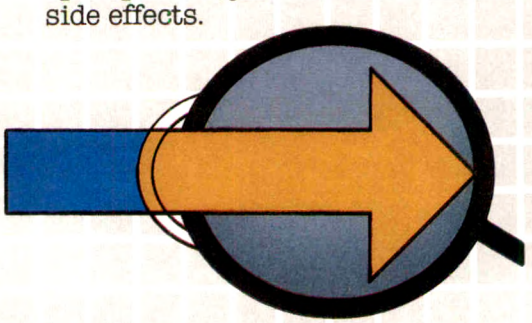
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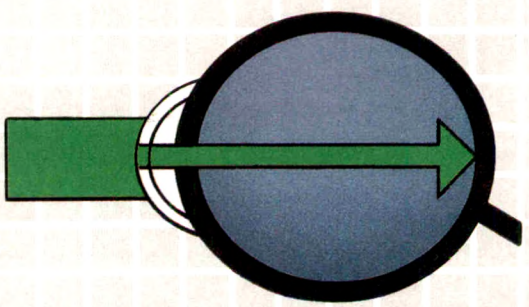


Propine (dipivefrin HCl) molecule depicting addition of the two pivalyl side chains.

This addition creates a more lipophilic compound which is absorbed better into the eye — 17 times better penetration.[1] Once inside, it is converted to epinephrine by enzymes within the eye. This action makes Propine more potent than comparable concentrations of epinephrine, yet with fewer side effects.



Propine (dipivefrin HCl): better penetration with minimal side effects.



Epinephrine: less efficient penetration with greater chance of side effects.

The first prodrug for glaucoma therapy.

Concomitantly with timolol or pilocarpine, or as initial therapy, Propine™ (dipivefrin HCl) provides:

Effective control of IOP

Therapeutic response comparable to 2% pilocarpine q.i.d.
Reduces mean pressure 20 to 24%
Facilitates outflow and decreases aqueous production

Fewer side effects than epinephrine or pilocarpine

Fewer external side effects than epinephrine[2,3]
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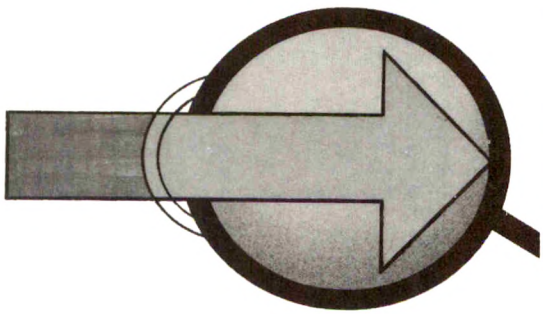
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- Concomitantly with timolol or pilocarpine
- Switch epinephrine and pilocarpine users to Propine
- Initial therapy: effective and safe choice for the new glaucoma patient.

Propine™ (dipivefrin HCl)... another advancement in research and development by the leader in ophthalmics.

References:
1. Mandell AI. et al. Dipivalyl epinephrine: A new prodrug in the treatment of glaucoma. Ophthalmology 1978 Mar;85(3):268-275.
2. Kohn AN. et al.Clinical comparison of dipivalyl epinephrine and epinephrine in the treatment of glaucoma. Am J Ophthalmol 1979;87:196-201.
3. Kass MA. et al. Dipivefrin and epinephrine treatment of elevated intraocular pressure: a comparative study. Arch Ophthalmol 1979 Oct;97(10):1865-1866.
4. Allergan Report Series No. 168, 1980.
5. Allergan Report Series No. 169, 1980.

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CONTAINS: Dipivefrin HCl*...0.1% with: benzalkonium chloride 0.004%; edetate disodium; mannitol; sodium chloride; sodium metabisulfite; hydrochloric acid to adjust pH and purified water. *Licensed under U.S. Patent Nos. 3,839,584 and 3,809,714. **INDICATIONS:** Propine™ (dipivefrin HCl) is indicated as initial therapy for the control of intraocular pressure in chronic open-angle glaucoma. Patients responding inadequately to other antiglaucoma therapy may respond to addition of Propine. In controlled and open label studies of glaucoma, Propine demonstrated a statistically significant intraocular pressure-lowering effect. Patients using Propine twice daily in studies with mean durations of 76-146 days experienced mean pressure reductions ranging from 20-24%. Therapeutic response to Propine twice daily is somewhat less than 2% epinephrine twice daily. Controlled studies showed statistically significant differences in lowering of intraocular pressure between Propine and 2% epinephrine. In controlled studies in patients with a history of epinephrine intolerance, only 3% of patients treated with Propine exhibited intolerance, while 55% of those treated with epinephrine again developed an intolerance. Therapeutic response to Propine twice daily therapy is comparable to 2% pilocarpine 4 times daily. In controlled clinical studies comparing Propine and 2% pilocarpine, there were no statistically significant differences in the maintenance of IOP levels for the two medications. Propine does not produce miosis or accommodative spasm which cholinergic agents are known to produce. The blurred vision and night blindness often associated with miotic agents are not present with Propine therapy. Patients with cataracts avoid the inability to see around lenticular opacities caused by constricted pupil. **CONTRAINDICATIONS:** Propine™ should not be used in patients with narrow angles since any dilation of the pupil may predispose the patient to an attack of angle-closure glaucoma. This product is contraindicated in patients who are hypersensitive to any of its components. **PRECAUTIONS: Aphakic Patients.** Macular edema has been shown to occur in up to 30% of aphakic patients treated with epinephrine. Discontinuation of epinephrine generally results in reversal of the maculopathy. **Pregnancy Category B.** Reproduction studies have been performed in rats and rabbits at daily oral doses up to 10 mg/kg body weight (5 mg/kg in teratogenicity studies), and have revealed no evidence of impaired fertility or harm to the fetus due to dipivefrin HCl. There are, however, no adequate and well-controlled studies in pregnant women. Because animal reproduction studies are not always predictive of human response, this drug should be used during pregnancy only if clearly needed. **Nursing Mothers.** It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when Propine is administered to a nursing woman. **Usage in Children.** Clinical studies for safety and efficacy in children have not been done. **Animal Studies.** Rabbit studies indicated a dose-related incidence of meibomian gland retention cysts following topical administration of both dipivefrin hydrochloride and epinephrine. **ADVERSE REACTIONS: Cardiovascular Effects.** Tachycardia, arrhythmias and hypertension have been reported with ocular administration of epinephrine. **Local Effects.** Burning and stinging was the most frequent side effect reported (6%) with Propine alone. Injection was observed in 6.5% of the patients. Epinephrine therapy can lead to adrenochrome deposits in the conjunctiva and cornea. **HOW SUPPLIED:** Propine (dipivefrin HCl) is supplied on prescription only as a 0.1% sterile solution in plastic dropper bottles in the following size: 10 ml — NDC 0023-0260-10.

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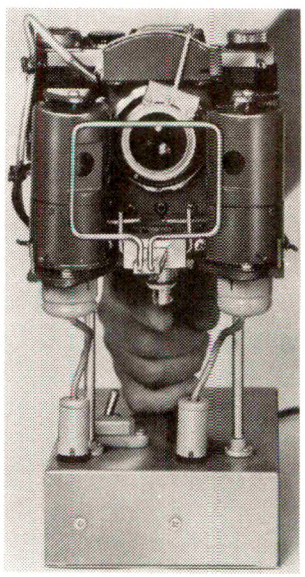
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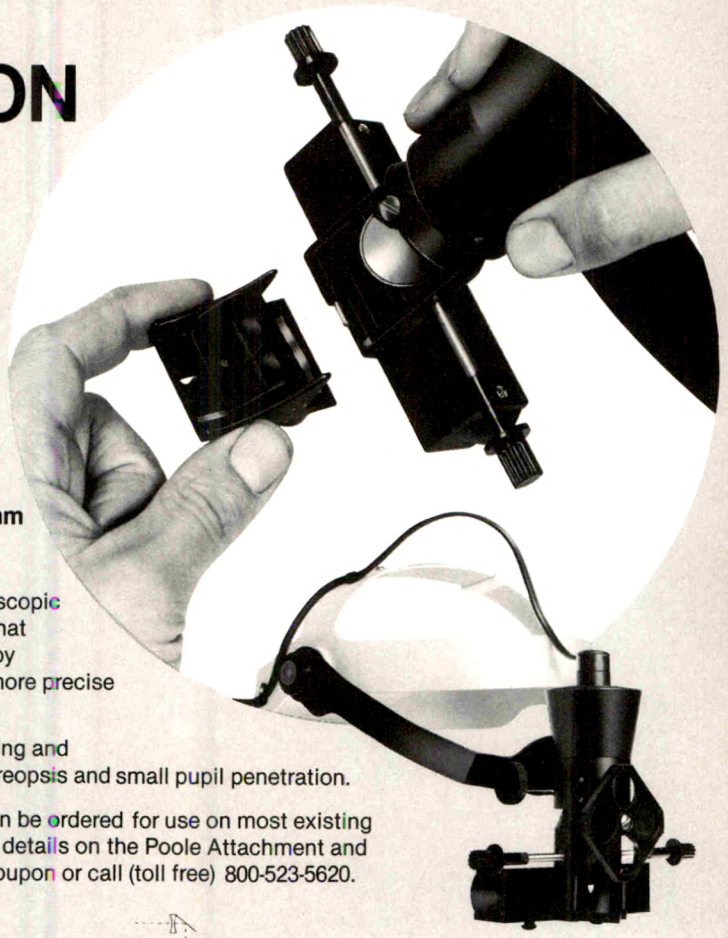
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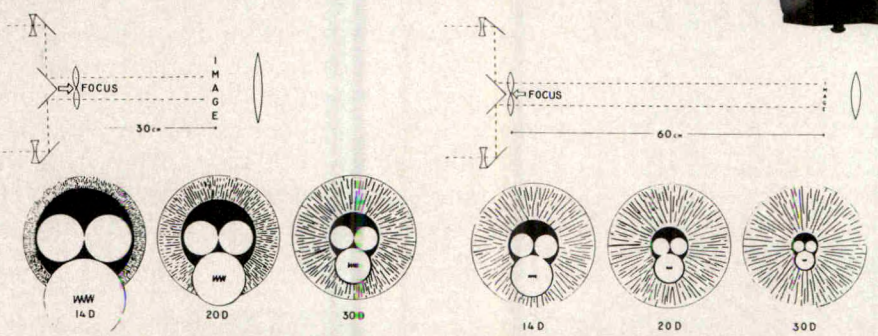


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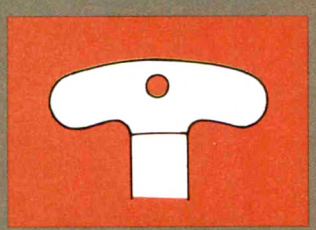


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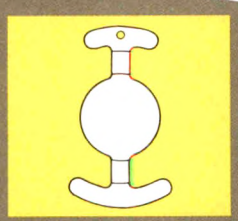
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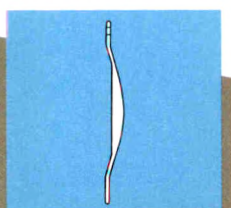
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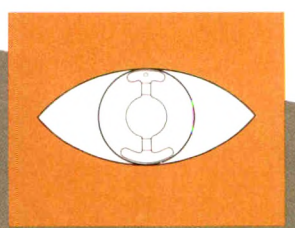
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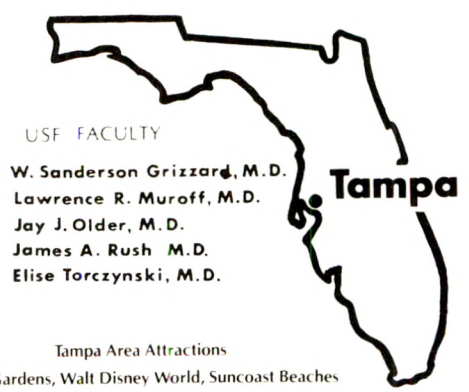
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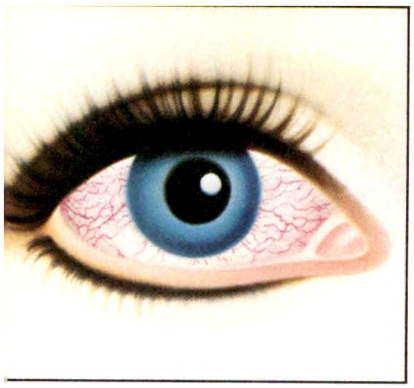
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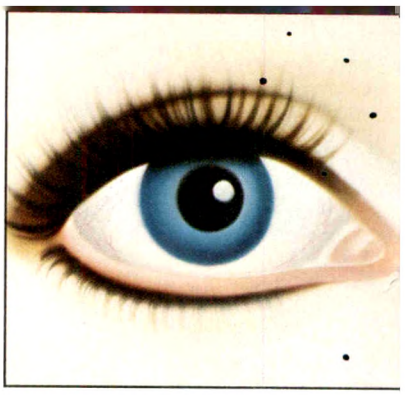
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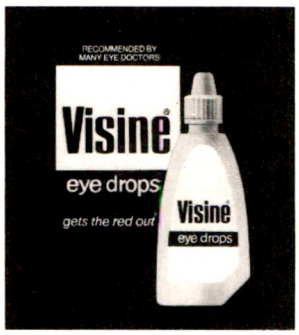
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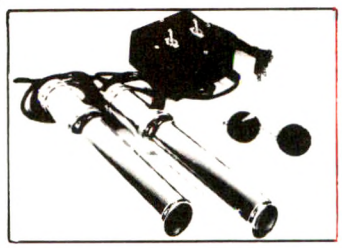
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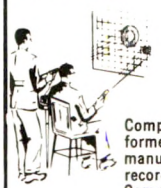
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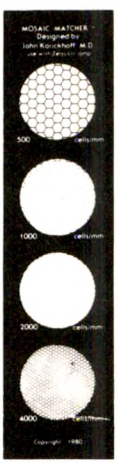
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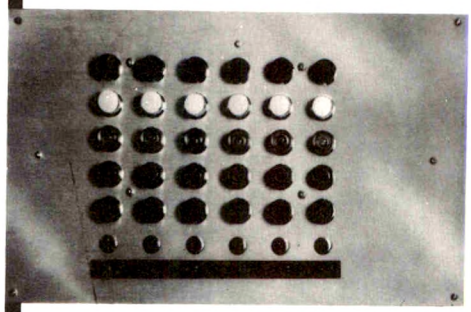
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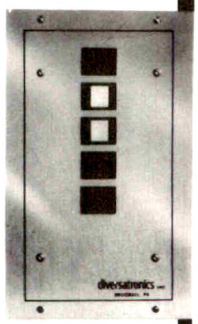
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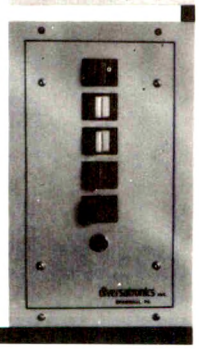
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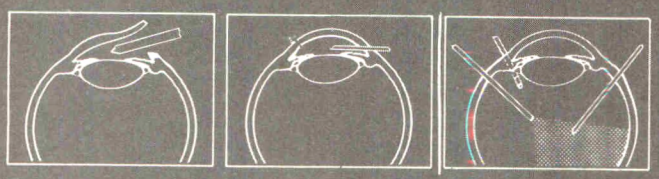
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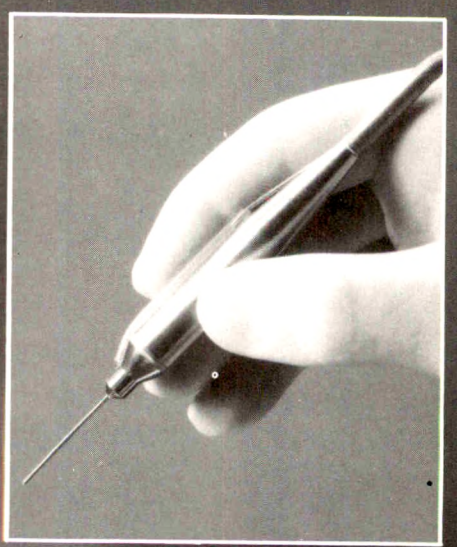
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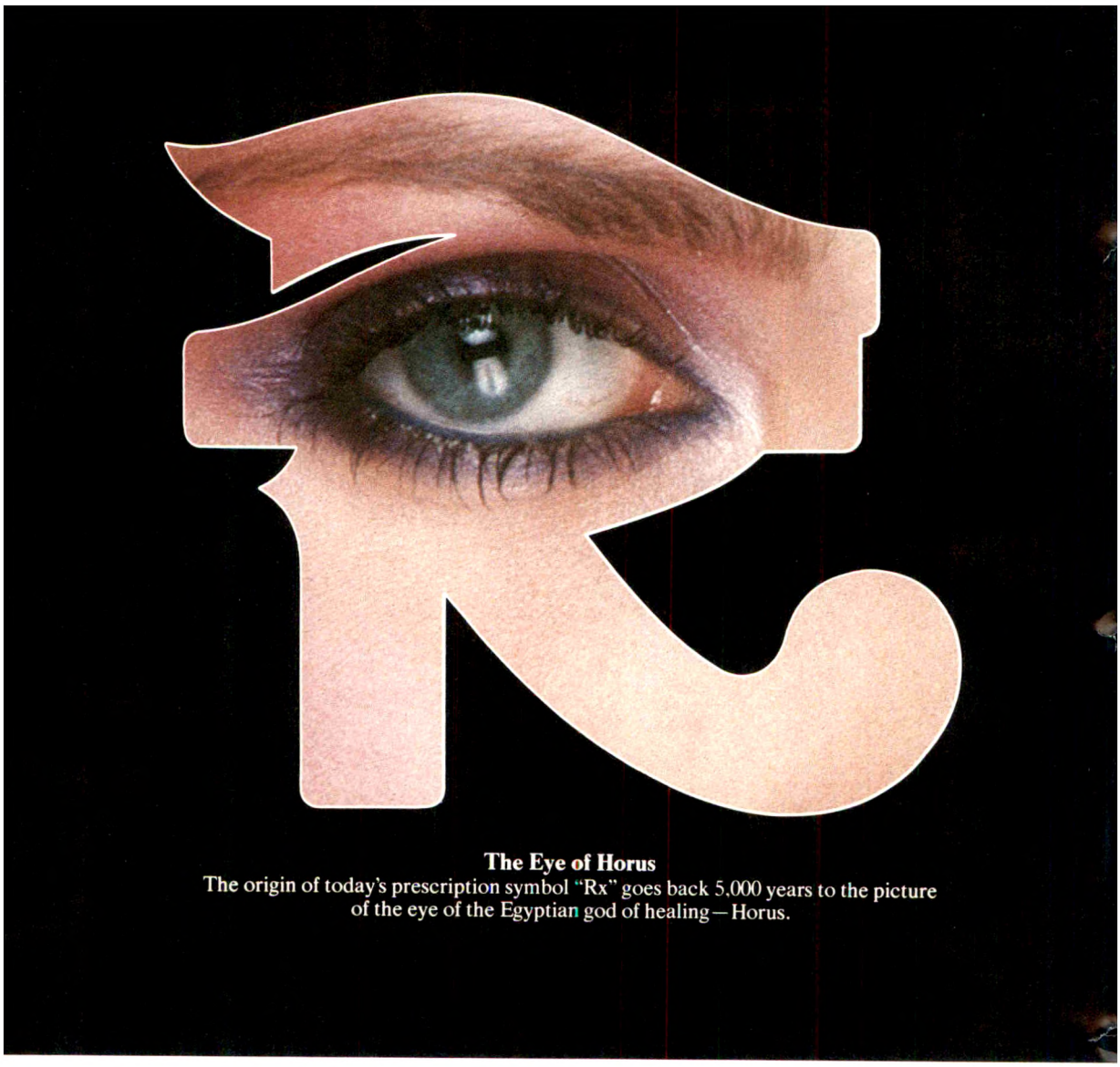
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The Eye of Horus
The origin of today's prescription symbol "Rx" goes back 5,000 years to the picture of the eye of the Egyptian god of healing—Horus.

DESCRIPTION: VIROPTIC® is the brand name for trifluridine (also known as trifluorothymidine, F_3TdR,F_3T), an antiviral drug for topical treatment of epithelial keratitis caused by Herpes simplex virus. The chemical name of trifluridine is 5-trifluoromethyl-2′ -deoxyuridine.

VIROPTIC sterile opthalmic solution contains 1% trifluridine in an aqueous solution with acetic acid and sodium acetate (buffers), sodium chloride, and thimerosal 0.001% (added as a preservative).

CLINICAL PHARMACOLOGY: Trifluridine is a fluorinated pyrimidine nucleoside with *in vitro* and *in vivo* activity against Herpes simplex virus, types 1 and 2 and vacciniavirus. Some strains of Adenovirus are also inhibited *in vitro*.

Intraocular penetration of trifluridine occurs after topical instillation of VIROPTIC into human eyes. Decreased corneal integrity or stromal or uveal inflammation may enhance the penetration of trifluridine into the aqueous humor. Unlike the results of ocular penetration of trifluridine *in vitro*, 5-carboxy-2′ -deoxyuridine was not found in detectable concentrations within the aqueous humor of the human eye.

INDICATIONS AND USAGE: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% is indicated for the treatment of primary keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus, types 1 and 2. VIROPTIC is also effective in the treatment of epithelial keratitis that has not responded clinically to the topical administration of idoxuridine or when ocular toxicity or hypersensitivity to idoxuridine has occurred. In a smaller number of patients found to be resistant to topical vidarabine, VIROPTIC was also effective.

The clinical efficacy of VIROPTIC in the treatment of stromal keratitis and uveitis due to Herpes simplex or ophthalmic infections caused by vacciniavirus and Adenovirus has not been established by well-controlled clinical trials. VIROPTIC has not been shown to be effective in the prophylaxis of Herpes simplex virus keratoconjunctivitis and epithelial keratitis by well-controlled clinical trials. VIROPTIC is not effective against bacterial, fungal or chlamydial infections of the cornea or nonviral trophic lesions.

During controlled multicenter clinical trials, 92 of 97 (95%) patients (78 of 81 with dendritic and 14 of 16 with geographic ulcers) responded to VIROPTIC therapy as evidenced by complete corneal re-epithelialization within the 14 day therapy period. In these controlled studies, 56 of 75 (75%) patients (49 of 58 with dendritic and 7 of 17 with geographic ulcers) responded to idoxuridine therapy. The mean time to corneal re-epithelialization for dendritic ulcers (6 days) and geographic ulcers (7 days) was similar for both therapies. In other clinical studies, VIROPTIC was evaluated in the treatment of Herpes simplex virus keratitis in patients who were unresponsive or intolerant to the topical administration of idoxuridine or vidarabine. VIROPTIC was effective in 138 of 150 (92%) patients (109 of 114 with dendritic and 29 of 36 with geographic ulcers) as evidenced by corneal re-epithelialization. The mean time to corneal re-epithelialization was 6 days for patients with dendritic ulcers and 12 days for patients with geographic ulcers.

CONTRAINDICATIONS: VIROPTIC brand Trifluridine Ophthalmic Solution, 1%, is contraindicated for patients who develop hypersensitivity reactions or chemical intolerance to trifluridine.

WARNINGS: The recommended dosage and frequency of administration should not be exceeded (see Dosage and Administration).

PRECAUTIONS:

General: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% should be prescribed only for patients who have a clinical diagnosis of herpetic keratitis.

VIROPTIC may cause mild local irritation of the conjunctiva and cornea when instilled but these effects are usually transient.

Although documented *in vitro* viral resistance to trifluridine has not been reported following multiple exposure to VIROPTIC, the possibility exists of viral resistance development.

For Topical Treatment of
Epithelial Keratitis Caused by Herpes Simplex Virus

VIROPTIC®

(Trifluridine)

OPHTHALMIC SOLUTION, 1%

STERILE

95% of Patients Responded*

During controlled clinical trials 95% of patients (92 of 97) responded to VIROPTIC therapy. Dendritic and geographic ulcers were re-epithelialized in a mean time of 6 and 7 days, respectively.

Significantly More Convenient

- Less frequent applications
- No middle of the night dosage
- None of the blurring and inconvenience of ointments

5 to 9 Drops Daily

One drop every 2 hours while awake until herpetic lesion has completely re-epithelialized, then one drop every 4 hours for 7 days post-re-epithelialization.



0 days



5 days after VIROPTIC therapy

Effective in 138 of 150 patients* Unresponsive or Intolerant to Idoxuridine or Vidarabine

In studies of patients unresponsive or intolerant to idoxuridine or vidarabine, VIROPTIC demonstrated 92% effectiveness. Dendritic and geographic ulcers were re-epithelialized in a mean time of 6 and 12 days, respectively.

*Data on file, Medical Division, Burroughs Wellcome Co.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Mutagenic Potential. Trifluridine has been shown to exert mutagenic, DNA damaging and cell transforming activities in various standard *in vitro* test systems, and clastogenic activity in *Vicia faba* cells.

Although the significance of these test results is not clear or fully understood, there exists the possibility that mutagenic agents may cause genetic damage in humans.

Oncogenic Potential. The oncogenic potential of trifluridine is unknown at this time. The oncogenic potential of trifluridine in rodents is being evaluated.

Pregnancy: The drug should not be prescribed for pregnant women unless the potential benefits outweigh the potential risks.

Nursing Mothers: It is unlikely that trifluridine is excreted in human milk after ophthalmic instillation of VIROPTIC because of the relatively small dosage (≤ 5.0 mg/day), its dilution in body fluids and its extremely short half-life (approximately 12 minutes). The drug should not be prescribed for nursing mothers unless the potential benefits outweigh the potential risks.

ADVERSE REACTIONS: The most frequent adverse reactions reported during controlled clinical trials were mild, transient burning or stinging upon instillation (4.6%) and palpebral edema (2.8%). Other adverse reactions in decreasing order of reported frequency were superficial punctate keratopathy, epithelial keratopathy, hypersensitivity reaction, stromal edema, irritation, keratitis sicca, hyperemia, and increased intraocular pressure.

OVERDOSAGE: Overdosage by ocular instillation is unlikely because any excess solution be quickly expelled from the conjunctival sac.

Acute overdosage by accidental oral ingestion of VIROPTIC has not occurred. However, should such ingestion occur, the 75 mg dosage of trifluridine in a 7.5 ml bottle of VIROPTIC is not likely to produce adverse effects. Single intravenous doses of 15-30 mg/kg/day in children and adults with neoplastic disease produce reversible bone marrow depression as the only potentially serious toxic effect and only after 3-5 courses of therapy. The acute oral LD_{50} in the mouse and rat was 4379 mg/kg or higher.

DOSAGE AND ADMINISTRATION: Instill one drop of VIROPTIC Ophthalmic Solution, 1% onto the cornea of the affected eye every two hours while awake for a maximum daily dosage of nine drops until the corneal ulcer has completely re-epithelialized. Following re-epithelialization, treatment for an additional seven days of one drop every four hours while awake for a minimum daily dosage of five drops is recommended.

If there are no signs of improvement after seven days of therapy, or complete re-epithelialization has not occurred after 14 days of therapy, other forms of therapy should be considered. Continuous administration of VIROPTIC for periods exceeding 21 days should be avoided because of potential ocular toxicity.

HOW SUPPLIED: VIROPTIC Ophthalmic Solution, 1% is supplied as a sterile ophthalmic solution in a plastic Drop-Dose® dispenser bottle of 7.5 ml. Store under refrigeration 2° to 8° C (36° to 46° F).

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ESTELLE DOHENY EYE FOUNDATION

PROGRESS IN VITREOUS SURGERY

Director Kenneth R. Diddie, M.D., February 20-21, 1981 • New Otani Hotel Los Angeles

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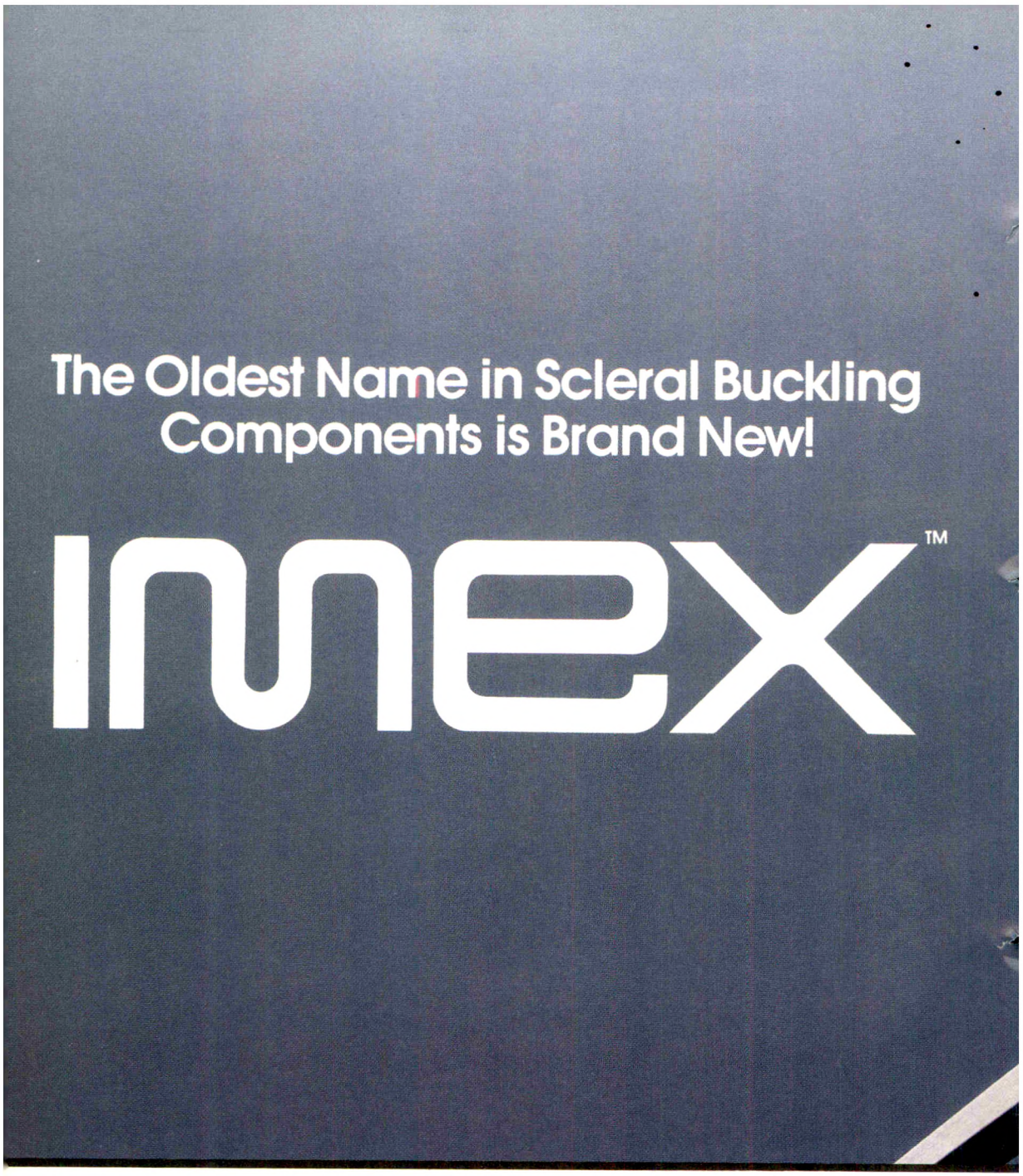
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12	13	14	15			

GUEST SPEAKERS

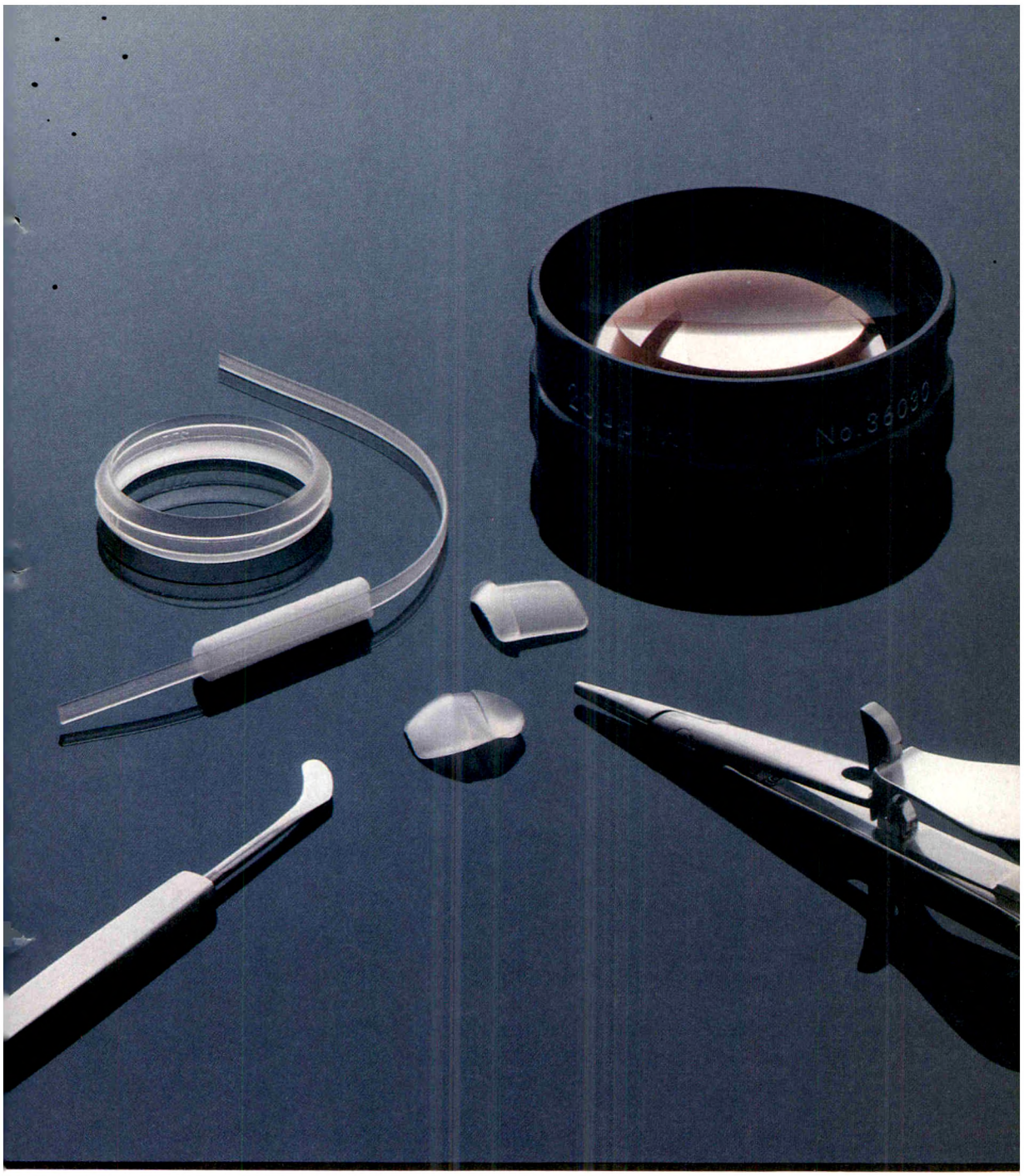
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PIERRE GUIBOR, M.D. – *New York, NY*
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Moderator: Hampson A. Sisler, M.D.
Speakers: Robert Chase, M.D., William Cooper, M.D., Robert Day, M.D., Robert Schimek, M.D., John T. Simonton, M.D., Stephen Trokel, M.D.

GLAUCOMA
Moderator: James E. Burris, M.D.
Speakers: Alfonse A. Cinotti, M.D., Max Forbes, M.D., G. Peter Halberg, M.D., Bernard Kronenberg, M.D., George Spaeth, M.D.

INTRA OCULAR LENSES
Moderator: John R. Finlay, M.D.
Speakers: Leeds E. Katzen, M.D., Larry G. Leiske, M.D., Steven P. Shearing, M.D.

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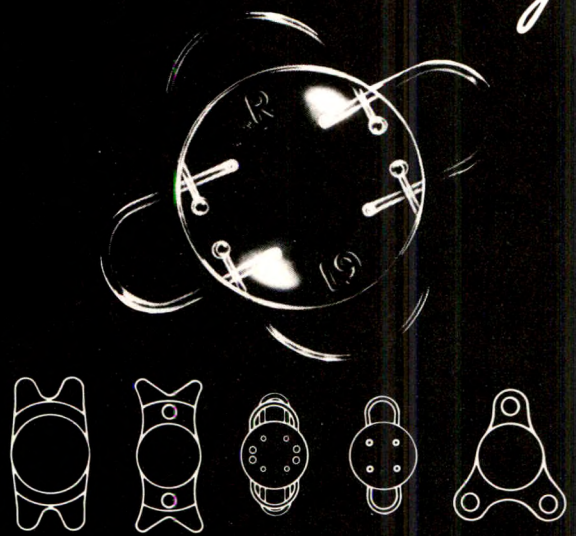
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THE XVII PALO ALTO COURSE ON DIAGNOSIS & TREATMENT OF RETINAL & CHOROIDAL DISEASES
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THE THREE-DAY COURSE WILL PRESENT:
. . . up-to-date information on retinal & choroidal diseases
. . . basic knowledge of retinal fluorescein angiography, flurophotometry, ultrasonography, & laser photocoagulation
. . . indications, results, & complications of photocoagulation
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Lloyd M. Aiello, M.D.—Joslin Clinic, Boston
Lawrence V. Basso, M.D.—Palo Alto Medical Clinic, Palo Alto
Devron Char, M.D.—University of California, San Francisco
Steven T. Charles, M.D.—University of Tennessee, Memphis
Peter Egbert, M.D.—Stanford University
Robert M. Ellsworth, M.D.—Cornell University, New York
Robert L. Jack, M.D.—Palo Alto Retinal Group & Stanford University
Francis A. L'Esperance, M.D.—Columbia University, New York
Hunter L. Little, M.D.—Palo Alto Retinal Group & Stanford University
A. Edward Maumenee, M.D.—John Hopkins Hospital, Baltimore
Irene Maumenee, M.D.—John Hopkins Hospital, Baltimore
G. Richard O'Connor, M.D.—Proctor Foundation, University of California, San Francisco
Stephen J. Ryan, M.D.—Estelle Doheny Eye Foundation, Los Angeles
Arthur Vassiliadis, Ph.D.—Zweng Memorial Retinal Research Foundation

ADVANCE REGISTRATION IS REQUIRED:

Course Fee: $400 (includes 2 lunches, buffet dinner, & the 4th Zweng Memorial Lecture Banquet)
Registration is payable to: Zweng Memorial Retinal Research Foundation
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Course Coordinator: (415) 323-0231
22 Hours Category 1 credit CMA's Continuing Education

THIRD ANNUAL DALLAS SPRING OPHTHALMOLOGY SYMPOSIUM
MARCH 13, 14, 15, 1981 FAIRMONT HOTEL DALLAS, TEXAS

VISITING FACULTY

Leo Bores, MD	Santa Fe	Marshall Parks, MD	Washington, DC
Edward Cotlier, MD	New Haven	Gary Rogers, MD	Columbus
David Hiles, MD	Pittsburgh	Ronald Smith, MD	Los Angeles
Herbert Kaufman, MD	New Orleans	Howard Slavin, J.D.	Los Angeles
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Priscilla Berry, MD	Everett Moody, MD
Gary Fish, MD	William Phelps, MD
Dwain Fuller, MD	William Snyder, MD
William Hutton, MD	David Stager, MD
John Lynn, MD	Albert Vaiser, MD

TOPICS

- REFRACTIVE SURGERY (Friday, March 13)
  Radial Keratotomy (Theory, Animal Studies, Technique, Human Results, Proposed Collaborative Study), Keratophakia, Keratomileusis, Epikeratophakia, Intraocular Lenses
- PEDIATRIC ANTERIOR SEGMENT SURGERY (Saturday, March 14)
  New Approaches to Congenital Cataracts, Subluxated Lenses, Secondary Membranes, Anterior Vitrectomy, Management of Pediatric Ocular Trauma
- UPDATE ON RETROLENTAL FIBROPLASIA (Sunday, March 15)
  Historical Perspectives, Differential Diagnosis, Medical Treatment, Surgical Treatment, Medical-Legal Implications

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For information please write: The Eye Foundation (AFORE)
421 North 18th Street, Suite 105
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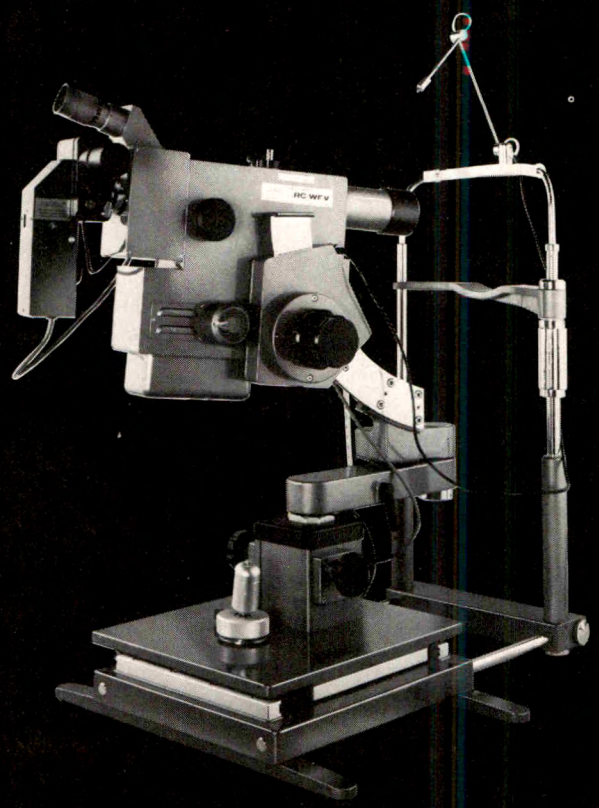
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TOPICS

- ☐ All basic state of the art concepts and methods relative to currently available contact lenses, hard/soft/gas permeable
- ☐ Basic methods and general overview of modern techniques in refractive keratoplasty
- ☐ Perspectives for future developments in contact lens technology/refractive keratoplasty
- ☐ Overview of alternative methods eg. ortho keratology
- ☐ Didactic lectures and hands on workshops
- ☐ Basic and advanced contact lens assistance course/workshop

FACULTY

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PERRY S. BINDER, M.D.
San Diego, California

DAVID S. CHASE, M.D.
Burlington, Vermont

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Solution-Sterile
Ointment-Sterile

Each ml or gram contains gentamicin sulfate equivalent to 3.0 mg gentamicin

- **Broad-spectrum activity against many gram-negative and gram-positive oculopathogens (see prescribing information below) frequently implicated in commonly seen infections of the external eye and its adnexa.**
- **Single-entity antibiotic.**
- **Generally low potential for sensitivity reactions and irritation.**

DESCRIPTION Gentamicin sulfate is a water-soluble antibiotic of the aminoglycoside group active against a wide variety of pathogenic gram-negative and gram-positive bacteria.

GARAMYCIN **Ophthalmic Solution** is a sterile, aqueous solution buffered to approximately pH 7 for use in the eye. Each ml contains gentamicin sulfate (equivalent to 3.0 mg gentamicin), disodium phosphate, monosodium phosphate, sodium chloride, and benzalkonium chloride as a preservative.

GARAMYCIN **Ophthalmic Ointment** is a sterile ointment, each gram containing gentamicin sulfate (equivalent to 3.0 mg gentamicin) in a bland base of white petrolatum, with methylparaben and propylparaben as preservatives.

ACTIONS The gram-positive bacteria against which gentamicin sulfate is active include coagulase-positive and coagulase-negative staphylococci, including certain strains that are resistant to penicillin. Group A beta-hemolytic and nonhemolytic streptococci, and *Diplococcus pneumoniae*. The gram-negative bacteria against which gentamicin sulfate is active include certain strains of *Pseudomonas aeruginosa*, indole-positive and indole-negative *Proteus* species, *Escherichia coli*, *Klebsiella pneumoniae* (Friedlander's bacillus), *Haemophilus influenzae* and *Haemophilus aegyptius* (Koch-Weeks bacillus), *Aerobacter aerogenes*, *Moraxella lacunata* (diplobacillus of Morax-Axenfeld), and *Neisseria* species, including *Neisseria gonorrhoeae*. Although significant resistant organisms have not been isolated from patients treated with gentamicin at the present time, this may occur in the future as resistance has been produced with difficulty *in vitro* by repeated exposures.

INDICATIONS GARAMYCIN **Ophthalmic Solution** and **Ointment** are indicated in the topical treatment of infections of the external eye and its adnexa caused by susceptible bacteria. Such infections embrace conjunctivitis, keratitis and keratoconjunctivitis, corneal ulcers, blepharitis and blepharoconjunctivitis, acute meibomianitis, and dacryocystitis.

CONTRAINDICATIONS GARAMYCIN **Ophthalmic Solution** and **Ointment** are contraindicated in patients with known hypersensitivity to any of the components.

WARNINGS GARAMYCIN **Ophthalmic Solution** is not for injection. It should never be injected subconjunctivally, nor should it be directly introduced into the anterior chamber of the eye.

PRECAUTIONS Prolonged use of topical antibiotics may give rise to overgrowth of nonsusceptible organisms, such as fungi. Should this occur, or if irritation or hypersensitivity to any component of the drug develops, discontinue use of the preparation and institute appropriate therapy.

Ophthalmic ointments may retard corneal healing.

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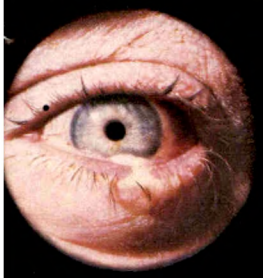
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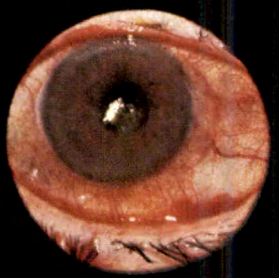
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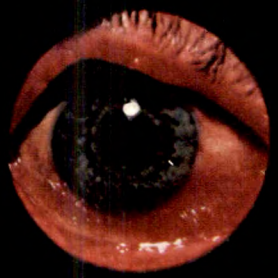
Acute catarrhal conjunctivitis with episcleritis. Etiology: S. aureus. Before treatment.



Corneal ulcer. Etiology: S. aureus. Before treatment.



Acute hemorrhagic conjunctivitis. Etiology: Pneumococcus. Before treatment.

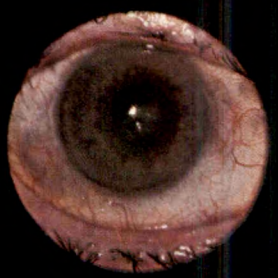


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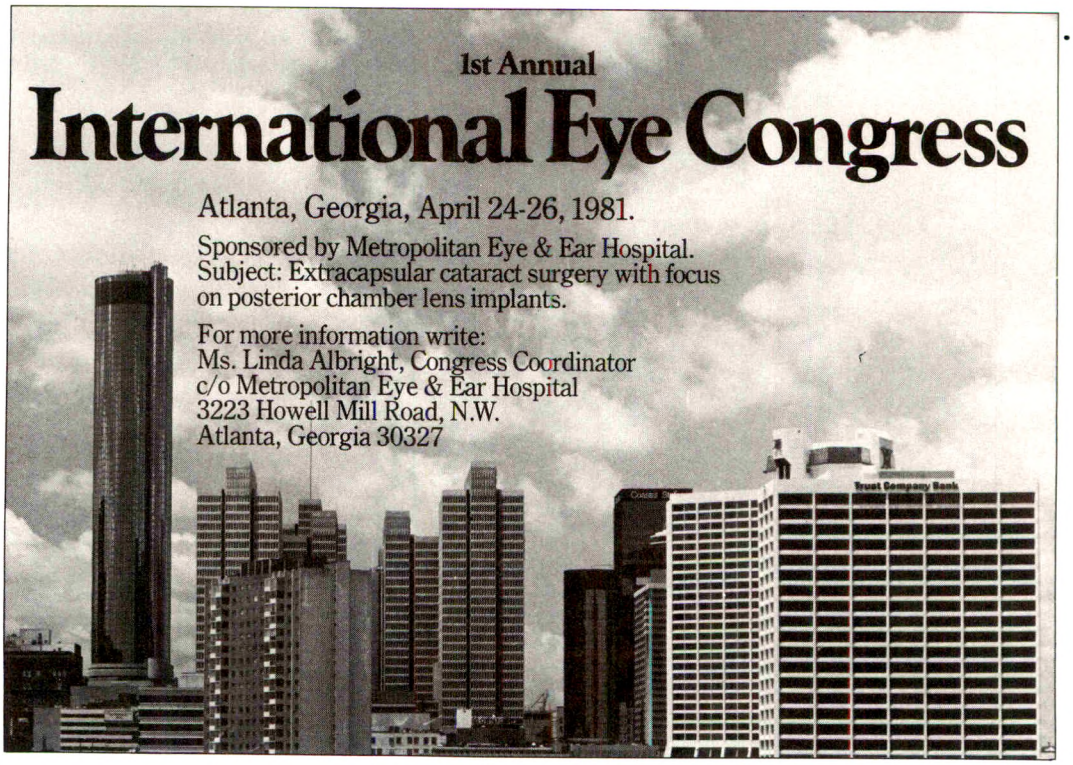
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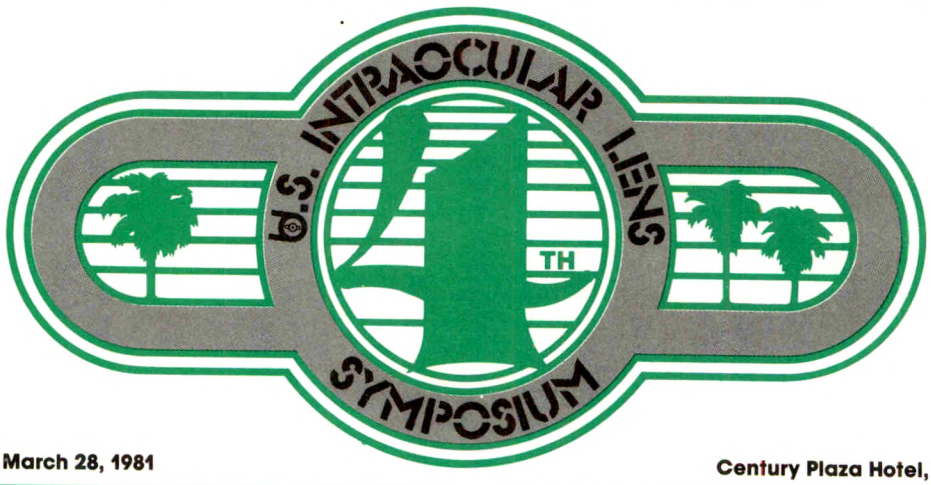
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Format:

This year we will offer courses on Wednesday, March 25 only. They will consist of 3 separate 2 hour sessions, giving you the opportunity to attend 3 different courses in one day, as well as view the latest products and scientific developments of our exhibitors. Thursday, Friday, and Saturday will be devoted to lecture & symposia. No old repetitious material or "my lens" panels will be allowed.

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Courses:

Complications and IOLs

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Extracapsular Techniques

Endothelial Photography

Anterior Chamber IOLs—
4 hour session

Posterior Chamber IOLs—
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Symposia:

In Defense of Intracapsular Surgery

Long Term Effect of IOLs on Endothelial Cell Counts

The Posterior Capsule in Pseudophakia

Delivery of the Nucleus

Clinical Pathological Conferences.

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25 Hours: CME-AMA Credit Category 1

Nurses & Technicians Program:

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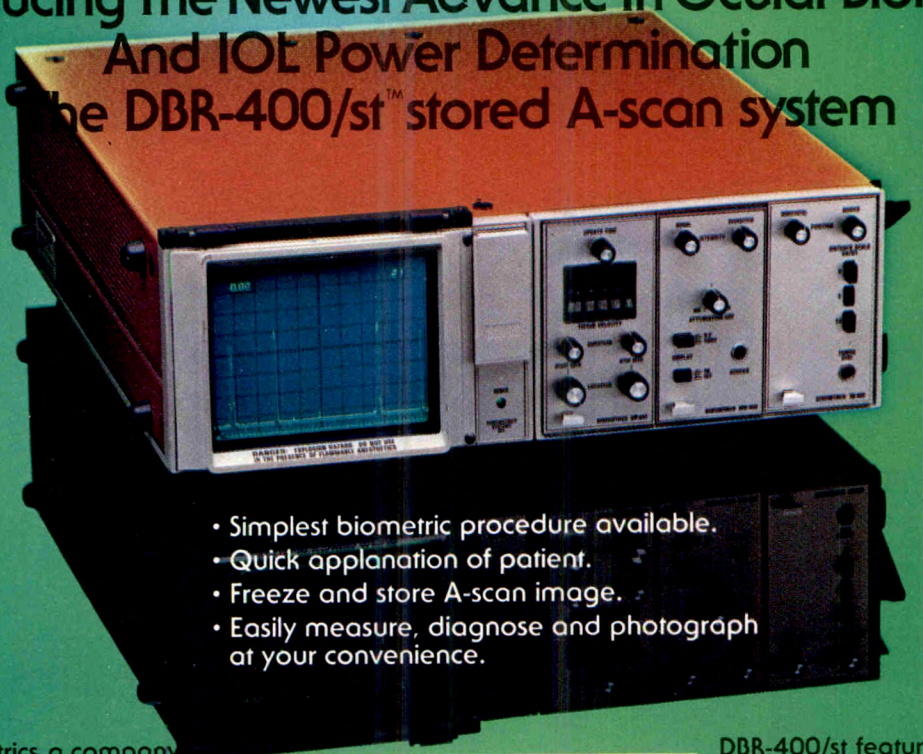
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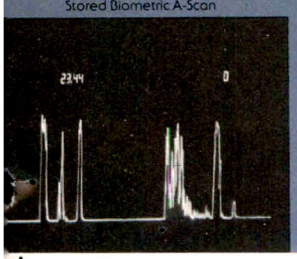
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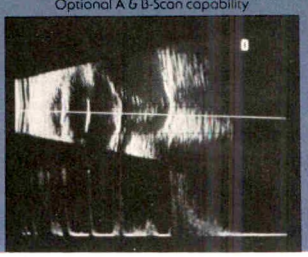


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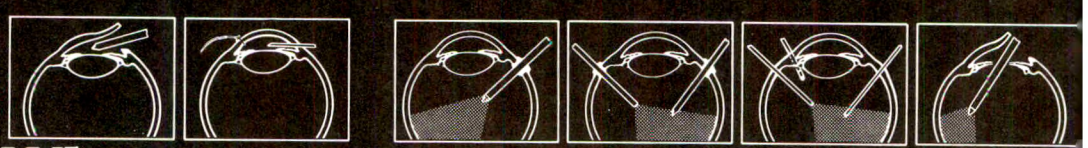
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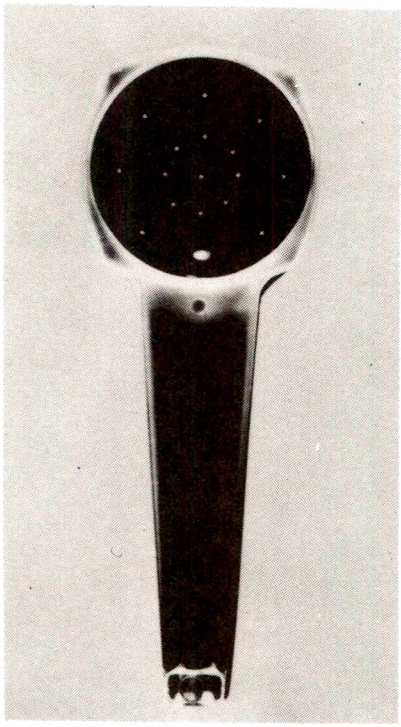
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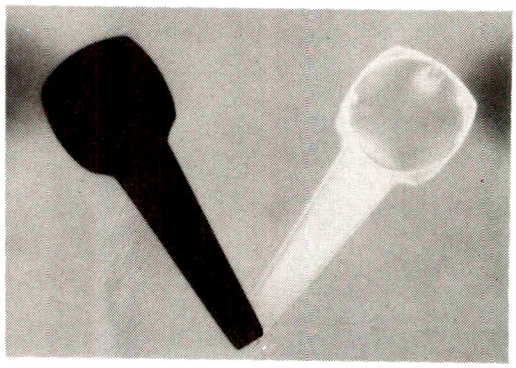
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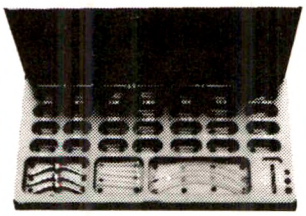
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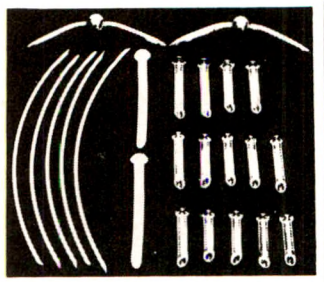
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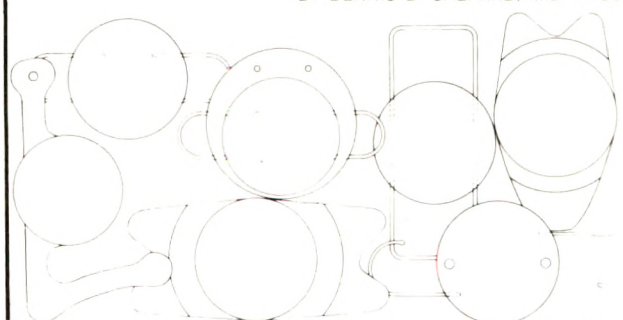
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HETEROGENEITY OF RETINAL DEGENERATION AND HEARING IMPAIRMENT SYNDROMES

J. BRONWYN BATEMAN, M.D., ERWIN D. RIEDNER, SC.D., L. STEFAN LEVIN, D.D.S., AND IRENE H. MAUMENEE, M.D.

Baltimore, Maryland

Pigmentary retinopathy associated with hearing loss occurs both in genetic disorders and as a result of environmental factors. Genetic conditions with this combination of abnormalities include Usher's, Alström's, Refsum's, Cockayne's, and Alport's syndromes. Rubella embryopathy, an intrauterine environmental insult, may also cause degenerative pigmentary retinopathy and hearing loss.

Usher's syndrome (retinitis pigmentosa and hearing loss) is an autosomal recessive disorder; its genetic heterogeneity, however, has been proposed but not well documented.[1-4] Conversely, the cause of the syndrome of macular pigment epitheliopathy with normal vision and hearing loss, independently described by Diallinas[5] and Amalric,[6] is less clear; genetic factors and rubella embryopathy have been implicated.[7-10]

Careful study of individual cases and families is valuable in delineating disorders before defining the underlying biochemical defects.

SUBJECTS AND METHODS

We selected nine patients with progressive retinal degeneration and hearing loss and four patients with stable maculopathy and hearing loss in order to assess the heterogeneity of syndromes with retinal abnormalities and hearing impairment (Table 1). All patients underwent an ophthalmologic examination, including electroretinogram, electrooculogram, perimetry (Goldmann), and a dental examination. Audiologic evaluation included pure-tone air- and bone-conduction tests, speech-reception thres-

TABLE 1

CLASSIFICATION OF PATIENTS WITH RETINAL AND AUDIOLOGIC ABNORMALITIES

Classification	Case No.
Progressive retinal degeneration with hearing loss	
Classic Usher's syndrome	1-5
Crystalline retinopathy	6
Retinitis pigmentosa, hearing loss, and enamel dysplasia	7
Clumped pigmentary retinopathy and hearing loss	8 and 9
Stable maculopathy with hearing loss	
Amalric-Diallinas syndrome	10-12
Maculopathy, hearing loss, and enamel dysplasia	13

From the Departments of Ophthalmology (Drs. Bateman and Maumenee) and Otolaryngology (Drs. Riedner and Levin), Johns Hopkins University School of Medicine and Hospital, Baltimore, Maryland. This study was supported in part by Research Career Development Award K04 DE00021 from the National Institutes of Health (Dr. Levin) and research grant R01 01773 from the National Eye Institute (Dr. Maumenee). Dr. Bateman was a Heed Foundation Fellow (1979).

Reprint requests to Irene H. Maumenee, M.D., Wilmer Eye Institute, Room 111, Johns Hopkins Hospital, Baltimore, MD 21205.

TABLE 2

SUMMARY OF HISTORY AND PHYSICAL EXAMINATIONS

Case, Age at Examination (yrs), Sex	Onset of Nyctalopia (Age in yrs)	Central Vision	Consanguinity	Family History*	Best Corrected Visual Acuity	
					Right Eye	Left Eye
1, 38, F	3	Stable	No	1 brother, affected (E); mother, drusen; father, unaffected (H)	6/6 (20/20)	6/12 (20/40)
2, 37, M	25	Stable since age 20 yrs	No	1 sister, affected (E); mother, drusen; father, unaffected (H)	HM	LP
3, 18, M	3	Stable	No	2 brothers, 2 sisters, unaffected (H); 1 sister affected (H); parents, unaffected (H)	6/15 (20/50)	6/22 (20/70)
4, 18, F	16	Stable	No	2 sisters, unaffected (H); parents, unaffected (H)	6/7.5 (20/25)	6/6 (20/20)
5, 45, M	3	Declining since age 35 yrs	Parents	3 brothers, 3 sisters, unaffected (H); 2 brothers affected (H); parents, unaffected (H)	HM	CF
6, 37, F	25	Stable	No	1 sister, unaffected (H); parents, unaffected (H)	6/9 (20/30)	6/7.5 (20/25)
7, 15, M	None	Stable	No	1 half-sib, unaffected (E); father, unaffected (E); mother, nystagmus	6/240 (5/200)	3/60 (8/200)
8, 11, M	3	Not testable	No	1 brother, affected (E); parents, unaffected (E)	1/60 (3/200)	1/50 (4/200)

*E, by examination: H, by history.
†Vitreous cells assessed on a scale of 1 (rare) to 4 (many).

Refractive Error					
Right Eye	Left Eye	Lens	Vitreous[†]	Fundus Abnormalities	Teeth
−1.00	−1.25	Posterior subcapsular cyst	1+	Pale optic nerve head; arteriolar narrowing; pigmentary bone spicule formation; macula, mild pigment granularity	Normal
Aphakia	Aphakia	Absent	1+	Pale optic nerve head; arteriolar narrowing, pigmentary bone spicule formation; macular pigment hyperplasia	Normal
Plano	+0.50	Posterior subcapsular cyst	2+ cells	Pale optic nerve head; arteriolar attenuation; generalized pigment granularity, perivascular pigment clumping of periphery	Normal
−0.25	−0.25	Clear	Condensation	Slightly pale optic nerve head; arteriolar narrowing; pigment granularity of periphery	Normal
—	—	R.E.: absent, L.E.: posterior subcapsular cyst	2+ cells	Slightly pale optic nerve head; arteriolar narrowing; pigment granularity of periphery	Normal
−1.00	+0.50	Clear	1+	Minimal arterior narrowing; pigmentary bone spicule formation mid periphery; crystalline deposits throughout entire retina except macula	Normal
+2.75	+3.50	Few nuclear opacities	1+	Moderately pale optic nerve head; anteriolar narrowing; bone spicule formation mid-periphery; macula, minimal pigmentary alterations	Enamel dysplasia; delayed eruption, congenital absence of lower 2nd premolars, only 4 permanent posterior teeth occlude
Not testable	Not testable	Cortical lens opacities	Unknown	Large atrophic macular lesion with pigmentary dropout; large circular/oval pigment epithelial groupings in periphery and posterior pole except macula	Enamel dysplasia (probably environmental)

Table 2 continued on next pages

TABLE 2 *(Continued)*

SUMMARY OF HISTORY AND PHYSICAL EXAMINATIONS

Case, Age at Examination (yrs), Sex	Onset of Nyctalopia (Age in yrs)	Central Vision	Consanguinity	Family History*	Best Corrected Visual Acuity	
					Right Eye	Left Eye
9, 5, M	4	Stable	No	1 brother, affected (E); parents, unaffected (E)	3/9 (10/30)	3/9 (10/30)
10, 18, F	None	Stable	No	2 brothers, 2 sisters, unaffected (H); parents, unaffected (H)	6/24 (20/80)	6/6 (20/20)
11, 20, M	None	Stable	No	4 brothers, unaffected (H); parents, unaffected (H)	6/4.5 (20/15) (25 Δ esotropia, amblyopia)	6/9 (20/30)
12, 17, F	None	Stable	No	1 sister, unaffected (E); mother, unaffected (E); father, unaffected (H)	6/6 (20/20)	6/6 (20/20)
13, 33, F	None	Stable	No	2 sisters, unaffected (E); father, drusen; mother, asteroid hyalosis	6/30 (20/100)	6/30 (20/100)

*E, by examination: H, by history.
†Vitreous cells assessed on a scale of 1 (rare) to 4 (many).

hold, speech-discrimination assessment, impedance audiometry (tympanograms and acoustic reflex tests), and, when possible, short-increment sensitivity index and tone decay.

RESULTS

Patients 1 and 2 were siblings aged 38 and 37 years at the time of the study (Tables 2 and 3). Both had retinitis pigmentosa and hearing loss. Patient 1 had had nyctalopia since she was approximately 3 years old; conversely, patient 2 noted nyctalopia at the age of 25 years. Patient 2 had a visual acuity of R.E.: hand motion and L.E.: light perception at the time of examination. His sister had a visual acuity of R.E.: 6/6 (20/20) and L.E.: 6/12 (20/40) at approximately the same age. On audiometric evaluation both patients had moderate-to-severe[11] sensorineural hearing loss which was more pronounced in the higher frequencies. Speech discrimination was normal. The hearing loss was congenital and stable in both patients. Their teeth were normal.

Patients 3, 4, and 5 had retinitis pigmentosa. Profound hearing loss began before the age of 3 years and they had no speech discrimination. We found no dental abnormalities.

Patient 6 had had nyctalopia since her mid-20s. On ophthalmoscopic examina-

Refractive Error					
Right Eye	Left Eye	Lens	Vitreous†	Fundus Abnormalities	Teeth
Not testable	Not testable	Clear	Clear	Large circular/oval pigment epithelial groupings of periphery and posterior pole; minimal venous tortuosity	Normal
+5.00	+0.50	Clear	2+ cells	Macula, dull reflex with pigmentary clumping and irregularities	Normal
Plano	Plano	Clear	Clear	Macula, dull reflex with pigmentary clumping and irregularities	Normal
Plano	Plano	Clear	Clear	Macula, dull reflex with pigmentary clumping and irregularities	Normal
−3.00	−3.00	Clear	1+	Slightly pale optic nerve head; fine pigmentary clumping throughout retina; macula, metallic sheen with pigmentary mottling and prominent choridal vasculature	Enamel dysplasia; delayed eruption

tion she had peripheral crystalline retinopathy (Fig. 1). Electrophysiologic results supported the diagnosis of retinitis pigmentosa. She had moderate-to-severe sensorineural hearing loss and excellent speech discrimination. Serial audiograms showed that the hearing deficit, which had existed since early in life according to her parents and which was documented when she was 10 years old, was stable. Dentition was normal.

Case 7 represents, to the best of our knowledge, an undescribed combination of classic retinitis pigmentosa, hearing loss, and enamel dysplasia. Despite typical retinal degeneration and an absent electroretinographic response, the patient denied nyctalopia. There was minimal pigment granularity of the macula. There was a marked nonspecific defect on the Farnsworth-Munsell test. We found severe sensorineural hearing loss with reduced speech discrimination. The hearing loss was diagnosed before he was 2 years old and was stable thereafter. The severe hearing loss precluded performance of a dark-adaptation profile. The tooth surfaces were tapered, yellow, hard, and rough; on radiographs, the enamel was thinner than normal (Fig. 2).

Patients 8 and 9 were brothers who had nyctalopia with an unusual clumped pigmentary retinopathy of the periphery (Fig. 3, left). The clumped pigment areas

TABLE 3

RESULTS OF LABORATORY EXAMINATIONS

Case	Electro-oculogram		Electroretinogram*	Visual Fields†	
	Right Eye	Left Eye		Right Eye	Left Eye
1	1.18	1.24	P/S, no response	15	10
2	Not testable	Not testable	P/S, no response	Not testable	Not testable
3	1.32	1.24	P/S, no response	5	10
4	1.41	1.41	P/S, no response	30	20
5	Not testable	Not testable	P/S, no response	Not testable	Not testable
6	1.33	1.20	P/S, no response	30	35
7	Not testable	Not testable	R.E.: P/S, no response	Not testable	Not testable
8	Not testable	Not testable	Not testable	Not testable	Not testable
9	Not testable	Not testable	No rod response; cone response	Not testable	Not testable
10	2.79	2.16	P/S, no response	—	Full
11	2.71	1.94	P/S, no response	Full	Full
12	2.15	2.44	P/S, no response	Full	Full
13	1.11	1.36	S, normal; P, R.E.: 91%, L.E.: 89%	Enlarged blind spot; mid-peripheral scotoma	

*P, photopic conditions; S, scotopic conditions.
†Goldmann perimetry to I_4 isopter.

ranged in size from the diameter of a vein at the margin of the optic nerve head to 1 disk diameter. Patient 8 had clumped pigmentary retinopathy of the periphery with an atrophic macular lesion. He also had moderate psychomotor retardation. Patient 9 had had no pigment deposits when he was examined by an ophthalmologist at the age of 2 years. When he was 5 years old, he was found to have a clumped pigmentary retinopathy of the periphery (Fig. 3, left) with sparing of the macula (Fig. 3, right). The optic nerve head had a normal pink color and the veins were minimally tortuous. Electroretinography showed no rod response and a normal cone response. Hearing loss had been diagnosed in both patients before they were 2 years old, and was moderate-to-severe, sensorineural, and stable in both. Patient 8 also had a generalized enamel dysplasia, probably the result of his unexplained malabsorption syndrome early in life; the enamel was thin, hard, yellow and irregular. His brother, patient 9, had normal enamel and normal phytanic acid and lipid profiles.

Patients 10, 11, and 12 had bilateral macular pigment epitheliopathy with normal visual acuity and bilateral congenital profound hearing loss similar to that of Amalric-Diallinas syndrome. The macular lesions of all three patients were similar and showed a localized, prominent granularity of the pigment epithelium (Fig. 4). The retinal periphery was minimally involved by the process. There was no history of prenatal rubella in any of the three patients or of macular degeneration with hearing loss in their families. Patient 11 had unexplained seizures. On electrophysiologic testing, we found an altered electro-oculogram and a normal



Fig. 1 (Bateman and associates). Case 6, right eye. Peripheral view of fundus shows many reflective crystalline-like deposits, arteriolar narrowing, and perivascular pigment aggregates (× 2.5).

electroretinogram. There were no dental abnormalities.

Patient 13 had stable nonexudative maculopathy without flecks. The macular region showed generalized dropout of the pigment epithelium with mild granularity. The choroidal vasculature was prominent (Fig. 5). The retinal periphery showed minimal pigment granularity. The electroretinogram was minimally al-

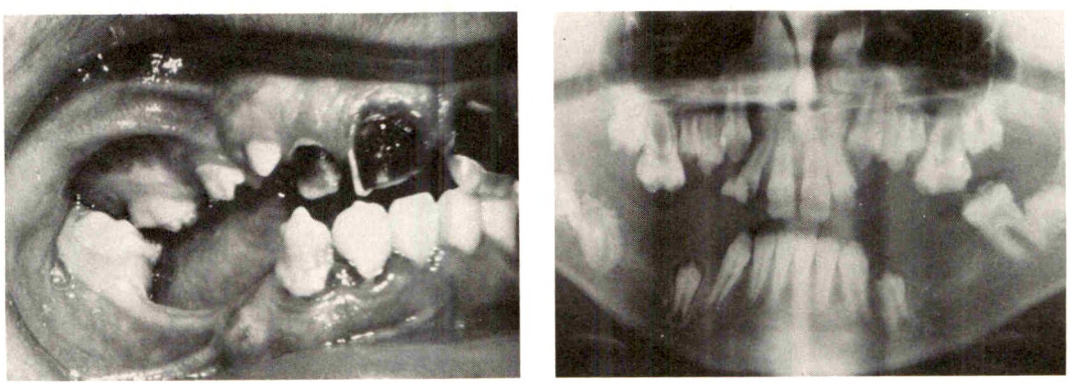


Fig. 2 (Bateman and associates). Case 7. Left, Enamel surfaces are dysplastic. Dark areas on labial surfaces of incisor teeth are dental caries. Right, Panoramic radiograph of the jaws. Enamel is thinner than normal. Radiolucent areas on crowns of maxillary incisors and lower molar are dental caries.

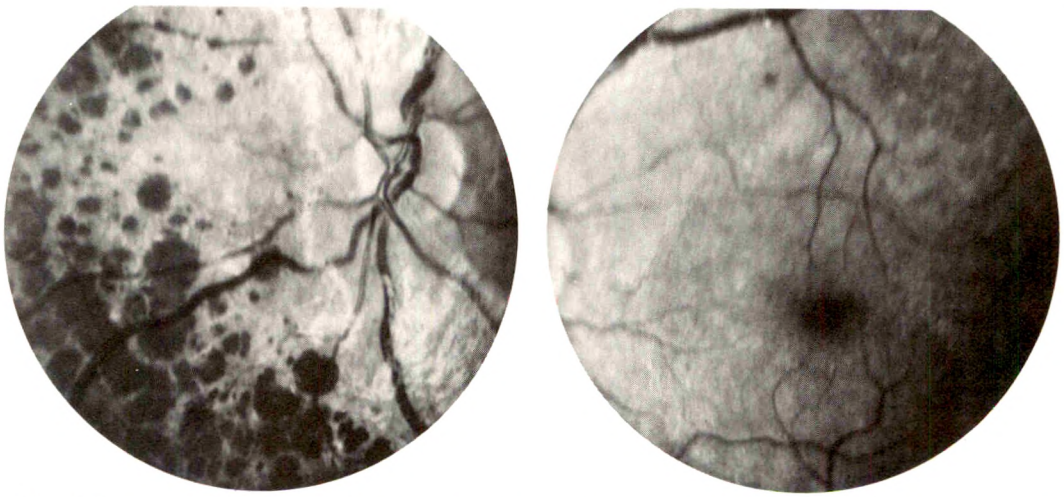


Fig. 3 (Bateman and associates). Case 9. Left, Right eye. Optic nerve and nasal retina show many areas of clumped pigment varying in size from a vein crossing the disk margin to 1 disk diameter. The optic nerve head is normal in color and the veins are minimally tortuous (×5). Right, Left eye. Macula and region within the vascular arcade show no clumped retinopathy (×5).

tered, and the electro-oculogram was markedly depressed. Hearing loss was moderate-to-severe and sensorineural; she had good speech discrimination. There was no evidence that her hearing had deteriorated since discovery of her impairment when she was less than 5 years old. The enamel was dark brown, irregular, and hard on some teeth (Fig. 6). On dental radiographs, the crown surfaces had a motheaten appearance.

Discussion

Usher's syndrome—Retinitis pigmentosa associated with hearing loss was first described by von Graefe[12] and later delineated by Usher.[13] Bell,[14] von Wilbaut,[15] Linenov,[16] and Nuutila[1] emphasized the

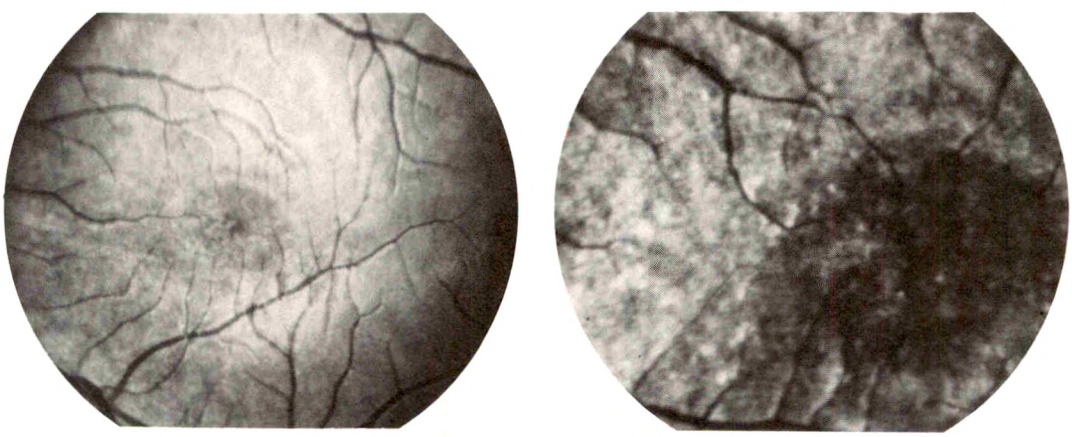


Fig. 4 (Bateman and associates). Case 10, left eye. Macula and region within the vascular arcade show localized marked granularity of the pigment epithelium (×2.5). Right, Higher-power view shows prominent granularity of pigment epithelium (×5).

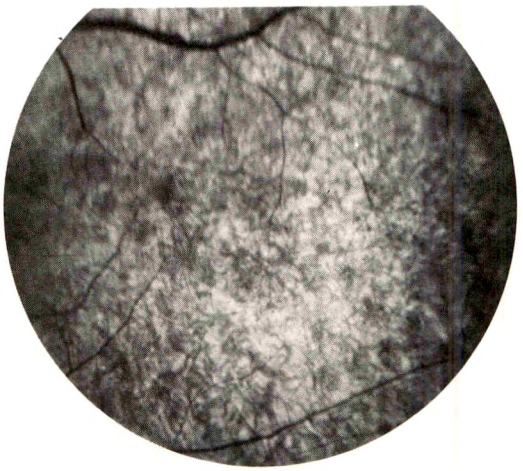


Fig. 5 (Bateman and associates). Case 13. Macula and region within the vascular arcade show generalized dropout of the pigment epithelium with mild granularity. The choroidal vasculature is prominent (×5).

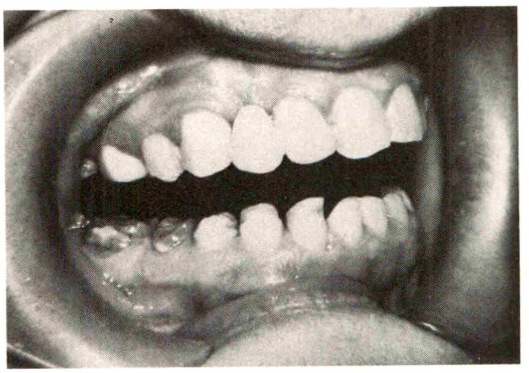


Fig. 6 (Bateman and associates). Case 13. Enamel surfaces of the lower teeth are dysplastic. Several maxillary teeth have been covered with artificial crowns.

autosomal recessive pattern of inheritance. Hallgren[17] noted that many patients with this disorder also had vestibulocerebellar ataxia and psychoses.

Several investigators have suggested that Usher's syndrome is genetically heterogeneous.[1-4] Nuutila[1] described 137 patients over 10 years of age who had pigmentary retinal dystrophy and sensorineural hearing impairment. Of these patients, 92% had a history of nyctalopia before the age of 10 years; the rest experienced the onset of symptoms after 20 years of age. Nuutila suggested genetic heterogeneity based on the age at onset of nyctalopia. Additionally, he noted four patients with progressive hearing impairment and retinitis pigmentosa, suggesting a third disorder; he did not state whether the progression of hearing loss had been documented by serial audiograms. He also noted intrafamilial constancy in the degree of hearing loss. There was no evidence of associated central nervous system disorders.

Merin, Abraham, and Auerbach[2] proposed the existence of at least two genetic disorders associated with retinitis pigmentosa and hearing loss. Type I consists of retinitis pigmentosa, congenital profound hearing impairment, absent vestibular response to caloric stimulation, and normal intellect. Type II consists of functionally milder retinitis pigmentosa than found in Type I, progressive hearing deficit, normal vestibular function, and normal intellect. They based their classification of Types I and II on their own observations and on Nuutila's study. Progression of hearing loss, described in nine patients with Type II, was solely based on anamnestic data. Additionally, Merin, Abraham, and Auerbach suggested that two other genetic types might exist. Patients with Type III have the same abnormalities as those with Type I, but also have mild vestibular ataxia when walking with closed eyes. Patients with Type IV are also similar to those with Type I, but are mentally retarded. However, they did not assess the contribution of multisystem sensory deprivation to this retardation.

Davenport and Omenn[3] also suggested heterogeneity in Usher's syndrome. Their classification, as summarized by Gorlin and associates,[4] was as follows: Patients with Type I have profound con-

genital deafness with onset of retinitis pigmentosa by 10 years of age. Patients with Type II have moderate-to-severe congenital hearing loss with onset of retinitis pigmentosa late in the second decade of life. Type III is characterized by retinitis pigmentosa, first noted at puberty, and progressive hearing loss. Type IV is similar to Type II and is inherited as an X-chromosome-linked recessive condition.

Davenport and Omenn[3] based their classification of Types I, II, and III on a review of published reports.. They based Type IV, compatible with an X-chromosome-linked recessive inheritance pattern, on two pairs of brothers maternally related as first cousins once removed; however, they examined only the proband (S. L. H. Davenport, M.D., oral communication, March 1980).

Hearing loss in Usher's syndrome has been shown to be sensorineural,[1,18-21] and often more pronounced in the high frequencies.[14,18-22] Most reports suggest that it is stable,[1,2,17,23,24] although some investigators have noted deterioration with age.[4,22,25,26]

McLeod and associates[20] presented evidence against basing classifications of Usher's syndrome on audiologic subgroups. They studied an eight-generation, highly consanguineous Tennessee family with six affected individuals from three sibships. The degree of hearing loss was similar within each sibship, but was different among the three, from moderate (with good speech discrimination) to profound (with no speech discrimination). Their study emphasized the intrafamilial audiologic variability of this genotype. The intrafamilial variability of Usher's syndrome is further underscored by the siblings described in our Cases 1 and 2. Patient 2 noted nyctalopia at an earlier age and, at the time of examination, had a narrower visual field and more severe central visual loss than his sister, Patient 1, did. Epistatic genes, sex-related influences, or environmental factors may account for the phenotypic variation within these families; in each family all affected members presumably had identical alleles for Usher's syndrome.

Our patients with Usher's syndrome can be divided into two groups: one with the ability to discriminate speech (Cases 1 and 2) and one without (Cases 3 to 5). However, there was no correlation between the onset of nyctalopia and the ability to discriminate speech. The medical histories indicated that hearing loss in our patients was probably congenital; serial audiograms, when available, showed stability after the first measurement.

Crystalline retinopathy—Patient 6, with moderate sensorineural hearing loss and late onset of nyctalopia, had unusual crystalline retinal deposits suggesting a metabolic defect different from that of Usher's syndrome.

Retinitis pigmentosa, hearing loss, and enamel dysplasia—Case 7 represents, to the best of our knowledge, a previously undescribed combination of retinitis pigmentosa, sensorineural hearing loss, and generalized enamel defect. This patient, who denied having nyctalopia, demonstrates the hazard of basing classifications on the history of this symptom as Nuutila[1] did. We could find no environmental cause (such as severe illness during tooth development) for the enamel dysplasia. Additional reports of patients with this constellation of abnormalities may delineate this condition as a distinct syndrome.

Clumped pigmentary retinopathy and hearing loss—Cases 8 and 9 are, we believe, unique. Both of these siblings had moderate-to-severe sensorineural hearing loss and similar clumped pigmentary degeneration of the retina. However, only Patient 8 had enamel dysplasia; this defect was probably caused by malab-

sorption syndrome early in life. Results of ocular and auditory examinations of both parents were normal. Both parents were of Sicilian extraction but denied consanguinity. Autosomal recessive inheritance of this disorder is likely, although X-chromosome linkage cannot be ruled out.

Amalric-Diallinas syndrome—Diallinas[5] and Amalric[6] described patients with hearing loss and a granular macular pigment epitheliopathy. Visual acuity was normal in all cases. An electroretinogram done in one of Diallinas' two patients was normal. Of Amalric's 15 cases, four had prenatal histories compatible with rubella infection. However, in another case the mother contracted rubella before becoming pregnant with the affected child. Amalric suggested that at least some of the cases might have been hereditary. Woitlez,[27] Cernea, Teodoresure, and Banu,[10] Remky, Klier, and Kobor,[8] and Charamis and associates[9] reported a total of 47 similar cases of maculopathy with normal visual acuity and hearing loss; they did not provide genetic data. Grimaud and associates[28] described 22 patients with profound hearing impairment associated with chorioretinopathy. Five cases were said to be hereditary, but they provided no genetic information. Each of the five patients had normal visual acuity and a normal electroretinogram. François, Haustrate-Gosset, and Donck[7] reported 76 cases of "Amalric's dystrophy" among 1,065 hearing-impaired patients. Eleven cases were classified as hereditary, although they did not document the modes of inheritance.

Retinal pigment epitheliopathy associated with hearing impairment is known to occur as the result of prenatal rubella infection.[29-35] At least 20% of patients with congenital rubella have widespread retinal pigmentary irregularities.[32] Moreover, approximately 40% of pregnant women with rising rubella antibody titers are clinically asymptomatic, although they may give birth to infants with rubella syndrome.[36] Isolated macular lesions have also been noted in infants whose mothers reported a rash during pregnancy; however, these cases were diagnosed as congenital rubella before the era of laboratory documentation.[37,38] Visual acuity is characteristically normal in patients with retinopathy as the only manifestation of rubella embryopathy.[31,32] Hearing loss secondary to prenatal rubella infection is typically sensorineural and the age at onset and course vary among individuals.[39] Our patients 10, 11, and 12 have profound hearing loss and retinal pigment epitheliopathy that is predominantly macular and similar to that found in rare cases of isolated macular lesions caused by presumed rubella embryopathy.[37] Thus, although our patients have the retinal and audiologic features of Amalric-Diallinas syndrome, we cannot rule out the possibility that our patients may have had rubella embryopathy. Indeed, Amalric-Diallinas syndrome may be the result of intrauterine rubella infection and may not be a genetic disorder.

Maculopathy, hearing loss, and enamel dysplasia—We found retinal maculopathy without the typical fundus and electroretinographic alterations of retinitis pigmentosa in Case 13. We did not find the salt-and-pepper macular epitheliopathy characteristic of the Amalric-Diallinas syndrome. This patient had a stable maculopathy without flecks, moderate, stable sensorineural hearing loss, and an enamel defect. Her visual acuity had been stable for five years. Electrophysiologic testing showed minimal alterations of the photopic and scotopic electroretinographic responses. The pattern of the enamel defect was compatible with that found in hereditary disorders.[40]

Summary

Retinal abnormalities associated with hearing loss may be inherited alone or

with additional manifestations. Environmental insults, such as rubella embryopathy, may also cause these abnormalities. We studied 13 patients with retinal abnormalities and hearing loss. Five had Usher's syndrome (retinitis pigmentosa and hearing impairment), one had crystalline retinopathy, two had associated enamel dysplasias, two had clumped pigmentary retinopathy, and three had Amalric-Diallinas syndrome. Our findings suggest considerable heterogeneity of syndromes involving retinal abnormalities and hearing loss.

ACKNOWLEDGMENT

Martin Lederman, M.D., performed electroretinographic studies in Case 8.

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Ophthalmic miniature

But she sure did miss the television. It was just shapes and light that didn't mean anything. She wished the cataracts would hurry and get ripe enough.

John D. MacDonald, *The Long Lavender Look*
Greenwich, Connecticut, Fawcett, 1970

RETINAL PIGMENT EPITHELIAL DEGENERATION ASSOCIATED WITH LEUKOCYTIC ARYLSULFATASE A DEFICIENCY

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Metachromatic leukodystrophy is a genetically determined disorder associated with accumulation of sulfatide caused by the deficient activity of arylsulfatase A. There are several forms of metachromatic leukodystrophy: late infantile, juvenile, adult, and a variant form with multiple sulfatase deficiency.[1] This disease mainly affects the myelin sheaths of the central and peripheral nervous systems. The ocular manifestations are nystagmus and visual loss in association with optic atrophy and gray maculas.[2]

We have studied two sisters with levels of leukocyte sulfatase A indistinguishable from those occurring in metachromatic leukodystrophy. However, they differed from patients with typical metachromatic leukodystrophy in that sulfatiduria was absent and there was readily detectable cerebroside sulfatase activity. In contrast, patients with metachromatic leukodystrophy have absent sulfatidase activity and large, easily measurable quantities of urinary sulfatide. Furthermore, the proband had nystagmus and progressive visual loss with retinal pigment epithelial degeneration. We believe this family had a variant of arylsulfatase A deficiency, which differed from the typical pattern seen in metachromatic leukodystrophy in that the progressive retinal pigment epithelial degeneration was the only clinical manifestation.

CASE REPORT

This 9-year-old girl had been found to have nystagmus at the age of 8 months, at which time an ocular examination disclosed mottling of the macular retinal pigment epithelium. She was referred here at the age of 6 years for evaluation of decreased visual acuity.

Ocular examination in March 1976 disclosed a searching nystagmus and a visual acuity of 6/150 (8/200) in both eyes. There was no discernible refractive error. Results of the anterior segment examination were normal. The irides were blue with normal pigment epithelium. Examination with the pupils dilated disclosed a clear lens and vitreous in both eyes. The optic nerve heads and retinal vasculature were normal. The foveas were atrophic in both eyes with diffuse bilateral mottling of the pigment epithelium of the posterior poles (Fig. 1). Color vision testing showed a mild red-green defect in both eyes. The electroretinogram demonstrated slightly decreased amplitudes for both A and B waves with normal implicit times using a bright W-16 stimulus. Results of pediatric and neurologic examinations were normal except for the visual defect. An electroencephalogram was normal. We examined members of her family, including her parents, an 11-year-old sister of the proband, grandparents, two aunts, and two uncles, and found them to be normal with the exception of a maternal uncle who had a history of depigmentation of the skin and hair, beginning at the age of 20 years.

Because it was difficult to arrive at a diagnosis for the proband, we had blood samples drawn for blood enzyme studies. The results of the enzyme studies were normal except for a deficiency in arylsulfatase A activity,[3,4] ruling out Krabbe's disease, the G_{M1} and G_{M2} gangliosidoses, Gaucher's disease, Fabry's disease, mannosidosis, fucosidosis, mucopolysaccharidosis (Sanfilippo's syndrome), and β-glucuronidase deficiency disease. Results of similar studies on other family members were normal except for reduced

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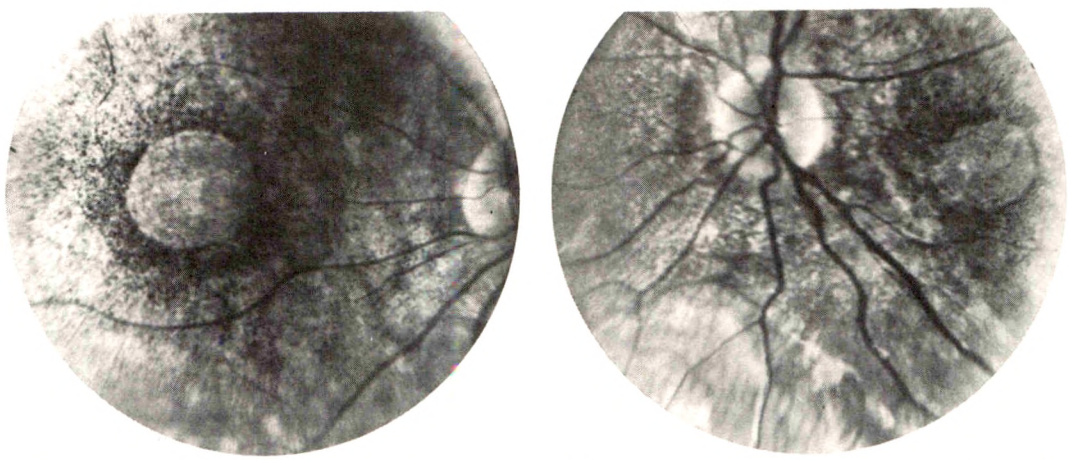


Fig. 1 (Weiter and associates). Foveal atrophy and diffuse mottling of the pigment epithelium at the posterior pole when the proband was 6 years old. Left, Right eye. Right, Left eye.

levels of arylsulfatase A activity in her parents and sister (Table). Skin fibroblasts from the patient, her sister, and their parents, challenged by loading the culture media with sodium sulfate S 35, hydrolyzed this material at the same rate as a normal cell line, indicating normal sulfatide turnover.[5] The leukocyte arylsulfatase A activity in both the proband and her older sister was in the range of metachromatic leukodystrophy, but both had appreciable leukocyte[6] and fibroblast[7] sulfatidase activity and neither excreted an excess of urinary sulfatide. Therefore, neither met all of the biochemical criteria for the diagnosis of metachromatic leukodystrophy.

Subsequent ocular examinations during the next three years showed slowly progressive retinal degeneration (Fig. 2). The macular and peripapillary regions of both eyes demonstrated diffuse salt-and-pepper changes in the retinal pigment epithelium. The foveas of both eyes had a sharply demarcated area of atrophy that included loss of the underlying choriocapillaris. Both eyes had several far peripheral areas of retinal atrophy.

At the present time, there are drusen in both optic disks and the ERG has not appreciably changed from earlier tests. Results of neurologic examinations are still normal and the patient is doing well in school.

Discussion

This family had a deficiency of the enzyme arylsulfatase A that appeared to be transmitted as an autosomal recessive trait. Furthermore, they had measurable quantities of sulfatidase activity without

TABLE

Leukocyte enzyme activity in the proband and her immediate family

Individual	Arylsulfatase A*	Arylsulfatase A/ Arylsulfatase B*	Sulfatidase*
Normal controls	63.2±9.0	0.44±0.11	6.69±1.81
Obligate heterozygotes for metachromatic leukodystrophy	45.9±19.1	0.28±0.13	2.65±1.58
Metachromatic leukodystrophy	27.1± 9.9	0.15±0.04	0
Proband	19.5	0.23	2.55
Sister	21.5	0.24	2.35
Father	40.6	0.47	3.82
Mother	49	0.28	4.14

*Expressed as nmoles/mg of protein/hr.

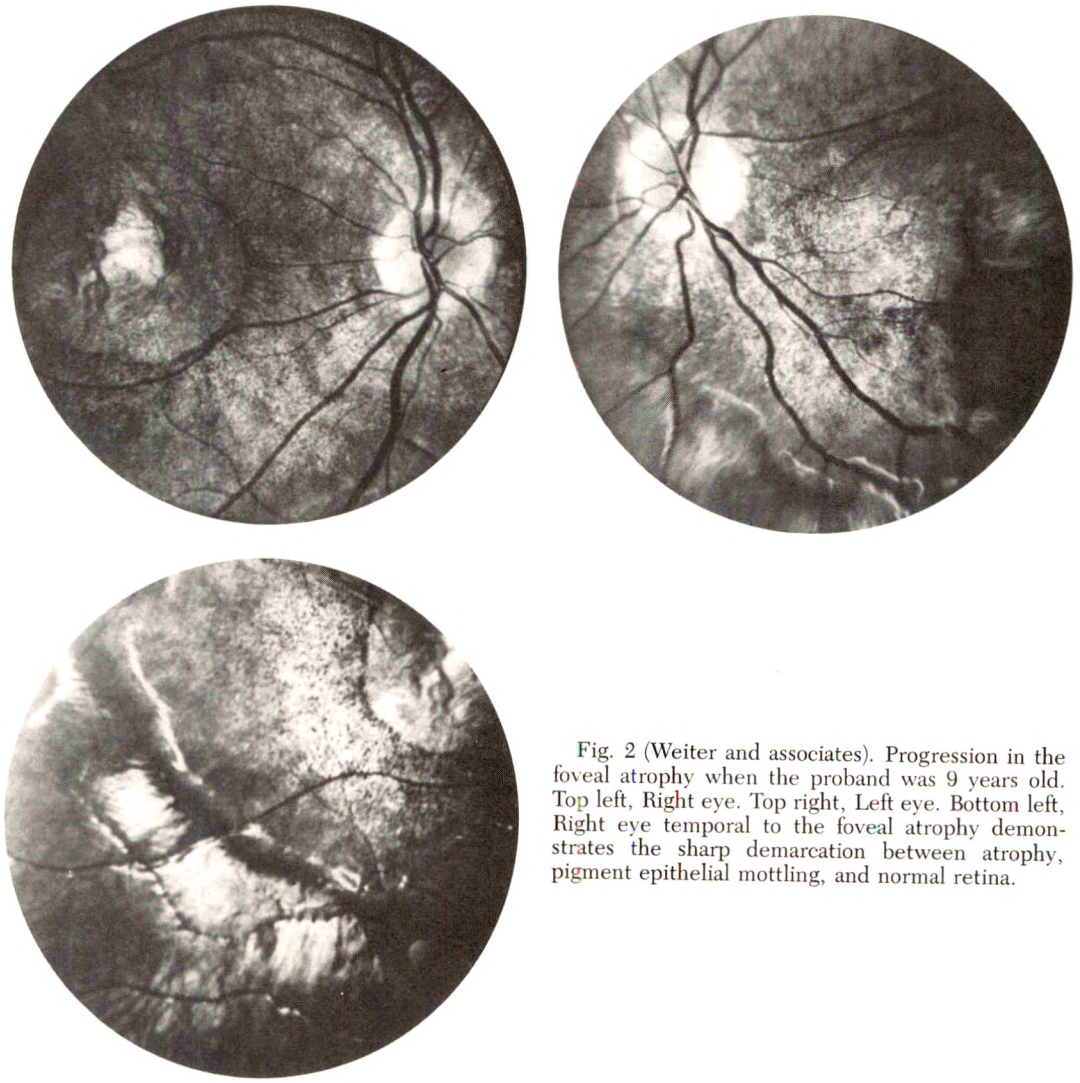


Fig. 2 (Weiter and associates). Progression in the foveal atrophy when the proband was 9 years old. Top left, Right eye. Top right, Left eye. Bottom left, Right eye temporal to the foveal atrophy demonstrates the sharp demarcation between atrophy, pigment epithelial mottling, and normal retina.

significant sulfatiduria, and therefore differed from patients with metachromatic leukodystrophy who have absolutely no sulfatidase activity and large, easily measurable quantities of urinary sulfatide. For the same reason, they differed from patients in the preclinical stage of adult metachromatic leukodystrophy, because the preclinical stage includes arylsulfatase A deficiency in the leukocytes and urine and massive urinary excretion of sulfatides.[8,9] One of us (E.H.K.) recently described patients with neurologic diseases other than metachromatic leukodystrophy who had low arylsulfatase A activity in leukocytes but no sulfatiduria and measurable quantities of sulfatidase activity against the radioactively labelled natural product.[10] Similar findings have been observed in otherwise normal relatives of patients with metachromatic leukodystrophy.[10-13] The distinctive features of the proband and her relatives were that no member of the family had

metachromatic leukodystrophy and there was no intellectual impairment in either the proband or her sister. To the best of our knowledge, one or both of these features has characterized every case of low levels of arylsulfatase A without metachromatic leukodystrophy. We believe that the proband and her sister had the same condition, probably inherited in an autosomal recessive fashion. The presence of the same biochemical abnormality in the proband and her sister was, in our opinion, strong evidence that both children were affected despite the lack of specific findings in the sister. The absence of specific findings in the sister might also be explained by decreased penetrance and expressivity of the gene responsible for the disease.

The ocular findings in the proband were not typical of metachromatic leukodystrophy. In metachromatic leukodystrophy, findings are usually related to the optic nerve and ganglion cell layer and consist of optic atrophy and occasionally a gray macula or cherry-red spot.[1,2] Recent electron microscopic studies of the retina in metachromatic leukodystrophy show that the membranous lysosomal residual bodies are confined to the ganglion cells.[14,15] We found increased lipofuscin levels in the retinal pigment epithelium as well as in the ganglion cells in the proband. Light microscopy demonstrated acid phosphatase in pigment epithelial cells as well as ganglion cells. (Both lipofuscin[16] and acid phosphatase are normal constituents of the adult retinal pigment epithelium, but we believed that the levels of both were increased.) The increased lipofuscin level may be linked to the metachromatic leukodystrophy by a mechanism similar to that responsible for the increase in lipofuscin that has been reported to accompany lysosomal glycogen storage in infantile Type II glycogenosis.[17]

The ocular changes in the present case were confined to the macular and peripapillary regions of both eyes, with marked atrophy in the foveas. The primary defect appeared in the retinal pigment epithelial photoreceptor complex with the addition of choriocapillaris atrophy in the foveas. The differential diagnosis of connatal retinal pigment epithelial degeneration includes Leber's congenital amaurosis, in utero maternal viral infection, and neuronal ceroid-lipofuscinosis.[18,19]

Leber's congenital amaurosis is an autosomal recessive disorder characterized by poor visual acuity, nystagmus present at birth, an absent ERG response, and progressive pigmentary changes at the posterior pole.[20] The reduced but present ERG response with normal latencies in our patient rules out this entity.

A congenital viral infection cannot be ruled out, although the absence of a maternal history of infections during pregnancy, the clinical pattern, and the progressive nature of the retinopathy argue against this diagnosis. Neuronal ceroid-lipofuscinosis is unlikely because of the absence of leukocytic abnormalities and the normal neurologic findings.

The present case of pigment epithelial degeneration does not fit into any recognized category. It is tempting to postulate a causal relationship between the arylsulfatase A deficiency and the retinal degeneration despite the findings in the patient's sister. Hara, Hayasaka, and Mizuno[21] studied lysosomal arylsulfatases A and B in normal bovine eyes and found high levels in the sensory retina, optic nerve, and retinal pigment epithelium. Because the retinal pigment epithelium has high levels of arylsulfatase A under normal conditions, it is reasonable to expect changes in this tissue in a deficiency state. A possible explanation for se-

lective tissue degeneration, as well as for the various clinical manifestations of metachromatic leukodystrophy, is that there are many molecular forms of arylsulfatase A in the different clinical variants.[22]

Summary

A family exhibiting a leukocytic arylsulfatase A deficiency, probably inherited in an autosomal recessive manner, differed from patients with typical metachromatic leukodystrophy in that sulfatiduria was absent and there was readily detectable cerebroside sulfatase activity. To our knowledge, this family was unique in that there were no known members with metachromatic leukodystrophy and the only neurologic abnormality was progressive retinal pigment degeneration in the proband.

Acknowledgments

Aubrey Milunsky, M.D., established the skin fibroblast lines. Hayato Kihara, M.D., performed the sodium sulfate S 35 test.

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BIRDSHOT RETINOCHOROIDOPATHY

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Ryan and Maumenee[1] recently reported a clinical syndrome consisting of diffuse posterior choroidopathy, retinal vasculitis, vitritis, and cystoid macular edema, which they termed birdshot retinochoroidopathy. The chorioretinal lesions in this disease are scattered and hypopigmented, bearing a resemblance to birdshot.

We studied the varied clinical manifestations of this disease in four patients. They not only had the typical spotted fundi but also distinctive electrophysiologic abnormalities. These characteristics distinguish patients with this disease from those with inflammatory and dystrophic conditions of the choroid, retinal pigment epithelium, or both.

CASE REPORTS

Case 1—In March 1972, we examined a 45-year-old man who had a nine-month history of decreased visual acuity for distance and decreased depth perception, night vision, and color vision (Table 1). He had had no ocular disease except mild myopia since childhood. His family history was noncontributory for ocular disease. Significantly, he had a demanding job which generated considerable anxiety.

On examination, best corrected visual acuity was R.E.: 6/6 (20/20) and L.E.: 6/7.5 (20/25). Results of an examination of the anterior segment of both eyes were normal, with no flare or cells within the anterior chambers. Applanation tonometry values were R.E.: 14 mm Hg and L.E.: 13 mm Hg. Biomicroscopy disclosed 4+ diffuse cells throughout the vitreous of both eyes which hindered visualization of the fundus. There was a posterior vitreous detachment in the left eye. There were diffuse hypopigmented areas of choroid and retinal pigment epithelium in the posterior pole, extending from the optic disk to the equator (Fig. 1). The right macula had fine retinal striae, whereas the left had a fine epiretinal membrane. Both eyes showed alternating dilatation and constriction of the retinal veins, with sheathing most prominent in the temporal retina. There were no punched-out areas of choroidal atrophy in the periphery anterior to the equator of either eye.

Fluorescein angiography showed many window defects which corresponded to areas involved ophthalmoscopically. In the early phase of the angiogram, there was increased transmission of dye within the choroidal and pigment epithelial lesions. The optic disk stained heavily early and leaked into the surrounding peripapillary retinal tissue (Fig. 2). Cystoid macular edema was present in both eyes, but was more marked on the left. Goldmann visual fields disclosed no detectable scotoma, although there was enlargement of the blind spot bilaterally. We believed that the patient had birdshot retinochoroidopathy with inflammation involving the retinal vasculature and the vitreous.

In April 1973, visual acuity decreased to R.E.: 6/9 (20/30) and L.E.: 6/12 (20/40) because of cystoid macular edema. He received 40 mg of triamcinolone acetonide retroseptally in both eyes. Within three weeks his visual acuity improved to R.E.: 6/6−1 (20/20−1) and L.E.: 6/7.5+1 (20/25+1). During the next three years, he suffered two recurrent inflammatory episodes, each treated with 40 mg of retroseptally administered triamcinolone acetonide. The right eye responded well on each occasion but the left eye did not improve.

In June 1976, visual acuity was R.E.: 6/12−2 (20/40−2) and L.E.: 6/18 (20/60). The patient believed that stress was contributing significantly to his ocular disability, so he retired from his job. Visual acuity in his right eye improved to 6/7.5+3 (20/25+3) almost immediately, and there was a noticeable resolution of his vitritis and cystoid macular edema.

In March 1978, visual acuity was R.E.: 6/7.5+2 (20/25+2) and L.E.: 6/15 (20/50), despite bilateral small posterior subcapsular cataracts and mild cellophane maculopathy. Color testing with the Nagel anomaloscope disclosed no green-red imbalance. Dark-adaptation testing disclosed bilaterally diminished cone and rod amplitude; this was most marked in his left eye. Results of an electro-oculogram (EOG) were below normal in his left eye, with a light peak-dark trough ratio of 2.20 in his right eye and 1.66 in his left eye (normal for our laboratory, $> 2.00 \pm 0.25$). The electroretinogram (ERG) showed reduced rod a- and b-wave amplitudes with borderline cone response (Table 2). At the present time, he is taking no medications and his visual acuity is stable.

Case 2—In June 1974, we examined a 53-year-old woman with a six-month history of decreased visual acuity for distance in her left eye, which had worsened the two weeks before her visit. She had

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TABLE 1

SUMMARY OF CLINICAL DATA

Findings	Case 1	Case 2	Case 3	Case 4
Age (yrs)	45	53	53	70
Sex	M	F	M	F
Length of follow-up	7 yrs	5 yrs	2 yrs	4 mos
Initial visual acuity				
R.E.	6/6 (20/20)	6/6−2 (20/20−2)	6/9 (20/30)	6/6−3 (20/20−3)
L.E.	6/7.5 (20/25)	6/12−2 (20/40−2)	6/18−2 (20/60−2)	6/60 (20/200)
Final visual acuity				
R.E.	6/7.5+2 (20/25+2)	6/6 (20/20)	6/6−1 (20/20−1)	6/6−1 (20/20−1)
L.E.	6/15 (20/50)	6/9 (20/30)	6/30 (20/100)	6/30 (20/100)
Responsive to corticosteroid	Yes	Yes	No	No
Fluorescein angiography	Both eyes: cystoid macular edema; choroidal and retinal vascular leakage	Both eyes: cystoid macular edema; choroidal and retinal vascular leakage	L.E.: cystoid macular edema. Both eyes: possible vascular leakage, bleached by extensive transmission defects	L.E.: cystoid macular edema, peripapillary leakage. Both eyes: extensive hyperfluorescence secondary to transmission defects
Electrophysiologic tests				
Dark adaptation	Both eyes: diminished cone and rod amplitude	Both eyes: normal	Both eyes: cones normal, no rod adaptation	Both eyes: cones normal, rod adaptation low
ERG	Both eyes: diminished rod a- and b-wave; diminished rod and cone b-wave:implicit time ratio	Both eyes: diminished rod b-wave; diminished rod and cone b-wave: implicit time ratio	Both eyes: diminished rod a- and b-wave; diminished rod and cone b-wave: implicit time ratio	L.E.: Diminished rod b-wave; diminished rod b-wave: implicit time ratio
EOG				
R.E.	2.20	2.31	1.07	2.51
L.E.	1.66	2.92	1.10	1.45

had no previous ocular disease. Her medical history was significant in that she had had a total abdominal hysterectomy for carcinoma of the cervix in 1968. She complained of stiffness and swelling in the proximal joints of her hands which, after a thorough examination, was diagnosed as osteoarthritis.

Best corrected visual acuity was R.E.: 6/6−2 (20/20−2) and L.E.: 6/12−2 (20/40−2). The anterior segments were normal with no flare or cell within the anterior chambers. Applanation tonometry values were 16 mm Hg in both eyes. Biomicroscopy disclosed 3+ vitreous cells bilaterally with many veil-like opacities. There was a posterior vitreous detachment bilaterally, with a detached epipapillary vitreous ring present. Both maculas had cystoid edema with wrinkling of the internal limiting membrane. Indirect ophthalmoscopy of both eyes disclosed normal optic disks with peripapillary choroidal atrophy. The retinal veins showed alternating dilatation and constriction with focal areas of sheathing. Scattered throughout the posterior pole were diffuse speckled subretinal lesions extending to the equator. We found numerous round, punched-out lesions at the equators of both eyes inferiorly.

Fluorescein angiography disclosed leakage of dye from the peripapillary retinal capillaries and the retinal veins (Fig. 3). The late phase of the angiogram demonstrated bilateral macular edema. We believed the patient had birdshot retinochoroidopathy and, because of her macular edema, she began taking 80 mg/day of orally administered prednisone. Be-

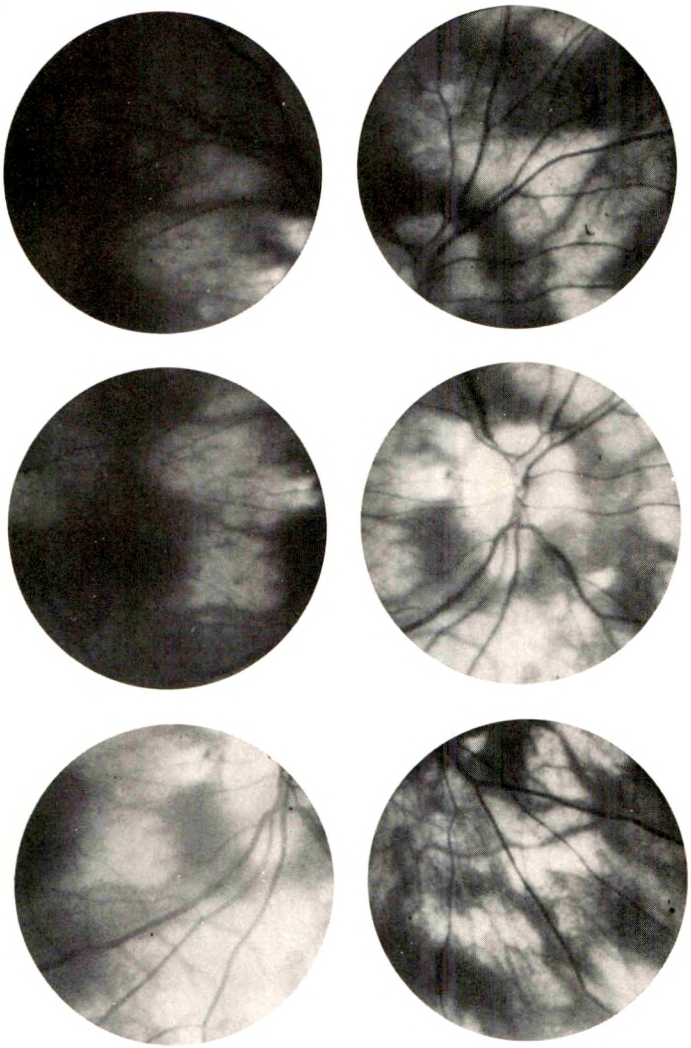


Fig. 1 (Kaplan and Aaberg). Case 1. Right eye in 1979. Multiple discrete hypopigmented lesions in the choroid and retinal pigment epithelium extend from the optic disk to the equator. There are large choroidal vessels in the atrophic areas, which have no surrounding hyperpigmentation.

cause her chest X-ray showed a calcified Gohn complex, she also took 300 mg/day of isoniazid. Her visual acuity remained 6/6−2 (20/20−2) in the right eye and improved to 6/9 (20/30) in the left eye. Her prednisone dosage was gradually decreased during the next four months. There was a noticeable decrease in vitreous inflammation.

In January 1979, ERGs showed an abnormally low rod b-wave with borderline low cone a- and b-waves and rod a-waves in both eyes (Table 2). Since her implicit time was prolonged, the b-wave:implicit time ratio was markedly low. Her EOG values were normal, as were results of her dark-adaptation test. The Nagel anomaloscope showed no red-green imbalance. Goldmann visual fields showed no detectable scotomas.

Case 3—A 53-year-old man, examined in August 1978, had had blurred vision and poor night vision in his left eye since April 1977. Family and social histories were noncontributory, as were systemic examinations.

On examination, best corrected visual acuity was R.E.: 6/9 (20/30) and L.E.: 6/18−2 (20/60−2). Near visual acuity was R.E.: J−2 and L.E.: J−5. The anterior segments of both eyes were normal with no flare or cells. Applanation tonometry values were 17 mm Hg in both eyes. Biomicroscopy disclosed 4+ cells diffusely scattered throughout the vitreous of both eyes. We observed cystoid edema in the left macula but not in the right. Indirect ophthalmoscopy of both eyes disclosed normal optic disks with a slight elevation of the nasal border. There were

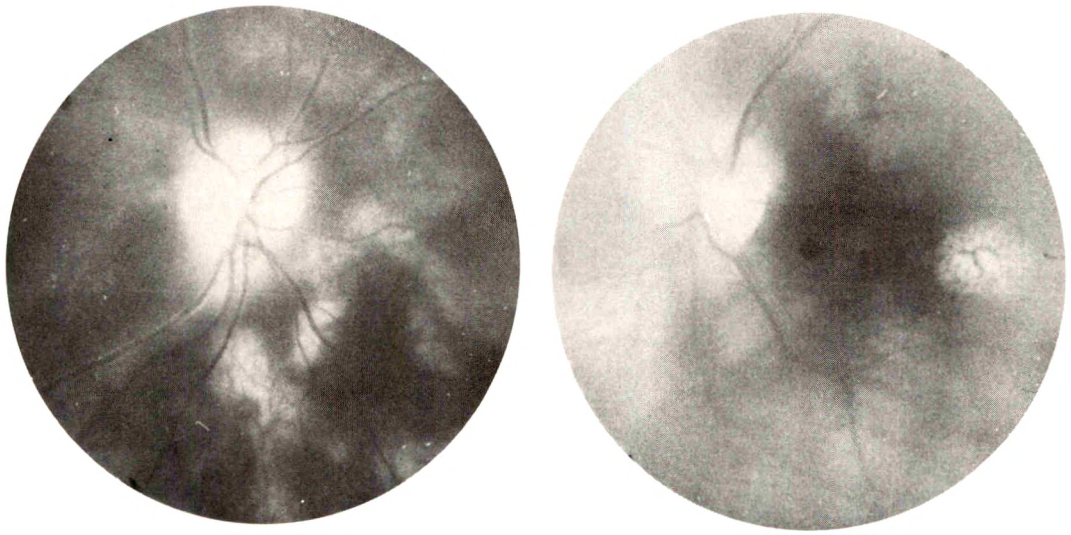


Fig. 2 (Kaplan and Aaberg). Case 1. Late-phase fluorescein angiograms of the right optic disk (left) and left macula (right) in 1972. Fluorescein leaked from the right optic disk into the surrounding peripapillary tissue. Cystoid macular edema is present in the left macula.

discrete subretinal depigmented lesions extending from the optic disk margins to the equators (Fig. 4). Peripheral to the equator, the fundi were normal. The retinal vasculatures were normal.

Fluorescein angiography showed marked hyperfluorescence through the areas of affected pigment epithelium and choroid. The transmitted fluorescein was so extensive that it obscured the retinal vasculature. However, late photographs of the left eye showed cystoid macular edema.

His EOG values were markedly reduced (R.E.: 1.07 and L.E.: 1.10). His ERG showed reduced rod a- and b-wave amplitudes that were low. The b-wave:implicit time ratios for both rods and cones were very low (Table 2).

The Farnsworth-Munsell 100-Hue test showed

TABLE 2

RESULTS OF ELECTRORETINOGRAPHIC STUDIES

Study	Case 1		Case 2		Case 3		Case 4		Normal Range
	R.E.	L.E.	R.E.	L.E.	R.E.	L.E.	R.E.	L.E.	
Light-adapted									
a-wave (mV)									
× 4	50	50	50	60	60	40	70	70	40-110
× 16	70	80	80	50	60	55	100	80	—
b-wave (mV)									
× 4	80	70	80	65	80	60	160	80	60-190
× 16	80	70	60	60	80	60	120	60	—
b-wave:implicit time ratio*	4.0	3.3	2.5	2.5	3.81	3.0	7.06	3.33	—
Dark-adapted									
a-wave (mV)									
× 4	120	120	130	140	70	80	200	140	130-350
×16	200	200	95	120	90	100	340	180	—
b-wave (mV)									
× 4	160	150	160	150	80	100	320	160	260-530
× 16	220	230	160	160	100	90	340	185	—
b-wave:implicit time ratio*	3.8	3.8	4.0	3.75	1.86	2.33	6.67	3.33	

*Calculated using the b-wave amplitude at × 4.

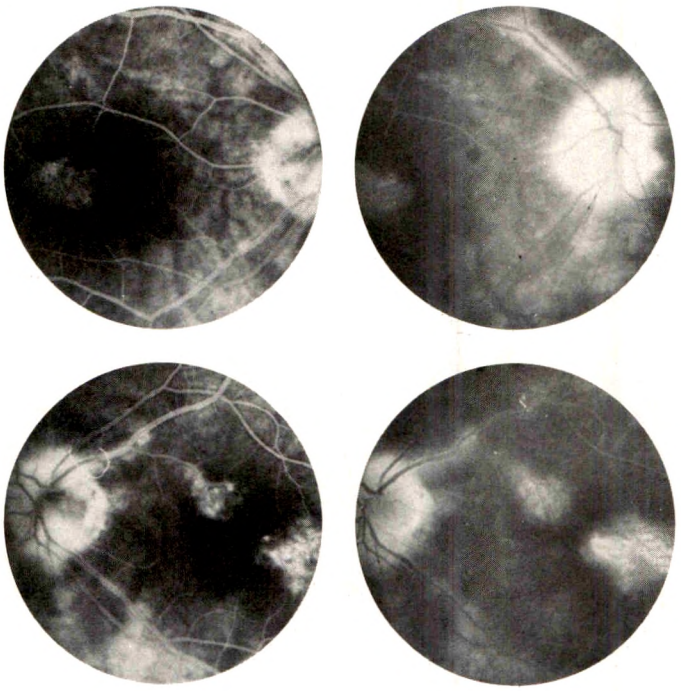


Fig. 3 (Kaplan and Aaberg). Case 2. Fluorescein angiograms of right eye (top left, midarteriovenous phase; top right, late phase) and left eye (bottom left, midarteriovenous phase; bottom right, late phase). There are many window defects which transmit dye, as well as leakage of fluorescein from the optic disk and retinal veins.

extremely poor color discrimination in his left eye (an achromatic pattern), with tritanopia in his right eye. Dark adaptation showed a normal cone amplitude, but no rod response in either eye. Goldmann visual fields disclosed an isolated scotoma in the temporal field of the right eye, with baring of the blind spot in the left eye.

We believed that the patient had birdshot retinochoroidopathy and prescribed 80 mg/day of prednisone because of cystoid macular edema in his left eye. The prednisone dosage was gradually decreased during the next six months. There was a slight improvement in the visual acuity of his right eye to 6/6−1 (20/20−1) and a worsening of the visual acuity of his left eye to 6/30 (20/100).

Case 4—A 70-year-old woman, examined in October 1979, had a two-year history of decreased visual acuity in her left eye. Two years previously, a retinal tear with fluid in that eye had been treated with cryotherapy. Her visual acuity had continued to decrease, and she complained of metamorphopsia and micropsia. Her medical and family history were not contributory.

On examination, best corrected visual acuity was R.E.: 6/6−3 (20/20−3) and L.E.: 6/60 (20/200). Her anterior segments were normal without evidence of inflammation; applanation tonometry values were 12 mm Hg in both eyes. Slit-lamp biomicroscopy disclosed bilateral vitreous cells, 1+ in the right eye and 3+ in the left. The right macula had a fine cellophane appearance inferior to the fovea. The left macula had prominent cystoid macular edema with an occasional punctate hemorrhage. Indirect ophthalmoscopy of both eyes showed atrophy of the retinal pigment epithelium, with focal areas of hypopigmentation of the choroid and retinal pigment epithelium extending from the optic disk toward the equator (Fig. 5). The retinal vessels were normal, with no venous caliber abnormalities or sheathing. Fluorescein angiography showed numerous hyperfluorescent transmission defects in both eyes. There was peripapillary vascular leakage of fluorescein about the left optic disk in the midarteriovenous stage, with the appearance of cystoid macular edema in the late stage (Fig. 6).

Her EOG was normal in the right eye but low in the left eye (R.E.: 2.51 and L.E.: 1.45). Her ERG showed a low rod b-wave amplitude in the left eye only, with a decreased b-wave:implicit time ratio. Dark adaptation testing showed a normal cone response in both eyes, with a decreased rod amplitude most marked in the left eye. No color testing was performed.

The clinical appearance and electrophysiologic tests indicated that she had birdshot retinochoroidopathy. She began taking 80 mg/day of prednisone even though the cytoid macular edema in her left eye had probably been present for almost two years. During the three-month follow-up period, there was no change in her visual acuity.

Discussion

Birdshot retinochoroidopathy is a distinct clinical entity characterized by diffuse posterior choroidopathy, vitritis, ret-

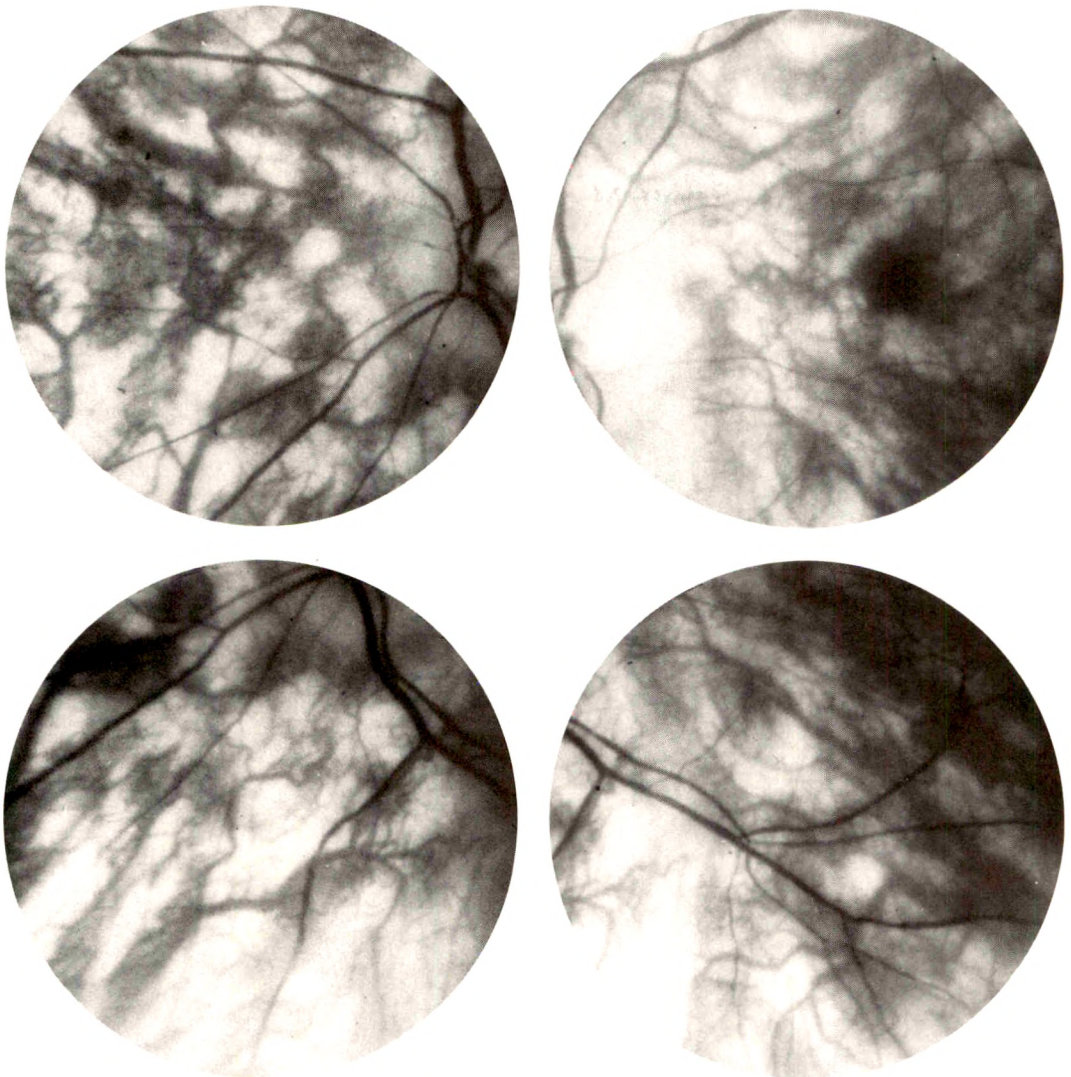


Fig. 4 (Kaplan and Aaberg). Case 3. Left eye in 1978. The subretinal lesions form atrophic patches. The optic disk and retinal vasculature are normal. The right eye appeared the same.

inal vascular leakage, and cystoid macular edema. Our patients, two men and two women, ranged in age from 45 to 70 years. The primary lesion was confined to the choroid and overlying retinal pigment epithelium and did not change in appearance throughout the follow-up period, which was seven years in the patient followed up the longest. There was at least a six-month delay between the onset of symptoms and the first ocular examination in each of our patients, so we may not have seen the lesions in their earliest stages.

This syndrome is a chronic disease with exacerbations and remissions which can progress to the loss of central vision. As a result, it is difficult to document the response to medical therapy. Nevertheless, two of our patients (Cases 1 and 2) had at least a transient improvement in visual acuity after corticosteroid treat-

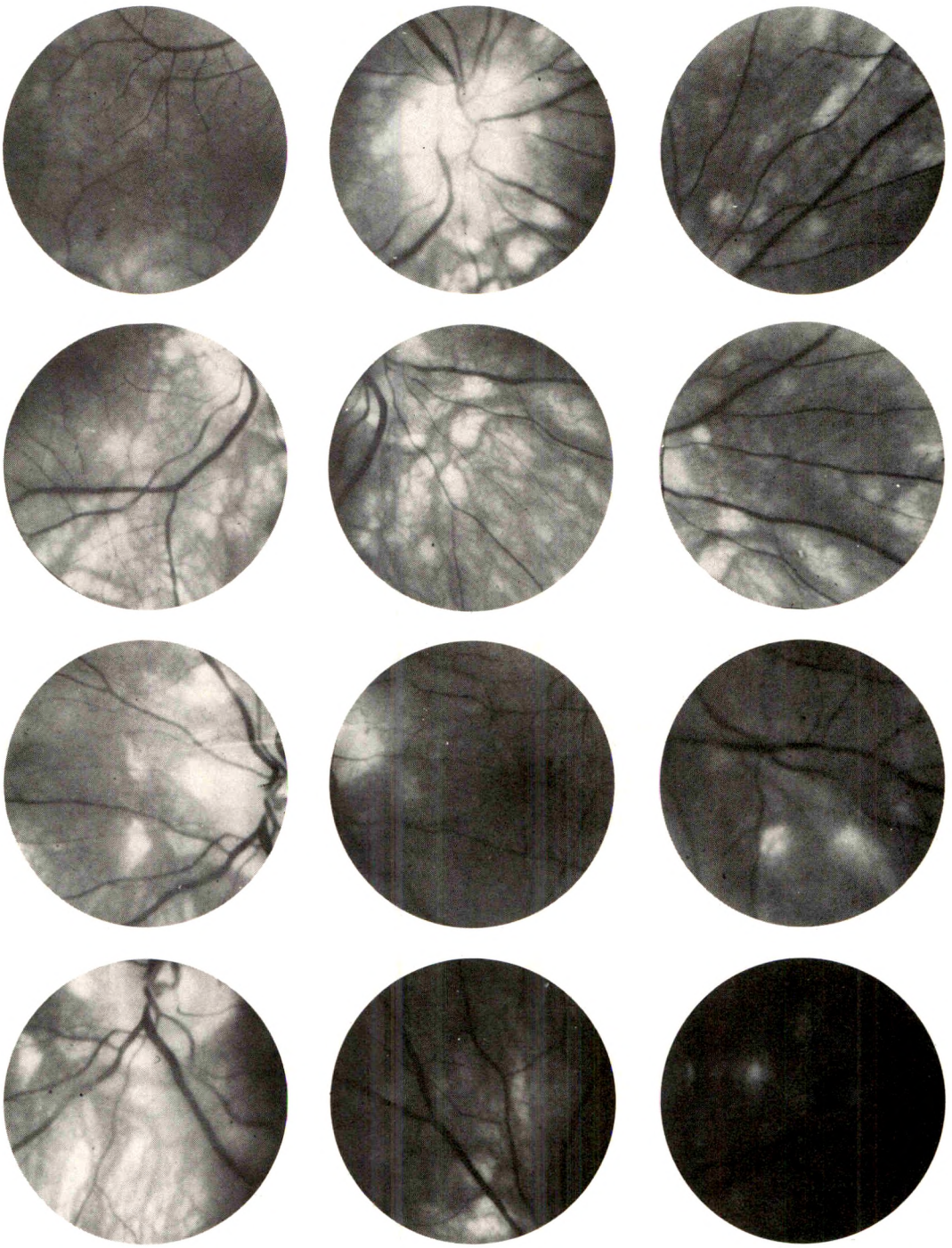


Fig. 5 (Kaplan and Aaberg). Case 4. Right eye (top two rows) and left eye (bottom two rows) in 1979. Although the right eye had more extensive atrophy of the retinal pigment epithelium and choroid, cystoid macular edema reduced visual acuity in the left eye.

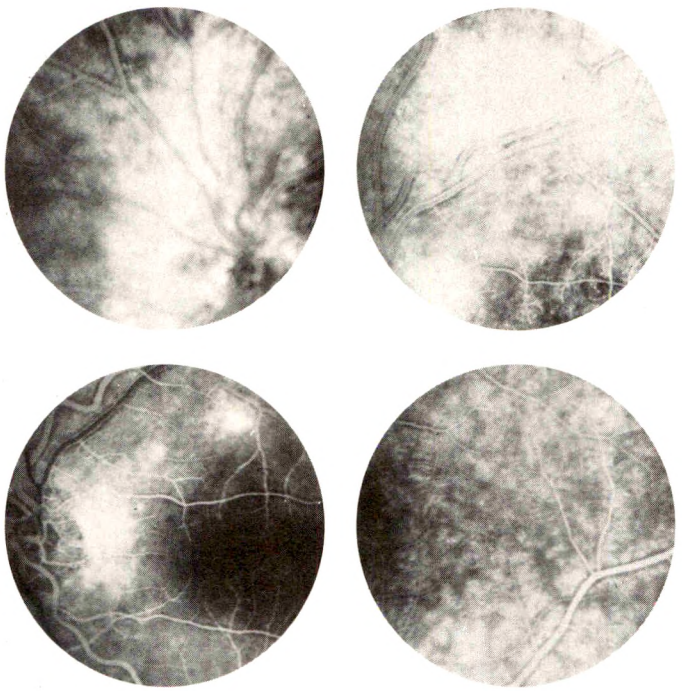


Fig. 6 (Kaplan and Aaberg). Case 4. Fluorescein angiogram of the left eye in 1979. Hyperfluorescent transmission defects can be seen in the early arteriovenous phase (bottom left). The later stages of the angiogram show leakage of fluorescein about the optic disk and the retinal vasculature.

ment, including resolution of their cystoid macular edema. Two other patients (Cases 3 and 4) did not respond to corticosteroid therapy. The presence of vitritis and retinal vascular leakage, as well as the apparent occasional response to corticosteroids in some patients, seems to imply that inflammation is a significant pathophysiologic factor in this disease.

The diffuse nature of the retinal lesions in birdshot retinochoroidopathy is reflected in our electrophysiologic studies. Each patient had an ERG with a diminished rod b-wave amplitude and cone and rod b-wave:implicit time ratios. The cone a- and b-wave amplitudes were less affected, being low borderline. The EOG was below normal in four of the eight eyes in our series, whereas dark adaptation was abnormal in six of the eight eyes. Because this disease seems to be a primary abnormality of the choroid, it must be assumed that these electrophysiologic abnormalities represent dysfunction of the overlying retinal pigment epithelium and sensory retina.

Ryan and Maumenee[1] reported 13 cases of birdshot retinochoroidopathy seen at the Wilmer Eye Institute. The clinical presentation of their patients seems identical to that of ours. Schatz and associates[2] reported a single case of birdshot retinochoroidopathy. However, they did not include details of the clinical course and diagnostic studies on their patient. Gass[3] studied a series of patients with vitiliginous chorioretinitis which seems to be identical to this syndrome.

Other diseases producing chorioretinal lesions include diffuse intraocular inflammation (Behçet's disease, sarcoidosis, Vogt-Koyanagi-Harada's syndrome, sympathetic ophthalmia), diffuse choroiditis (acute multifocal placoid pigment epitheliopathy, serpiginous choroiditis), and intraocular tumors (leukemia, reticulum cell sarcoma). Behçet's disease,[4] Vogt-Koyanagi-Harada's syndrome[5], Whipple's

disease,[6] sympathetic ophthalmia,[7] and serpiginous choroiditis[8] are diseases easily distinguished from birdshot retinochoroidopathy either by their associated systemic abnormalities or fundus appearance.

Sarcoidosis is a benign, chronic, multisystem disease which manifests posterior uveitis in many different ways.[9] None of our patients had the characteristic fluffy snowball vitreous opacities or the candle-wax drippings frequently seen in this disease. Furthermore, no evidence of systemic sarcoidosis was found in any of our patients. Patient 2 had a Gohn complex on chest X-ray for which a biopsy had been done many years before we saw her. Review of the pathologic specimen showed a caseating granulomatous tubercle consistent with tuberculosis. She also had normal serum angiotensin and lysozyme-converting enzyme levels.

Acute multifocal placoid pigment epitheliopathy is a bilateral diffuse choroiditis that usually occurs in young adults; it is marked by a rapid decrease in visual acuity. The acute lesions usually resolve in a short period of time, evolving on a day-to-day basis. In the final stages of their evolution, the lesions show irregular clumping and pigment proliferation.[10]

Leukemia and reticulum cell sarcoma can produce choroidal and retinal yellow-white, mound-like lesions with an accompanying vitreous infiltrate.[11] Our patients showed no hematologic evidence of leukemia. Furthermore, primary intraocular reticulum cell sarcoma probably does not exist; most tumors so diagnosed are probably non-Hodgkin's lymphomas.[12] The lesions seen in this latter disease are not as diffuse and symmetrical as those seen in birdshot retinochoroidopathy. Additionally, they do not resolve without evidence of multicentricity in other organ systems.

Birdshot retinochoroidopathy appears to be a distinct clinical entity in which choroidal, retinal, and vitreous abnormalities form a characteristic constellation. The marked inflammation in this disease probably indicates an infectious or autoimmune origin, although we have no evidence bearing directly on either possibility. The development of cystoid macular edema is responsible for the loss of central vision in these patients. It appears to be secondary to inflammation, because treatment with corticosteroids early in the course of the disease may be effective in reversing the macular edema.

Summary

Four patients, two men and two women ranging in age from 40 to 70 years, had birdshot retinochoroidopathy. The special features of this entity are diffuse posterior choroidopathy with associated vitritis and retinal vasculitis.

Decreased central vision is caused by cystoid macular edema. In the early course of this disease, the cystoid macular edema may be responsive to corticosteroids. The diffuse choroidopathy is associated with abnormal retinal pigment epithelium and sensory retinal function on electrophysiologic testing. No previously described choroidal or retinal pigment epithelium diseases appear to resemble this entity.

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Ophthalmic miniature

His most remarkable natural gift, however, is his vision. During a game, Bradley's eyes are always a glaze of panoptic attention, for a basketball player needs to look at everything, focusing on nothing, until the last moment of commitment. Beyond this, it is obviously helpful to a basketball player to be able to see a little more than the next man, and the remark is frequently made about basketball superstars that they have unusual peripheral vision. People used to say that Bob Cousy, the immortal backcourt man of the Boston Celtics, could look due east and enjoy a sunset. Ed Macauley once took a long auto trip with Cousy when they were teammates, and in the course of it Cousy happened to go to sleep sitting up. Macauley swears that Cousy's eyelids, lowered as far as they would go, failed to cover his coleopteran eyes.

William Howarth, Editor
The John McPhee Reader,
New York, Random House, Inc., 1976

LIPOFUSCIN OF HUMAN RETINAL PIGMENT EPITHELIUM

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Lipofuscin, the age pigment or "wear and tear" pigment of classical pathology, has gained importance in ophthalmology in recent years owing to several factors: its documented increase in the retinal pigment epithelium with age[1-3]; new experimental evidence that its accumulation in the retinal pigment epithelium is enhanced by diets deficient in vitamin E[4,5]; evidence that it acts as a filter in fluorescein angiography[6-8]; reports that macular retinal pigment epithelium contains a higher concentration of lipofuscin than nonmacular retinal pigment epithelium,[7] although a consistent dip in concentration has been observed in foveal retinal pigment epithelium[3]; and a report that the in vivo autofluorescence demonstrable in vitelliform macular dystrophy and fundus flavimaculatus emanates from lipofuscin.[9]

The morphology of lipofuscin granules varies from one tissue to another, but they all contain lipids and other substances that fluoresce with ultraviolet irradiation.[10,11] Lipofuscin granules of human retinal pigment epithelium differ morphologically from those of brain, heart, testes, and other organs in being relatively uniform in size and, in older individuals, almost uniform in electron density.[2] Biochemical analyses of brain lipofuscin show the presence of complex peroxidized phospholipids,[12] but, to the best of our knowledge, similar analyses of retinal pigment epithelial lipofuscin have not been made.

We report here spectral analyses of extracts of human retinal pigment epithelial cells of two age groups, those under 40 years of age and those over 40 years of age. Fluorescent lipids having the characteristics of lipofuscin were found in the old, but not in the young, pigment epithelial specimens. In discussing our findings, we present a diagram that depicts the multiplicity of factors involved in the formation of peroxidized lipids and of lipofuscin granules. It should serve as a guide to understanding physical and metabolic insults, dietary deficiencies, and other factors that lead to the formation of lipofuscin granules within the cell. We emphasize the particular vulnerability of the retinal pigment epithelium to lipofuscin accumulation because of the normal daily phagocytic load of membranes rich in polyunsaturated fatty acids, combined with the lifelong potential for light-induced free radical damage in these cells.

MATERIAL AND METHODS

Human eyes, obtained through two Oregon eye banks within 24 hours of

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death, were bisected at the ora serrata. Only those eyes showing no gross pathology of the posterior segment were used. The vitreous and neural retina were removed and the pigment epithelial cells then brushed out in distilled water. Cells from individuals over 40 years of age were pooled separately from those under 40 years of age. The suspensions were frozen at −18°C until all of the eyes of each group were accumulated. The preparations were then freeze-dried and afterward extracted in 10 ml of chloroform-methanol (2:1, v/v) at room temperature in a microhomogenizer. The crude extract was washed twice with water,[11] and the chloroform phase then collected and evaporated to dryness, first in a rotary evaporator and then under a stream of nitrogen. The dried extracts were dissolved in 2.0 ml of chloroform-methanol (1:9, v/v) and chromatographed on 0.9 × 20-cm Sephadex LH-20 columns.[11] This method is particularly applicable to the pigment epithelium because it provides effective separation of lipofuscin from vitamin A, which is present in high concentrations in the pigment epithelium.[13] Both lipofuscin and vitamin A are fluorescent and, moreover, have overlapping spectra. Therefore, it is essential to separate them first in tissue sources such as retinal pigment epithelium where they would be extracted together. Fractions of 1.0 ml each were eluted from the columns with chloroform-methanol (1:9, v/v). The fluorescence of each effluent was determined in a Turner Photofluorometer Model 111, using filter No. 7-60 for excitation (365 nm) and filter No. 47B-2A for emission (435 nm).

Fluorescence spectra of pooled fractions obtained from the Sephadex columns were determined with a fluorescence spectrophotometer. Thin-layer chromatography was carried out on Silica gel G coated plates, with retinol and retinyl esters as standards.[13]

A piece of equatorial retina was excised from one eye of each pair of eyes and fixed for ultrastructural evaluation as described previously.[2] Retinal pigment epithelium retained its overall integrity and surprisingly good morphology for up to 24 hours. Some specimens were prepared without osmic acid fixation to illustrate the nonosmiophilic components of lipofuscin granules.

Results

The retinal pigment epithelium of infants contained no recognizable lipofuscin (Fig. 1) despite the presence of fluorescent granules when the same specimen was examined by fluorescence microscopy.[2] Secondary lysosomes present within these cells apparently contain some fluorogens, which is not surprising since these organelles are the forerunners of lipofuscin granules. By the fourth decade of life (Fig. 2), typical lipofuscin granules are present in the subapical cytoplasm. By the sixth decade (Fig. 3), the number of lipofuscin granules usually equals or surpasses the number of melanin granules, and by the ninth decade

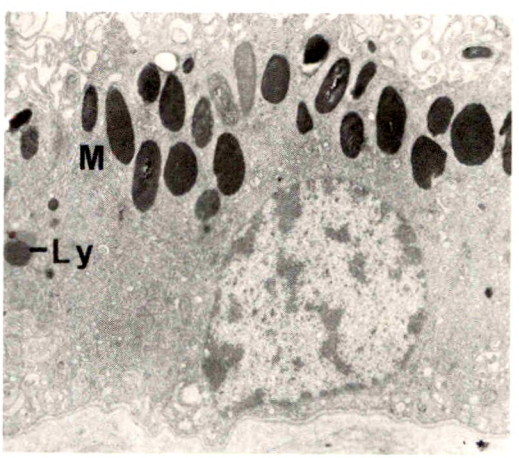


Fig. 1 (Feeney-Burns, Berman, and Rothman). Retinal pigment epithelium from a 1-year-old child. Melanin (M) is the only pigment present in the cytoplasm. Secondary lysosomes (Ly) are the forerunners of lipofuscin granules (× 5,300).

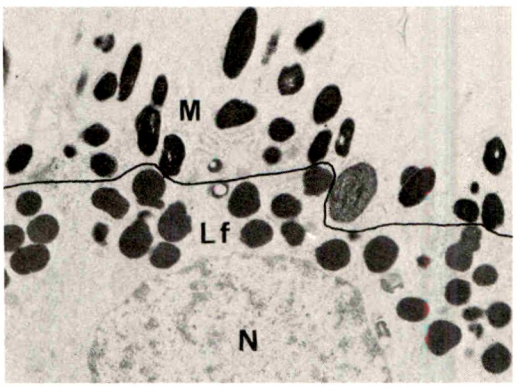


Fig. 2 (Feeney-Burns, Berman, and Rothman). Retinal pigment epithelium of a 30-year-old individual. A few lipofuscin (Lf) granules are present (× 75,00).

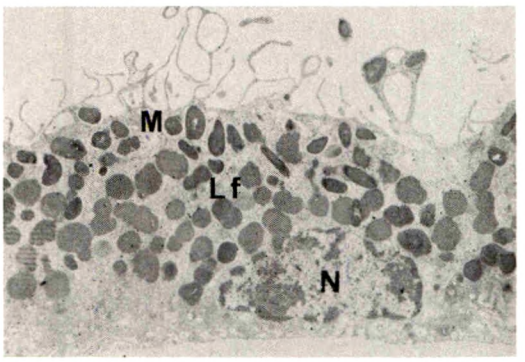


Fig. 3 (Feeney-Burns, Berman, and Rothman). Retinal pigment epithelium of a 50-year-old individual. Lipofuscin is abundant (× 5,300).

(Fig. 4), in every specimen examined, lipofuscin granules outnumbered melanin granules. Moreover, in the older eyes, fusion of lipofuscin with melanin created abundant melanolipofuscin granules.[2]

Chloroform-methanol extracts of pigment epithelial cells subjected to gel chromatography show three distinct fluorescent peaks (Fig. 5) in both young (panel A) and old (panel B) eyes. Peak I was several-fold higher in old eyes than in young ones, but peaks II and III were approximately the same in the two age groups.

The fractions from human pigment epithelium eluting in peak I, A and B (Fig. 5) were pooled and concentrated for examination of fluorescent spectra. In the extracts from young individuals, no

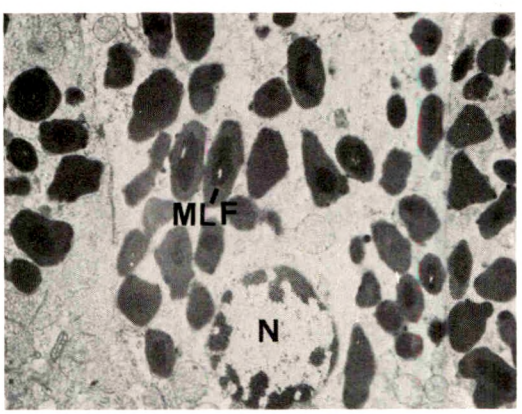


Fig. 4 (Feeney-Burns, Berman, and Rothman). Retinal pigment epithelium of an 80-year-old individual. Lipofuscin granules predominate. Melanin is sparse, and many granules are a composite of lipofuscin and melanin (MLF) (× 5,300).

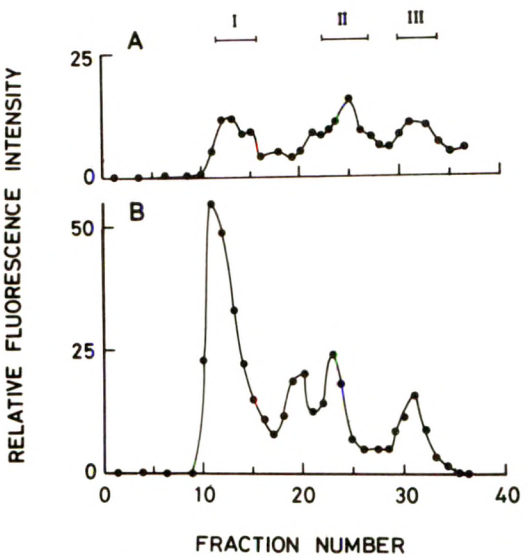


Fig. 5 (Feeney-Burns, Berman, and Rothman). Elution profiles after gel filtration on Sephadex columns of chloroform-methanol extracts of pigment epithelium from human eyes under 40 years of age (panel A) and over 40 years of age (panel B). Three fluorescent peaks are seen, but peak I (lipofuscin) is quantitatively greater in the retinal pigment epithelium of older individuals.

clearly recognizable spectra could be obtained. In contrast to this, however, the pigment epithelial extracts from individuals over 40 years of age had fluorescent spectra characteristic of lipofuscin. As shown in Figure 6, there was a sharp excitation maximum at 360 nm (using emission at 430 nm) and, additionally, a shoulder from 340 to 355 nm. The emission maximum (using excitation of 360 nm) appeared at 430 nm.

The fractions in peak II of both young (Fig. 5, panel A) and old (Fig. 5, panel B) individuals were shown by fluorescence excitation and emission spectra to consist of vitamin A. Further examination of the fraction by thin-layer chromatography showed, as would be expected,[13] that it was mainly in the form of an ester with an R_f value similar to that of the reference marker, retinyl palmitate. The material in the third peak, present in both age groups (Fig. 5), was not examined further. It appears in a similar eluting position on Sephadex columns as the low-molecular-weight fluorescent compounds observed by Csallany and Ayaz[11]; they also were unable to identify it.

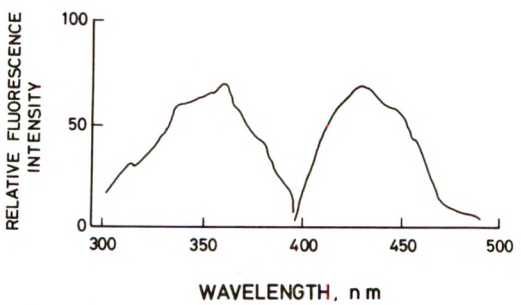


Fig. 6 (Feeney-Burns, Berman, and Rothman). Fluorescent spectra of pigment epithelium from individuals over 40 years of age. The material examined consisted of pooled fractions from peak I (Fig. 5, panel B). Retinal pigment epithelium extracts from elderly individuals had fluorescent spectra characteristic of lipofuscin (excitation and emission at 360 and 430 nm, respectively). No spectra could be obtained from the comparable fractions obtained from individuals under 40 years of age.

Discussion

These studies show that fluorescent substances in human pigment epithelium extractable with chloroform-methanol can be resolved into two principal components. Lipofuscin was identified by characteristic fluorescence spectra in pigment epithelium from individuals over 40 years of age; it was not clearly identifiable in individuals under 40 years of age. The second component, retinyl ester, was present in pigment epithelium of all age groups, as would be expected.

The morphology of the retinal pigment epithelium cells correlates well with the biochemical data; young pigment epithelium had little or no lipofuscin (Figs. 1 and 2), whereas old pigment epithelium was engorged with lipofuscin granules (Figs. 3 and 4).

Lipofuscin formation is a complex, multifaceted biological process. Lipid peroxidation, a complex process occurring in all cell membranes and involving the interaction of oxygen-derived free radicals with polyunsaturated fatty acids, is an essential step. Cellular processes for controlling or minimizing lipid peroxidation formation involve many different enzymes, cofactors, and vitamins. Diverse factors such as light, oxygen, and trace elements also enter the picture at different molecular levels.

As an aid in formulating a working hypothesis of lipofuscin formation, we have integrated into a single scheme (Fig. 7) a large body of information derived from a variety of chemical, biochemical, and morphological disciplines. It is color-coded in order to show the specific points of entry of oxygen (green) on the one hand, and to distinguish between free radical reactions (red), enzymatic reactions (blue), and nonenzymatic reactions (black) on the other.

It is known from studies of a variety of cell types such as neurons, cardiac mus-

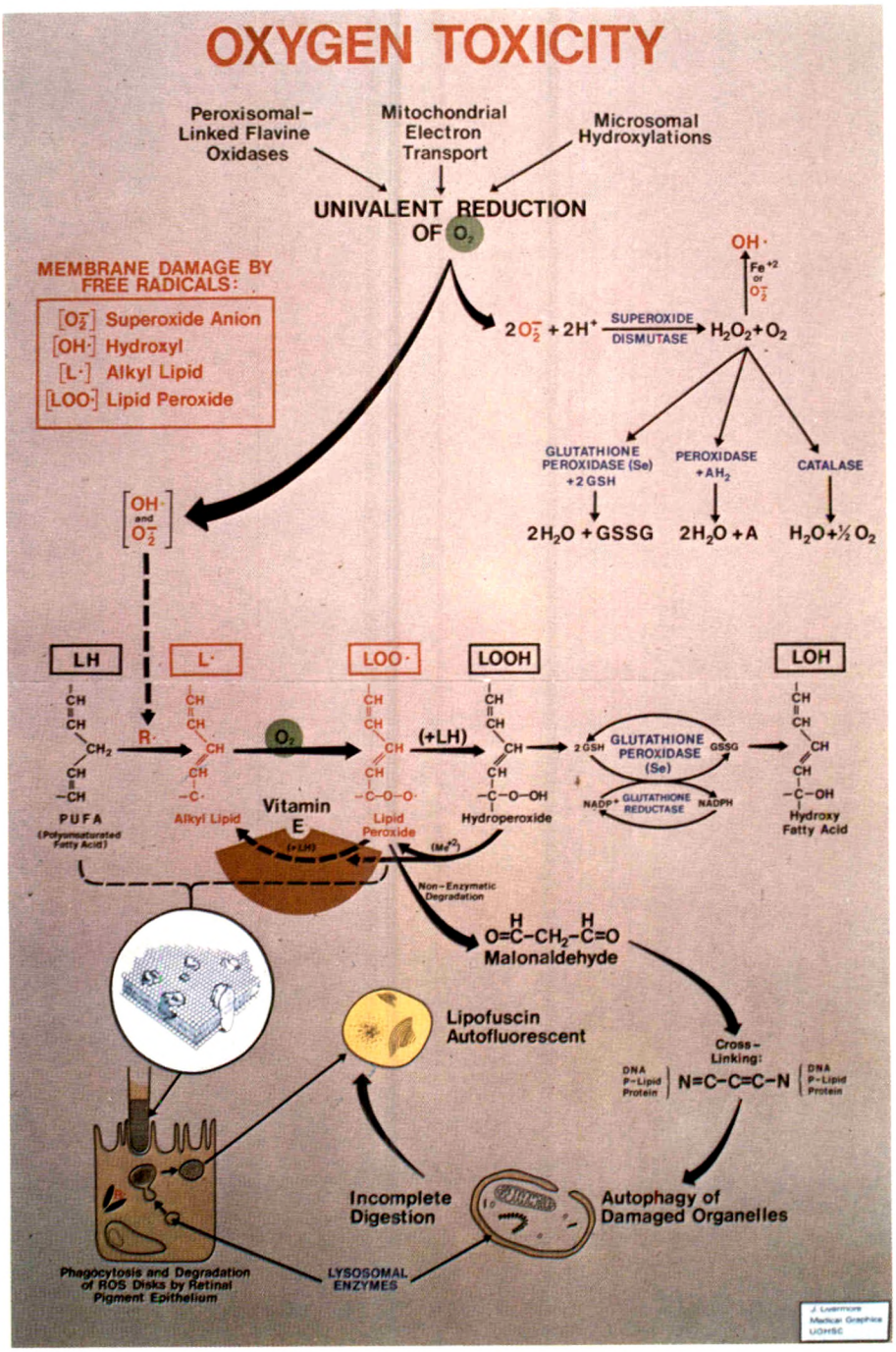


Fig. 7 (Feeney-Burns, Berman, and Rothman). The formation of lipofuscin, shown in the lower center of the diagram in the autofluorescent golden yellow color seen by fluorescence microscopy. Substances appearing in red are free radicals, whereas atmospheric oxygen appears in green. Enzymes which function in the protective avenues appear in blue. The fluid mosaic model of membrane structure is white. Photoreceptor outer segment membranes contain the polyunsaturated fatty acids (PUFA) that become peroxidized and eventually converted to lipofuscin. ROS indicates rod outer segments.

cle, testes, and others that lipofuscin is formed within lysosomes after autophagic sequestration of damaged or aged organelles,[14-16] as depicted in the lower right and center of Figure 7. The retinal pigment epithelium is a special case because lipofuscin granules in these cells arise mainly from phagocytosis of photoreceptor outer segments,[17,18] as shown schematically in the lower left part of Figure 7. The key intermediate, regardless of what tissue we are considering, is malonaldehyde, whose aldehydic group bestows upon it properties similar to formaldehyde or glutaraldehyde, the fixatives used in preservation of cell structure for morphologic examination. In the case of living cells, malonaldehyde is the principal and most characteristic breakdown product of lipid peroxidation. It is highly reactive and forms irreversible cross-links (through the formation of Schiff bases) in a completely nonspecific manner with the amino groups of a wide variety of macromolecules such as DNA, phospholipids, and proteins.

But how do lipids become peroxidized? The central part of the panel in Figure 7 shows the chemical structure of a hypothetical portion of a polyunsaturated fatty acid (PUFA), which is present as a component of the lipid bilayer of nearly all cell membranes. Rod outer segments contain a large concentration of highly unsaturated fatty acids, for example, about 50% docosahexaenoic acid (22:6).[19,20] Peroxidation of these unsaturated bonds can be initiated by several types of free radicals, resulting in the formation of an alkyl lipid ($L^{\bullet}$), shown in red in Figure 7. This is a lipid free radical which, in the presence of oxygen (shown in green), is peroxidized to another lipid free radical, $LO_2^{\bullet}$. The latter, being highly unstable, is capable of acting on neighboring polyunsaturated fatty acid molecules in the cell membrane, converting them, in turn, to free radicals. A chain reaction is thus initiated which spreads through the lipid layer, creating a membrane lesion.

If this process were to proceed uncontrolled, few membranes would remain intact in living organisms. We know that this is not the case because the lipid peroxide free radical ($LO_2^{\bullet}$) can be disposed of by a number of different cellular mechanisms, depending on the microenvironment of the molecule. One principal agent is α-tocopherol, which acts as a free radical sink. Vitamin E is a powerful antioxidant which is capable of terminating or interrupting the auto-oxidative chain reaction by absorbing the unpaired free radical electron. The importance of vitamin E in prevention of lipid peroxidation and lipofuscin formation was demonstrated by Fletcher, Dillard, and Tappel[10] and Reddy and co-workers[21] nearly a decade ago.

The lipid peroxide free radical ($LO_2^{\bullet}$) can be converted by an alternate pathway to a hydroperoxide, that is, be "neutralized" by interaction with a native polyunsaturated fatty acid molecule (LH). Hydroperoxides can enter into auto-oxidative chain reactions if certain divalent metal ions are present or, in the presence of the selenium-containing enzyme glutathione peroxidase, it can be converted to harmless hydroxy fatty acid (LOH).

Free radicals, shown at the top of Figure 7, derive from the univalent reduction of oxygen, an alternative to the major pathway of oxygen reduction by two-electron transfer through the cytochrome system. The most studied of the free radicals are superoxide anion and hydroxyl free radical, although singlet oxygen may also be involved. Fridovitch[22] has shown that all oxygen-metabolizing organisms have evolved protective mechanisms that either minimize the production of free radicals or destroy them as rapidly as they are formed. These reactions are diagrammed in the upper right

corner of Figure 7. Superoxide dismutase converts superoxide anion to hydrogen peroxide, which in turn can be reduced to water by the action of (a) glutathione peroxidase with glutathione acting as hydrogen donor, (b) peroxidase, with ascorbic acid commonly the H^+ donor, or (c) catalase, whose heme moiety is the H^+ donor. Further details of these reactions and primary references have been published.[22,23]

The role of melanin granules should be emphasized here also, because they probably are important in protecting pigment epithelial cells from free radicals ($R^{\cdot}$). Although the mechanism whereby melanin performs its photoprotective function is not well understood, one commonly held view is that the semi-quinone structure of melanin is able to absorb free radicals directly, leaving the structure of the melanin polymer unchanged.[24] Loss of melanin, for example, by depigmentation of the retinal pigment epithelium, could enhance light-induced free radical damage in these cells. A vicious cycle encompassing demelanization, increased free radical damage, increased lipofuscin formation, and increased lipofuscin-melanin fusion could well set the stage for age-related degenerations of the retina such as senile macular degeneration.

Finally, the apparent role of dietary lipids in the accumulation of lipofuscin in pigment epithelial cells should be reemphasized. Studies on rat liver indicate that the proportion of polyunsaturated fatty acids in the cell membranes reflects the polyunsaturated fatty acid composition of the diet.[25] Furthermore, the lipid peroxide content of the liver cell membranes and the rate of malonaldehyde formation increases directly with the degree of unsaturation of the ingested lipids. If dietary lipids similarly affect the composition of the membranes of the retina, then ingestion of lipids rich in polyunsaturated fatty acids may increase the susceptibility of retinal membranes to lipid peroxide formation, malonaldehyde production, and lipofuscin accumulation in the retinal pigment epithelium. In earlier dietary studies, Fletcher, Dillard, and Tappel[10] and Reddy and associates[21] showed that tissues of rats fed diets high in polyunsaturated fats and low in vitamin E accumulated twice the extractable fluorescent pigments as those fed the same diet supplemented with vitamin E. Also, importantly, histologic studies show that monkeys[26] and rats[4,5] fed similar dietary regimens gradually accumulate fluorescent lipofuscin granules in the retinal pigment epithelium. Thus, research on the causes of degenerative age changes in the human retina might profitably be focused on our modern diet.

Summary

Analysis of the fluorescent spectra of chloroform-methanol extracts of human retinal pigment epithelium confirmed the presence of lipofuscin pigments in the pigment epithelium of older individuals. Similar fractions in the pigment epithelium of young individuals were present in insufficient quantities for spectral analysis. Electron microscopy of the pigment epithelium of these eyes showed few or no lipofuscin granules in young eyes but large numbers in older eyes.

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BILATERAL EXUDATIVE RETINAL DETACHMENT IN ECLAMPSIA WITHOUT HYPERTENSIVE RETINOPATHY

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Duke-Elder and Dobree[1] stated that von Graefe was the first to describe retinal detachment as an ocular complication in toxemia of pregnancy. Many other authors have subsequently described this condition [2-4] and, except for a single instance reported by Gitter and associates,[5] there has been unanimous agreement that retinal detachment in eclampsia is always accompanied by signs of severe hypertensive retinopathy.

We examined and treated a patient with eclampsia and bilateral retinal detachment without signs of hypertensive retinopathy. Fluorescein angiograms performed during the acute stage of the disease and after recovery clarify several aspects of this disease.

CASE REPORT

A 23-year-old woman was hospitalized in the 28th week of her first pregnancy, after the sudden onset of convulsions and loss of consciousness. Routine obstetric examinations performed monthly before hospitalization had disclosed nothing unusual. Blood pressure on admission was 150/95 mm Hg. Pertinent laboratory findings included the following values: hemoglobin, 12.9 g/100 ml; hematocrit, 42%; serum urea nitrogen, 42 mg/100 ml; and creatinine, 0.95 mg/100 ml. Quantitative examination for proteinuria showed a daily excretion of 3.5 g. Ophthalmic examination showed a flat retina with no signs of hypertensive retinopathy. Eclampsia was diagnosed and the patient was given 10-ml intravenous injections of a 10% solution of magnesium sulfate every four hours until her blood pressure was controlled.

Three days later, the patient complained of severe deterioration of visual acuity. An ocular examination disclosed similar findings in both eyes. Visual acuity was light perception with good projection. Biomicroscopy of the anterior segment yielded no positive findings. Intraocular pressure was 16 mm Hg. Ophthalmic examination showed high bullous retinal detachment, involving the temporal retinas and maculas in both eyes.

We found no retinal tears or holes. There were no signs of hypertensive retinopathy. Figures 1 and 2 show the bullous detachment pattern of the left eye. The same day, signs of fetal distress appeared and a caesarean section was performed. A 800-g fetus was delivered, but survived only three days.

When we examined the patient 12 hours after the operation, the area of retinal detachment was smaller than at the first examination, and had shifted from the temporal to the upper halves of both retinas.

The next examination, done 36 hours after the caesarean section, showed such marked flattening of the retinas that the detachment was limited mainly to the posterior pole in both eyes and the retinas appeared edematous (Fig. 3, top left). Again, there were no findings compatible with hypertensive retinopathy. Fluorescein angiography demonstrated normal retinal vasculature. The arteriovenous phase showed many small, randomly scattered subretinal foci of active fluorescein leakage (Fig. 3, top right). These spot-like foci continued to increase in size and

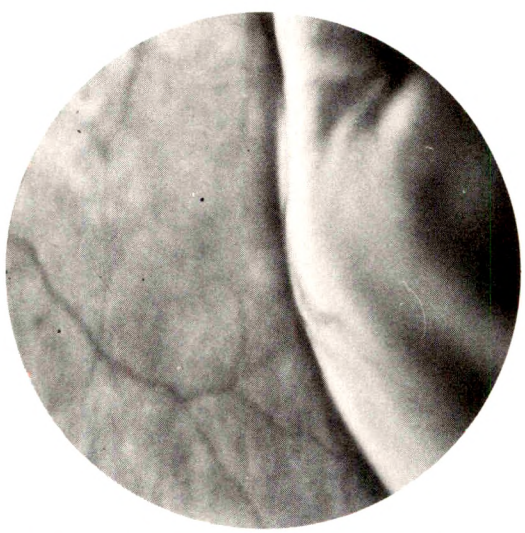


Fig. 1 (Oliver and Uchenik). Bullous retinal detachment in the left eye without signs of hypertensive retinopathy.

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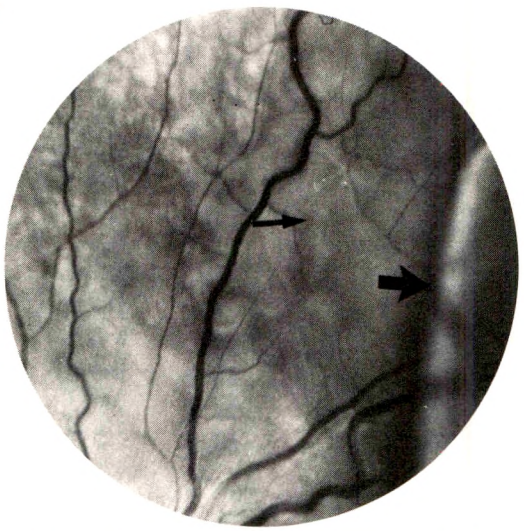


Fig. 2 (Oliver and Uchenik). Bullous retinal detachment in the left eye (large arrow) borders area of flat detachment (small arrow) and flat retina without signs of hypertensive retinopathy.

to coalesce (Fig. 3, bottom left); thus, in the late stages of angiography, we observed marked pooling of dye in the subretinal space (Fig. 3, bottom right).

We performed follow-up ocular examinations and fluorescein angiography every two weeks. There was a gradual improvement of the fundus appearance and visual acuity in both eyes. Six weeks after the first examination, her visual acuity was 6/7.5 (20/25) in both eyes. There was diffuse mottling throughout the fundi, but mainly in the posterior pole (Fig. 4, left). Fluorescein angiograms showed similar patterns in both eyes. The mottling of the retinal pigment epithelium was characterized by many small, irregular hyperfluorescent and hypofluorescent foci (Fig. 4, right). There was no leakage of dye from the choroid, and no subretinal fluid (Fig. 4, right). The patient's general condition was good. Her blood pressure was 110/70 mm Hg, and results of urinanalysis were normal.

Discussion

In 1929, Fry[2] reported two cases of retinal detachment in eclampsia and reviewed 19 previously reported cases. Few cases were reported until 1968, when Gitter and associates[5] described a patient with many bilateral cyst-like elevations of the posterior pole, without marked signs of retinal angiospasm. Duke-Elder and Dobree[1] stated that the ophthalmoscopic sign of toxemia is hypertensive retinopathy, demonstrated by severe constriction of retinal arteries, flame-shaped hemorrhages, cotton-wool exudates, edema of the retina, and, in advanced cases, optic disk edema. They emphasized that these signs are likely to occur when the systolic blood pressure rises above 160 mm Hg and the diastolic blood pressure above 100 mm Hg, and are very marked when these values reach 200/130 mm Hg. Our patient's blood pressure did not exceed 150/95 mm Hg, which may explain the complete absence of retinal angiospasm.

The ophthalmoscopic findings in our case and in that reported by Gitter and associates,[5] lead us to assume that retinal detachment in eclampsia is not necessarily related to the presence or severity of hypertensive retinopathy. This observation differs from the assumption of Moore[6] and Friedenwald[7] that retinal detachment in toxemia is related to retinal vascular damage. Ballantyne and Michaelson[8] assumed the presence of massive exudation from the choroidal vasculature, causing elevation of the retina, an opinion strongly supported by the angiograms of our patient. Leakage of dye from the choroid and its pooling in the subretinal space in the acute stages of the disease may be related to pigment epithelium defects. The existence of these defects in the acute stages of the disease is strongly suggested by the mottled appearance of the fundus and the window-effect pattern of the angiograms done during the convalescent period. The pattern in our case, although it was not of inflammatory origin, and its similarity to those reported by Kanter and Goldberg[9] emphasize the exudative nature of retinal detachment in toxemia of pregnancy and its relationship to choroidal vascular pathology.

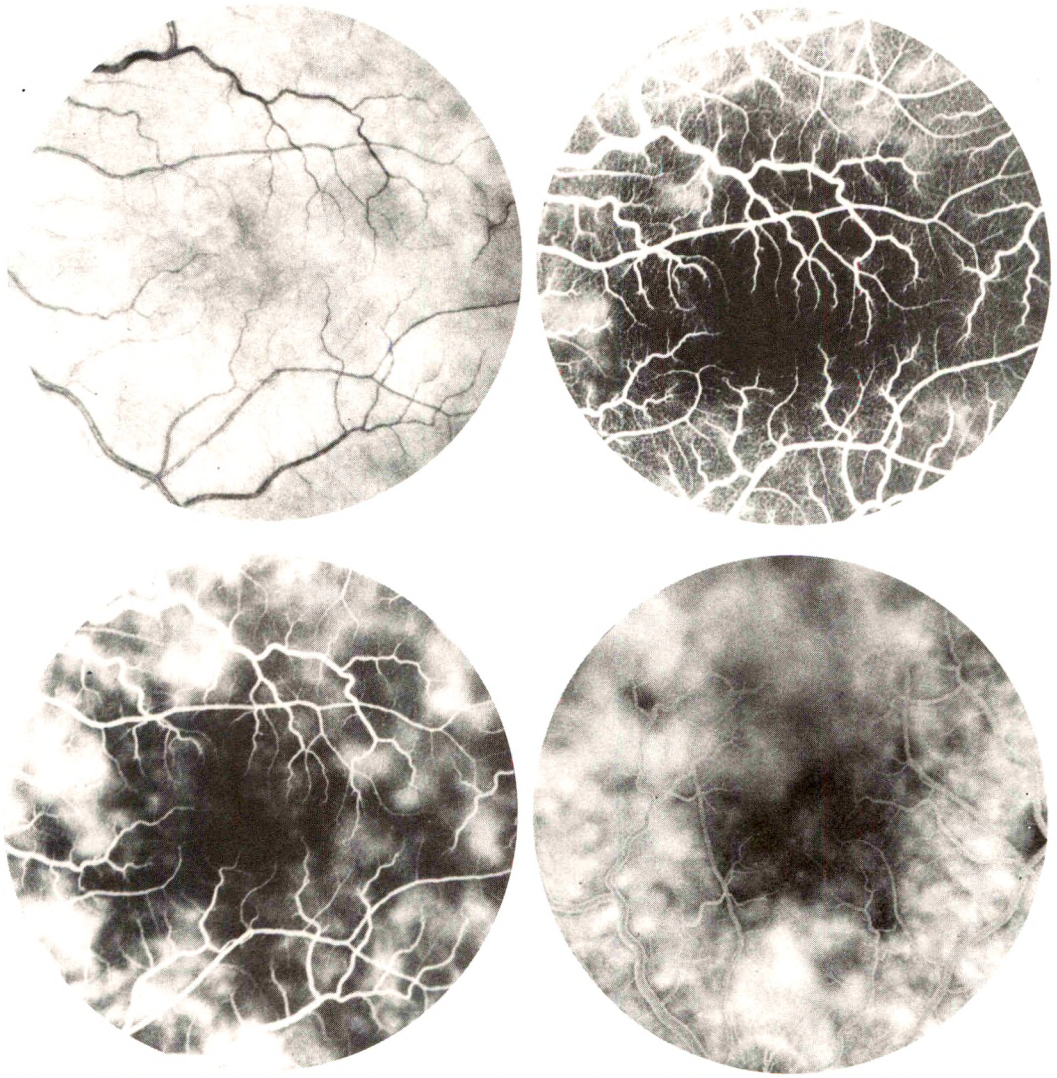


Fig. 3 (Oliver and Uchenik). Top left, Macula of the left eye 36 hours after delivery. There is a flat, exudative retinal detachment with no signs of hypertensive retinopathy. Top right, Arteriovenous phase shows many foci of subretinal fluorescein leakage but no hypertensive retinopathy. (The right eye appeared essentially the same.) Bottom left, At a later stage, the foci of subretinal leakage show enlargement and coalescence. Bottom right, Late stage shows marked pooling of dye in the subretinal space.

Others have considered the progression of retinal angiospastic vascular changes a sign of increasing severity of toxemia[3,10] and have correlated them with fetal mortality.[10,11] Moreover, Sadowsky, Serr, and Landau[10] have used these changes as a guideline for termination of pregnancy. It is doubtful whether retinal detachment alone should be considered a sign of increasing severity of toxemia. Since this condition is rare, only the assessment of additional cases will clarify the matter.

However, retinal detachment in

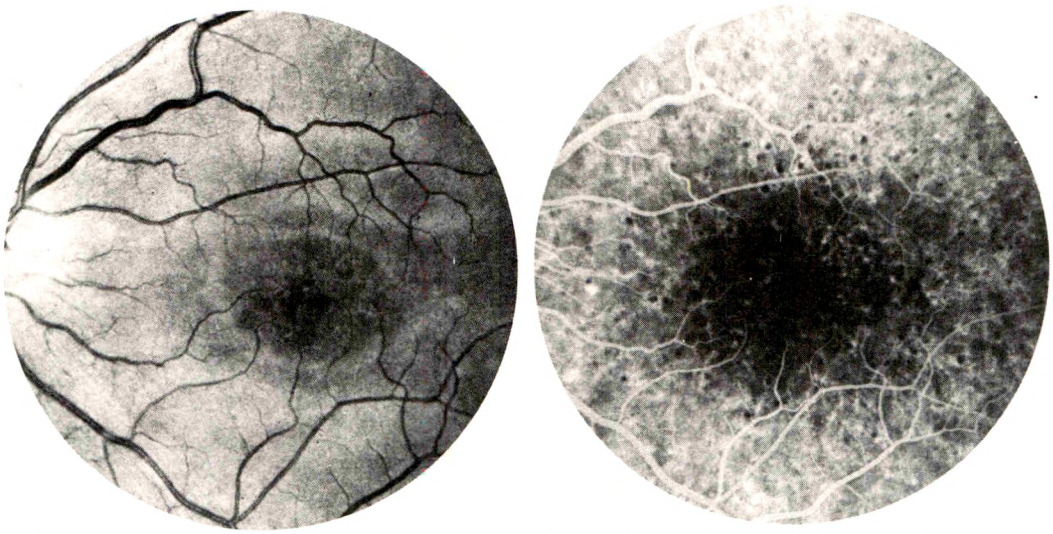


Fig. 4 (Oliver and Uchenik). Left, Fundus of the left eye 45 days after delivery. There is mottling in the area of the macula. (The right eye appeared essentially the same.) Right, Fluorescein angiogram done 45 days after delivery shows irregular mottling, secondary to diffuse pigment migration in the choroid and in the pigment epithelium of the retina.

eclampsia may well be an indication for termination of pregnancy because of its potential threat to the mother's visual acuity. Since surgical treatment of retinal detachment in similar cases has proved disastrous,[12] it may well be that in cases where there is successful medical control of the toxemia but unsatisfactory regression of the retinal detachment, termination of pregnancy will be based on ophthalmologic indications.

Summary

A patient with bilateral bullous retinal detachment caused by eclampsia of pregnancy had no signs of hypertensive retinopathy. Fluorescein angiograms during the acute stage of the disease showed normal retinal vasculature and widespread choroidopathy, probably of vascular origin, causing massive exudation to the subretinal space. Prompt recovery with good visual acuity occurred after the pregnancy ended. The only residual findings were diffuse mottling of the fundus secondary to pigmentary migration in the choroid and in the pigment epithelium of the retina disclosed by fluorescein angiography.

Termination of pregnancy may be indicated in those cases where the systemic signs of toxemia are controlled medically but there is no flattening of the detached retina.

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Fifty years ago this month in THE JOURNAL:

In a series of twelve cases of ophthalmia neonatorum, two were gonorrheal blennorrheas, six were silver nitrate blennorrheas, and four represented what is known as inclusion blennorrhea.

Material from the eye of such a case was inoculated into the scarified conjunctiva of a macacus rhesus and a reaction resembling inclusion blennorrhea of the newborn developed, although no epithelial-cell inclusions were found in the conjunctival scrapings.

There is a distinct type of conjunctivitis affecting principally the newborn, which ultimately heals without scarring. It is characterized by a much later onset than occurs in gonorrheal blennorrhea, and by the presence of follicles and of inclusion bodies. This disease may be transmitted by contact.

The condition is probably more common than is generally stated, because suitable methods of examination have not been employed.

The significance of epithelial-cell inclusions is as yet purely hypothetical.

William M. James, M.D., Inclusion blennorrhea
Am. J. Ophthalmol. 13:1088, 1930

ULTRASTRUCTURAL FEATURES OF PROGRESSIVE IDIOPATHIC EPIRETINAL MEMBRANE REMOVED BY VITREOUS SURGERY

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Thin, nonvascularized (simple) epiretinal membranes composed of a delicate glial-cell layer on the inner retinal surface are a common finding in eyes obtained at autopsy.[1,2] More complex epiretinal membranes that cause surface wrinkling of the retina have been observed in more than 20% of eyes from elderly persons.[3] Epiretinal membranes are rare in young patients and severe visual loss is infrequent in eyes with no history of inflammatory or vascular disease, surgery, or trauma.[4-9]

The cause of nonvascularized epiretinal membranes is unknown but is believed to be related to naturally occurring or acquired defects in the internal limiting membrane of the retina.[1,10] Previous ultrastructural studies of idiopathic epiretinal membranes with mild macular puckering have shown that the epiretinal tissue is fibroglial in nature.[1,11]

Whether the ultrastructural features of progressive idiopathic epiretinal membranes differ from those of simple or complex epiretinal membranes or from secondary epiretinal membranes associated with various ocular diseases is unclear.

We studied the ultrastructural features of a unilateral, idiopathic epiretinal membrane removed by vitreous surgery in a young patient with progressive visual loss and compared the ultrastructural features of this specimen to those of simple and secondary epiretinal membranes.

CASE REPORT

A 27-year-old woman experienced a sudden reduction of visual acuity in her left eye in September 1973. The visual acuity at that time was 6/6 (20/20) in her right eye and 6/15 (20/50) in her left eye. There was a nonvascularized epiretinal membrane affecting the left macula. This membrane was about 0.33 disk diameter in size and it was located slightly inferotemporal to the macula. The membrane was causing tangential traction on the surrounding retina, as shown by radial striae in the internal limiting membrane of the retina (Fig. 1, top left). Some of these striae involved the macula, probably explaining the reduced visual acuity. We found no other abnormalities in the left eye, and the right eye was entirely normal. The vitreous gel was normal in both eyes and ophthalmoscopy showed no posterior vitreous detachment.

We examined the patient periodically without prescribing therapy. In 1975 visual acuity in the left eye had improved to 6/9 (20/30). However, the epiretinal membrane appeared to have enlarged slightly, the retinal striae persisted, and there was slightly increased distortion of the retinal vessels surrounding the macula (Fig. 1, top right). Visual acuity in the left eye improved to 6/6–1 (20/20–1) by December 1978, despite further changes in the epiretinal membrane. At that time the membrane appeared to have enlarged inferotemporally, although the edge nearest the macula was less prominent and may have partially separated from the inner retinal surface (Fig. 1, bottom left). Even so, the striae involving the macula and the distortion of the retinal vessels appeared unchanged.

The patient experienced sudden additional visual loss in April 1979. Best corrected visual acuity was only 6/60 (20/200), and there was a striking change in the appearance of the membrane (Fig. 1, bottom right). The membrane appeared larger and caused

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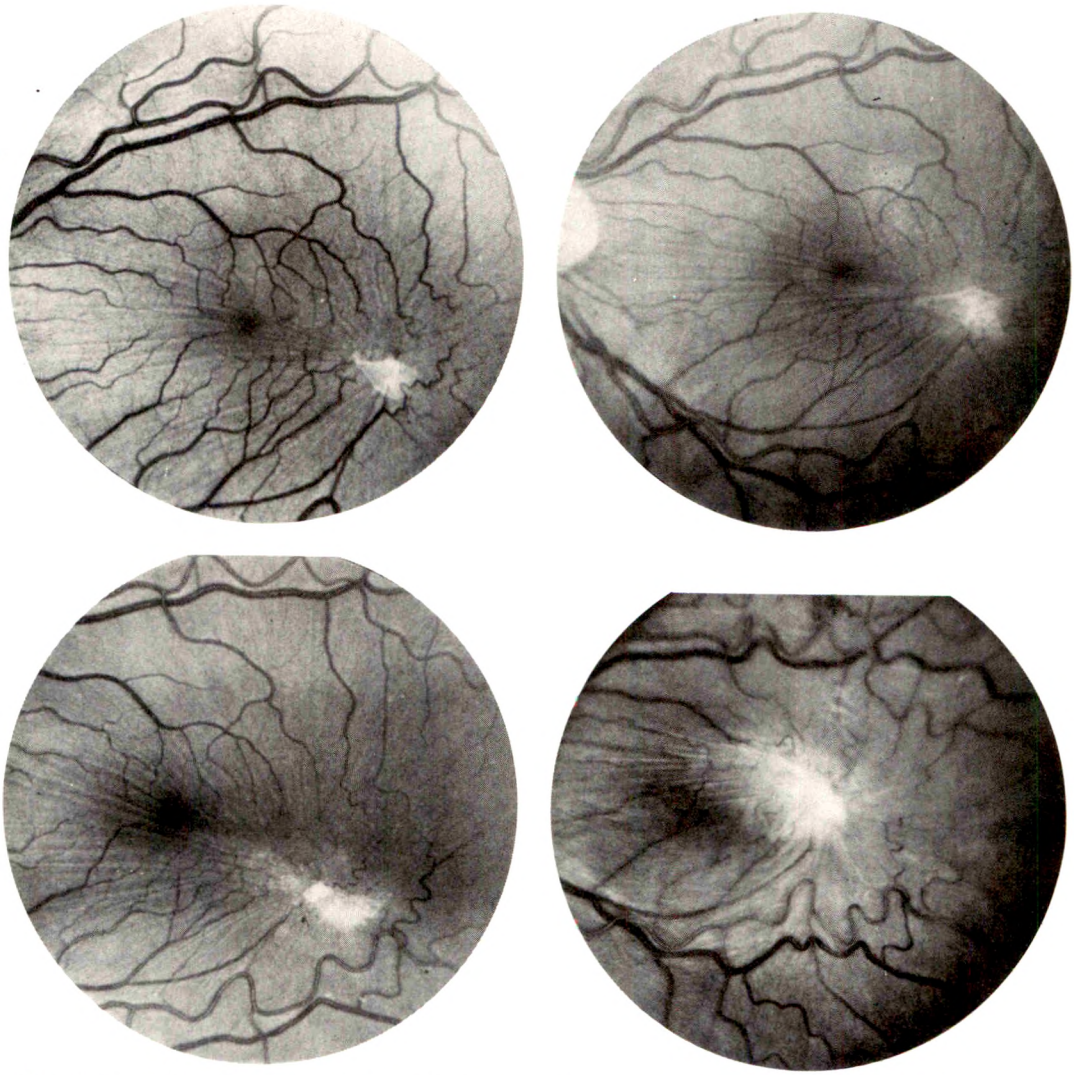


Fig. 1 (Kampik and associates). Top left, Nonvascularized epiretinal membrane over left macula in September 1973. Radial striae of the internal limiting membrane of the retina are present. Visual acuity was 6/15 (20/50). Top right, Slight enlargement of epiretinal membrane in 1975; visual acuity was 6/9 (20/30). Bottom left, Further enlargement of epiretinal membrane in December 1978; visual acuity was 6/6-1 (20/20-1). Edge nearest the macula may have partially separated from the inner retinal surface. Bottom right, Sudden visual loss to 6/60 (20/200) occurred in April 1979. There is severe centripetal traction with marked distortion of the center of the macula. Note the small cluster of superficial retinal hemorrhages above the superotemporal arcade superior to the macula.

severe centripetal traction with marked distortion of the center of the macula, which was pulled toward the nasal edge of the membrane. There was low detachment of the posterior retina caused by traction from the membrane, and a small cluster of superficial retinal hemorrhages was present just above the superotemporal arcade superior to the macula. The right eye continued to be normal, and the patient's general health was excellent.

We advised vitreous surgery to remove the epiretinal membrane because of the progressive nature of the disorder, the severe distortion of the posterior retina, and the marked visual loss. We performed a partial posterior vitrectomy and removal of the epiretinal membrane on May 4, 1979, using a technique previously described.[12] The operation was uncomplicated and the epiretinal membrane was readily separated from the inner retinal surface with

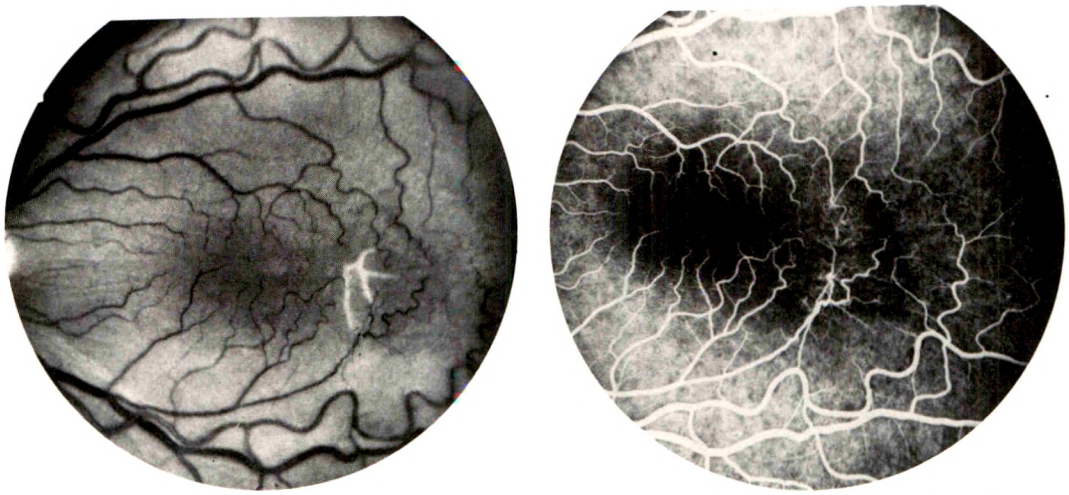


Fig. 2 (Kampik and associates). Left, There is much less distortion of the macula after removal of the epiretinal membrane. Visual acuity was 6/7.5 (20/25). The white dendritic pattern temporal to the center of the macula was unchanged during the first several months postoperatively. Right, Fluorescein angiogram showing persistent distortion of the small retinal vessels temporal to the macula in the area where the epiretinal membrane was.

a specialized vitreoretinal pick.[13] We removed the epiretinal membrane from the eye without fragmentation and placed it in fixative for later ultrastructural analysis.

Immediately after removing the epiretinal tissue, we found a white dendritic pattern in the deep retinal layers temporal to the center of the macula. We presumed this was intraretinal or subretinal scarring caused by the marked distortion and folding of the retinal tissue caused by the epiretinal membrane.

Postoperatively, the eye healed properly without complications. Visual acuity improved to 6/21 (20/70) within one week of surgery, and further improved to 6/9 (20/30) six weeks after the operation. The white dendritic pattern temporal to the center of the macula appeared unchanged during the first several postoperative months (Fig. 2, left).

Six months after the operation, best corrected visual acuity in the left eye was 6/7.5 (20/25) with -1.25 $+0.75 \times 105$. There were some irregularities of the inner retinal surface overlying the white dendritic pattern temporal to the macula when studied by ophthalmoscopy. Fluorescein angiography showed persistent distortion of the small retinal vessels and capillaries temporal to the macula in the area of the previous epiretinal membrane (Fig. 2, right). Mild fluorescein leakage from these vessels occurred, with staining of the adjacent retinal tissue but without involvement of the center of the macula.

Material and Methods

We placed the epiretinal membrane in a phosphate-buffered mixture of 1% glutaraldehyde and 4% formaldehyde solution for fixation. We then put the specimen into a 2.5% glutaraldehyde solution for two hours and later postfixed it with 2% phosphate buffered osmium tetroxide. After standard dehydration, the specimen was embedded in epoxy resin.

For phase-contrast microscopy, we stained semithin 1-μ sections with paraphenylenediamine. For electron microscopy, we stained ultrathin sections with uranyl acetate and lead citrate and examined them with a transmission electron microscope.

Results

Light microscopic examination of semithin sections by phase-contrast and electron microscopy showed a fibrocellular membrane with a contiguous large serpentine-shaped fragment of gathered internal limiting membrane of the retina (Fig. 3). The internal limiting membrane had no discontinuities in the removed segment and fine blood vessels were present on the retinal side of the membrane. The fibrocellular epiretinal mem-



Fig. 3 (Kampik and associates). Low-power electron micrograph of the removed epiretinal membrane showing a large serpentine-shaped fragment of gathered internal limiting membrane (arrows). A fibrocellular membrane composed of spindle-shaped and plumper cells is present on the inner surface of the internal limiting membrane (×1,900).

brane measured up to 100 μ in thickness and was avascular. Most of the cells were spindle-shaped but many cells had a plumper appearance.

With transmission electron microscopy, we distinguished three cell types and three types of collagenous fibrils. The predominant cells were spindle-shaped fibrocyte-like cells (Fig. 4). Those cells showed no orientation, polarity, or continuous basement membrane. They were characterized by large, elongated, irregular, and often markedly indented nuclei with coarse heterochromatin. Their cytoplasm showed abundant and prominent granular endoplasmic reticulum and, fre-

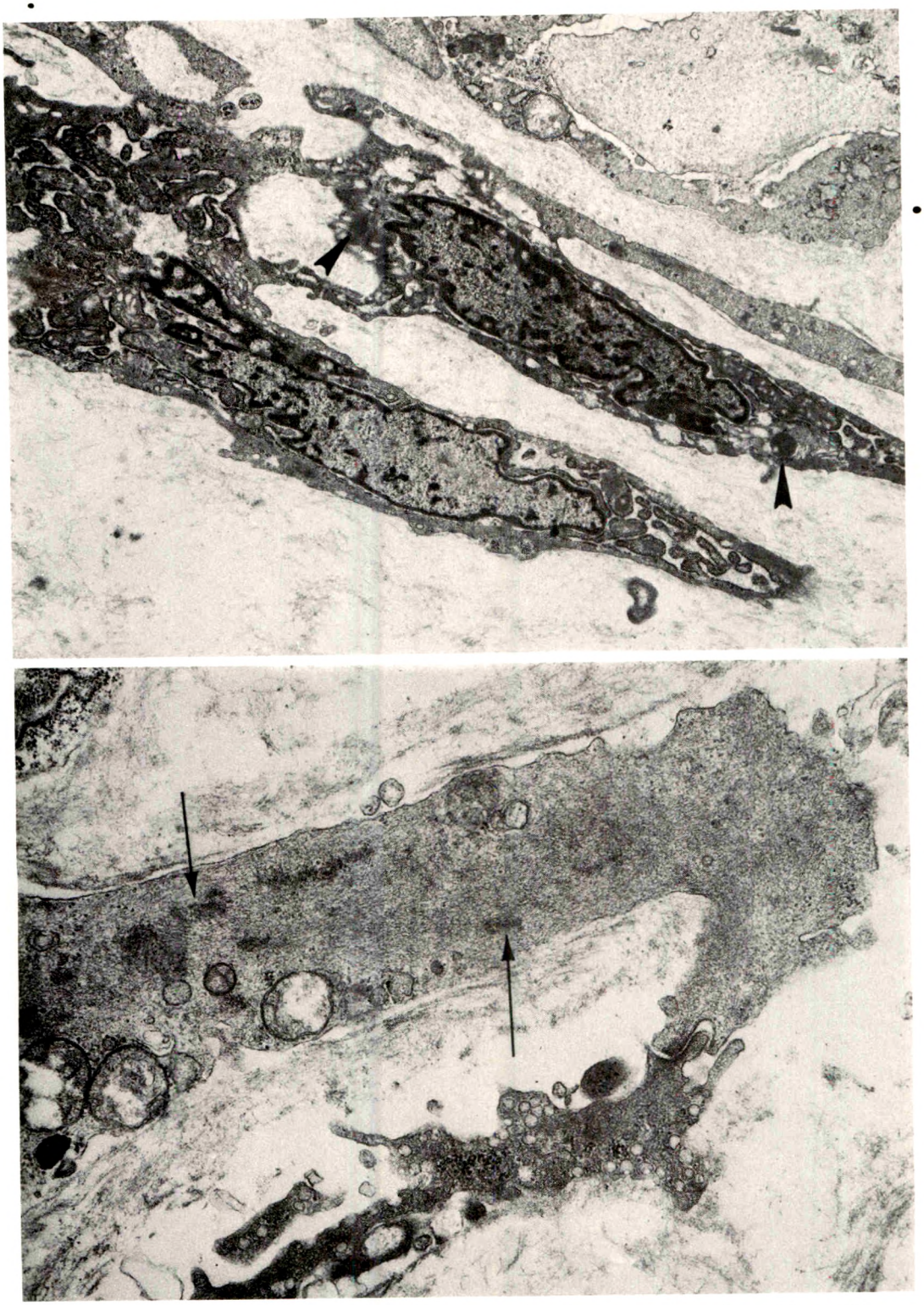


Fig. 4 (Kampik and associates). Top, The predominant cells in the epiretinal membrane. These cells were large, spindle-shaped, and fibrocyte-like with indented nuclei, prominent and dilated rough endoplasmic reticulum, and well-developed Golgi apparatuses. There are occasional electron-dense granules (arrowhead) (×5,950). Bottom, Higher magnification of similar fibrocyte-like cell shows cytoplasmic filaments 6 to 7 nm thick. There are areas of dense bodies (arrows) (×26,600).

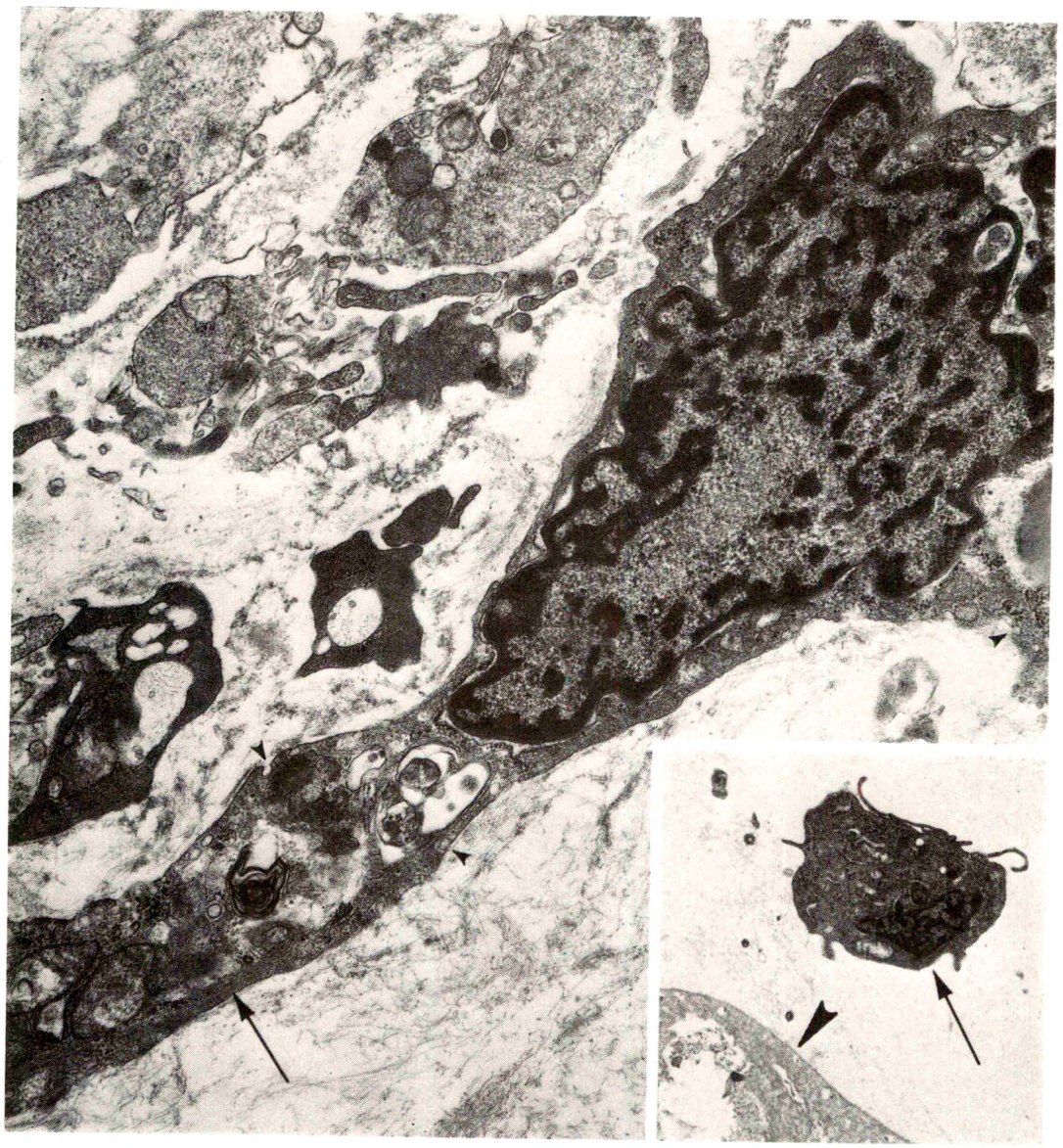


Fig. 5 (Kampik and associates). Plumper cell type (macrophage-like cell) with irregular nucleus and cytoplasmic filaments 6 to 7 nm thick (arrow) has numerous pinocytotic vesicles (arrowheads) and cytoplasmic granules containing complex lipid (×25,850). Inset, Macrophage-like cell (arrow) near internal limiting membrane (arrowhead) (×6,000).

quently, well-developed Golgi apparatuses and glycogen deposits. Occasionally we saw membrane-bound granules (lysosomes) of a different electron density. In several of those cells, intracytoplasmic filaments were 6 to 7 nm in diameter. In areas with those filaments we observed dense bodies, similar to those seen in fibroblasts in granulation tissue.[14] Some of the cells with these characteristics had occasional areas of a basal-lamina-like material along short distances of their cell borders.

The second cell type had nuclear and



Fig. 6 (Kampik and associates). Two adjoining cells, presumably glial, with multilaminated basement membrane and numerous cytoplasmic organelles. These cells surround what is probably a degenerated cell process with many membrane-bound granules (arrow) (×14,650).

cytoplasmic characteristics quite similar to those of the first type, but appeared plumper. These cells also contained intracytoplasmic filaments 6 to 7 nm thick. Additionally, they had many pinocytotic vesicles and occasional intracytoplasmic granules containing complex lipid (Fig. 5). These cells resembled macrophages. We saw them in the folds of the wrinkled internal limiting membrane and scattered throughout the epiretinal membrane.

In one area, a degenerated cell process containing many membrane-bound granules of various electron densities was surrounded by other cell processes that had many cytoplasmic organelles and a continuous basement membrane (Fig. 6). These latter cell processes seemed to belong to the third cell type seen in the

epiretinal membrane specimen in smaller numbers. This third type was characterized by large, regular, oval nuclei with a much finer distribution of chromatin and a nucleolus (Fig. 7). The cytoplasm showed filaments 8 to 10 nm thick, and had fewer cellular organelles than those of the other cell types in the membrane. This cell type and its processes showed a sometimes multilaminated basement membrane whose electron-dense fibrils were about 23 to 25 nm thick. We identified these cells as fibrous astrocytes.[15,16]

The abundant collagen component of the preretinal membrane consisted of densely packed fibrils with a diameter of 10 to 15 nm in most areas. Those fibrils had no discernible periodicity.[17] There were notably patchy and sometimes strand-like formations, in which the fibrils were arranged in a more nearly parallel fiber-like pattern (Fig. 8). In some areas the collagen fibrils blended with the inner aspect of the internal limiting membrane, whereas in other areas there was no close contact with the membrane. In addition to these fibrils, we found a more granular material, especially near the fibrocytes.

The third collagenous component was the basement membrane material, 23 to 25 nm thick, of the glial cells.

In the surgical specimen, the coiled fragment of the internal limiting membrane showed no breaks. Collagen with a cross-section diameter of 10 to 15 nm covered most of the inner (vitreous) surface of the internal limiting membrane.



Fig. 7 (Kampik and associates). Glial cell with large regular nucleus and nucleolus. Multilaminated basement membrane with fibrils 23 to 25 nm thick is present (×12,880). Inset, Cytoplasm contains many filaments 8 to 10 nm thick (×40,060).

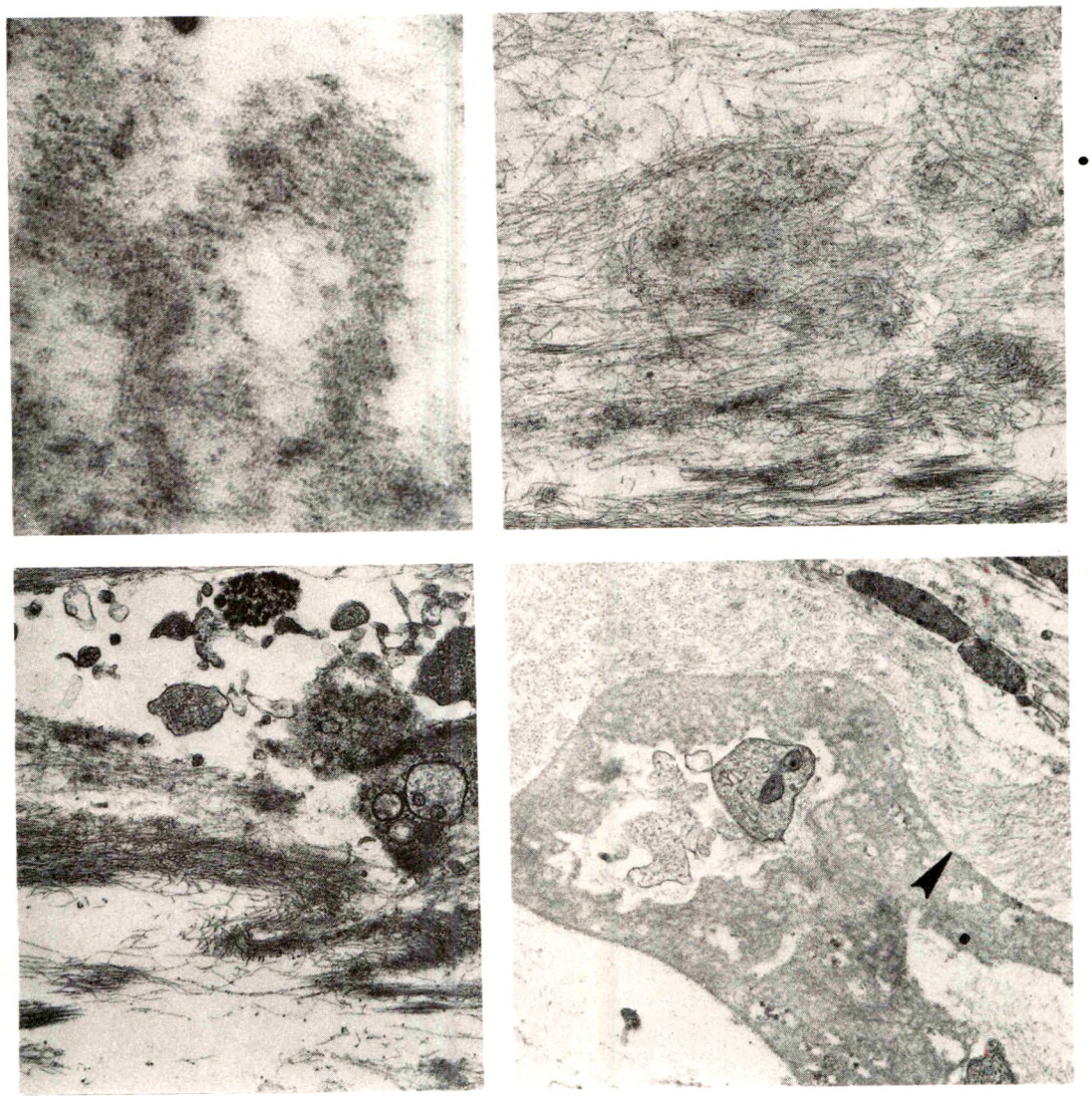


Fig. 8 (Kampik and associates). Top left, Granular collagenous material seen near fibrocyte-like cells (×79,280). Top right and bottom left, Predominant collagenous component (×23,330). The fibrils measure 10 to 15 nm in thickness and have no discernible periodicity. The fibrils are arranged in strands and clusters. Bottom right, Vitreous collagen fibrils blending with internal limiting membrane (arrowhead) (×10,540).

We found a cell process from a macrophage-like cell in direct contact with the internal limiting membrane in only one area (Fig. 9). We observed capillaries with intact endothelial cell junctions on the outer (retinal) side of the internal limiting membrane, partially associated with fibrous astrocytes. We did not see nerve fibers or other retinal elements in this area.

Discussion

The development of the epiretinal membrane in this case over six years showed progressive changes from a simple epiretinal membrane, with only mild



Fig. 9 (Kampik and associates). Cell process of macrophage-like cell in contact with vitreal surface of internal limiting membrane (arrow). On the outer (retinal) side, there is a capillary with intact endothelial cell junctions (×6,875).

distortion of the inner retinal surface and mild reduction in visual acuity, to a severe preretinal fibrous membrane with marked distortion of the retina and severe visual loss. The progressive changes documented in this patient suggest that the occurrence of idiopathic nonvascularized epiretinal membranes in young patients is an acquired disorder, rather than a congenital condition, as Wise[4] suggested. Also, this case demonstrated that some idiopathic membranes can be removed surgically, resulting in less distortion of the posterior retina and partial visual improvement.

Our specimen of epiretinal membrane

was fibrocellular tissue adhering to a large, continuous fragment of the internal limiting membrane of the retina and containing three cell types and three collagenous components. The clinical and histologic character of the specimen of thickened epiretinal membrane in this case distinguishes it from simple epiretinal membranes. Simple epiretinal membranes are thought to be formed exclusively by accessory glial cells that migrate from the superficial retina through breaks in the internal limiting membrane and proliferate to form delicate cellular membranes on the inner retinal surface.[1] Bellhorn and associates[11] found that more complex idiopathic nonvascularized membranes were composed predominantly of glial cells associated with occasional phagocytic cells laden with dense granules.

Secondary epiretinal membranes causing macular pucker, such as those following retinal detachment,[18] are composed of glial cells, fibrocytes, macrophages, and, less frequently, retinal pigment epithelial cells in varying combinations. Retinal pigment epithelial cells are more often seen in association with retinal detachment and massive periretinal proliferation.[19-21] Kenyon and Michels[15] and Rentsch[16] found that glial cells and macrophages were the main cellular constituents of epiretinal membranes associated with retinal vein occlusion. Constable[22] found fibrocytic cells interspersed among collagen in vitreous and epiretinal membranes secondary to vitreous hemorrhage and perforating injuries.

The morphologically differentiating features between the severe idiopathic epiretinal membrane described here and simpler idiopathic epiretinal membranes are, therefore, the presence and predominance of fibrocyte-like and macrophage-like cells in addition to glial cells. These same cellular elements are present in several types of secondary epiretinal membranes. Thus, the presence of these other cell types—such as fibrocytes, macrophages, or retinal pigment epithelium—appears to be essential for development of complicated epiretinal membranes. Progression of the epiretinal membrane in this case may have been caused by the fibrocyte-like and macrophage-like cells seen in the specimen, since these cell types are not present in simple epiretinal membranes. However, the origin of these additional cell types and their biologic activity are not certain.

Two different pathogenetic interpretations may account for the morphologic findings of differing cell types in this specimen. First, the plentiful fibrocyte-like or macrophage-like cells in this membrane may simply represent different stages of maturation and activity of proliferating glial cells with associated variation of their morphology.[23] If this is true, the formation of the macular pucker may be caused by retinal glial cells which proliferate on the vitreal surface of the retina because of an unknown stimulus after they have migrated through a break in the internal limiting membrane.[1] Formation of new collagen (fibrils 23 to 25 nm thick) may be ascribed to these glial cells.[15,16,24] However, this theory does not explain the origin of the predominant vitreous collagen fibrils (10 to 15 nm thick), arranged in strands and clusters, except perhaps by a condensing effect of glial cells on the vitreous. This kind of effect has not been observed in simple epiretinal membranes in which glial cells are the only cellular component.

A second explanation for the presence of fibrocyte-like and macrophage-like cells is that they are forms of cortical vitreous cells,[25] or hyalocytes. These cells have been described as a type of granular connective tissue cell with phagocytic properties.[26] Ultrastructurally, they have been characterized as macrophage-like cells[26] or fibrocyte-like

cells.[25] Hogan[27] distinguished these cells from macrophages by the scarcity of their lysosomes. Their special morphologic features include a lobulated, irregular nucleus with coarse heterochromatin, a well-developed Golgi apparatus, a varying number of cytoplasmic, electron-dense granules, and absence of polarity.[16,17,26] Thus, the fibrocyte-like and macrophage-like cells observed in this specimen closely resembled hyalocytes.

Further supporting this possibility was the presence of intracytoplasmic filaments 6 to 7 nm thick and probably contractile,[17] since Balazs and associates[28] described amoeboid movement and dynamic changes of the cell surface of hyalocytes. The origin of these cells is controversial. Whether hyalocytes are sessile vitreous cells[25,26] or hematogenous in origin,[29-31] the ability of these cells to proliferate within the vitreous cavity after photocoagulation of the retina is known from experimental animal studies.[32] Because of this intravitreal proliferation, Gloor[32] and Gärtner[33] postulated the participation of hyalocytes in the formation of epiretinal fibrocellular membranes, although they did not demonstrate this clinically.

If the fibrocyte-like and macrophage-like cells are forms of hyalocytes, the abundance of the vitreous collagen fibrils may be explained, because some workers believe that these fibrils are produced by cortical vitreous cells.[25]

The possibility that the fibrocytes and macrophages observed in our specimen were of retinal pigment epithelial origin is unlikely. We never observed pigmented cells in the membrane, and there was no retinal hole through which pigment epithelial cells could gain access to the vitreous cavity. Also, the morphologic features of retinal pigment epithelium were not present.

It seems most probable that the fibrocyte-like and macrophage-like cells in our specimen were hyalocytes. If this was the case, we suggest that these cells participated in the formation of this progressive idiopathic epiretinal membrane by migrating and proliferating within the vitreous cavity, producing collagen fibrils and changing their own shapes within the fibrous scaffold, leading to contraction of the epiretinal membrane. In this respect the hyalocytes would resemble fibroblasts in granulation tissue, in which fibroblasts have a contractile function.[14] The stimulus leading to the proliferation and activity of the cellular elements in this epiretinal membrane remains unknown.

Summary

Clinical and electron microscopic studies of an idiopathic epiretinal membrane that was nonvascularized and progressive showed three types of cells: (1) predominant, fibrocyte-like cells; (2) macrophage-like cells; and (3) glial cells. Most of the abundant collagen showed characteristics of normal vitreous fibrils, although thicker collagen fibrils were also present.

These ultrastructural findings, when compared with previously reported features of simple and secondary epiretinal membranes, suggest that clinically complicated epiretinal membranes are composed of more than one cell type, whereas simple epiretinal membranes consist only of glial cells. The fibrocyte-like and macrophage-like cells seen in our case were interpreted as being hyalocytes, rather than being derived from glial or retinal pigment epithelial cells. We support this interpretation by the ultrastructural features of the cells, the presence of abundant normal vitreous collagen, the biologic characteristics of hyalocytes as described by other investigators, and the absence of the ultrastructural features of glial or retinal pigment epithelial cells.

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TREATMENT OF INTRAOCULAR PROLIFERATION WITH INTRAVITREAL INJECTION OF TRIAMCINOLONE ACETONIDE

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Intraocular proliferation of cells deriving from retinal glia[1,2] and the retinal pigment epithelium,[3,4] in the case of retinal detachment, and from episcleral tissue and other sources,[5,6] in cases of injury, has been identified as the cause of membrane formation. Surgical removal of these membranes by vitrectomy techniques sometimes results in successful treatment of these conditions. Unfortunately, however, in many cases reproliferation of preretinal membranes turns an initial success into a failure.

It is, therefore, logical to look for a means of inhibiting cell proliferation after the initial removal of strands. Corticosteroids have a high level of antimitotic activity,[7] and an inhibitory effect on the growth of fibroblasts,[8-12] proven by the delayed healing of corneal wounds during topically applied corticosteroid therapy.[13]

We used intravitreal injections of dexamethasone alcohol to provide the high concentrations necessary to achieve inhibition of proliferation.[14] The results were promising, but dexamethasone alcohol disappeared from the vitreous cavity of the experiemntal eye seven to 14 days after injection and probably disappears faster from a human eye that has undergone vitrectomy.

Clinical experience has shown that retinas sometimes detach as much as six weeks after an initially successful reattachment. We believed that a longer-acting and more powerful corticosteroid might be more advantageous than the rather quickly absorbed dexamethasone alcohol. We selected triamcinolone acetonide and tested it in the same way we previously tested dexamethasone alcohol. We extended the observation time, however, to three months.

MATERIAL AND METHODS

Preparation of cultured fibroblasts—We used male and female pigmented rabbits weighing 2.5 to 3.5 kg. Each rabbit was anesthetized with a slow intravenous injection of sodium pentobarbital (20 to 30 mg/kg). We obtained tissue from rabbit rump skin after thorough shaving of hair and cleaning of the skin surface with 70% isopropyl alcohol. We excised a small piece of skin with a scalpel. We cut tissue into 1-mm cubes and placed them in a small amount of tissue culture medium (Dulbecco's Modified Eagle Medium [DMEM] with 25 mg of HEPES buffer supplemented with 30% fetal calf serum and antibiotics and antimycotics [50 units/ml of penicillin, 50 μg/ml of streptomycin, and 2.5 μg/ml of amphotericin B]). The culture was left in an atmosphere of 5% carbon dioxide in air at a temperature of 37°C for five days. The medium was then changed twice weekly (DMEM with 10% fetal calf serum, antibiotics, and antimycotics) until the culture became confluent. We subcultured the cells by washing them twice in phosphate-buffered saline without magnesium or calcium and then incubated them at 37°C for ten minutes with 0.25%

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trypsin and 0.02% EDTA. The dispersed cells were centrifuged (800 rpm for ten minutes), resuspended, diluted and recultured. At the time of autotransplantation, we prepared the cells as a suspension in 1 ml of phosphate-buffered saline by the same dispersion procedure. The cell content of a sample was counted and diluted to a concentration of 2.5×10^6 cells/ml. We agitated the suspension by pipetting just before transplantation.

Autotransplantation of cultured fibroblasts—We divided 44 pigmented rabbits into two groups, one treated with triamcinolone acetonide and the other a control group. We anesthetized the rabbits and dilated their pupils with 10% phenylephrine HCl and 2% cyclopentolate. Each eye was displaced forward with gentle pressure to the orbit. Using an indirect ophthalmoscope, we inserted a 27-gauge needle and injected 0.1 ml of fibroblast cell suspension (2.5×10^5 cells) into the middle of the vitreous cavity from a site 5 to 6 mm posterior to the corneoscleral limbus inferotemporally. We kept the bevel of the needle face up to avoid damaging the retina with the injection stream. We injected 22 of the animals with 0.1 ml of triamcinolone acetonide, suspended in physiologic saline with a surfactant (0.005% tyloxapol), which improved the suspension of the corticosteroid, from a site 5 to 6 mm posterior to the corneoscleral limbus superotemporally. Control eyes (22 animals) received 0.1 ml of physiologic saline with 0.005% tyloxapol. We used clean but nonsterile techniques for the injection procedures.

We had examined all the animals and taken fundus photographs preoperatively. After autotransplantation, we performed ophthalmoscopy and slit-lamp examinations on days 1, 3, 7, 14, and 28 and at three months. Fundus photographs were taken and fundus drawings made at each examination.

We killed the rabbits with an overdose of intravenously injected pentobarbital three months after autotransplantation. We enucleated the globes immediately and placed them in cold, phosphate-buffered 5% glutaraldehyde; we removed the corneal buttons to allow faster infiltration of the fixative. We left the eyes in fixative for at least 24 hours before evaluation. We then rinsed them in a phosphate-buffered solution and bisected them circumferentially 4.5 mm posterior to the corneoscleral limbus. We examined calottes under a dissecting microscope and photographed them.

The eyes were evaluated for the development of traction retinal detachments and retinal neovascularization.

To confirm neovascularization, we postfixed specimens from selected cases with 2% osmium tetraoxide in 0.1 M phosphate buffer, dehydrated them in alcohol, and embedded them in epoxy resin. We then prepared serial 1-μ sections and stained them with paragon stain.[15] We also cut 0.05-μ sections and stained them with uranyl acetate and lead citrate for light microscopy and electron microscopy, respectively. We analyzed the findings with chi-square and Fisher's tests. We excluded one eye in the control group from this series because it had mechanical retinal damage.

Results

Our results confirmed that 98% of the suspended cells were viable as shown by their ability to exclude trypan blue. Since we performed autotransplantation immediately after preparing the cell suspension, we expected cellular viability to be close to this value.

After withdrawing the needle from the eye, we observed a small amount of vitreous loss followed by a shift of cells or corticosteroid, or both, toward the entrance site. Slow injection and leaving the needle in place for a time after

injection minimized this loss. We did not observe any vitreous hemorrhage. Within two hours after injection, cells became clumped and appeared as well-defined cell clusters. Triamcinolone acetonide tended to spread diffusely in the middle of the vitreous cavity. Although the injections were done from separate sites, clouds of cells and corticosteroid were sometimes close to each other; they did not, however, merge (Fig. 1). Postoperative inflammation of the anterior segment was minimal. None of the eyes became infected.

One day after autotransplantation, debris was visible on the lower half of the fundus in some untreated eyes (18 of 43 eyes). Treated eyes had significantly less debris (one of 44 eyes). In both groups, strands became distinct in the vitreous. They usually bridged the area between the two entrance sites, or were directed toward the midvitreous or posterior lens surface, or both.

By three days after injection, injected cells developed into elongated strands.

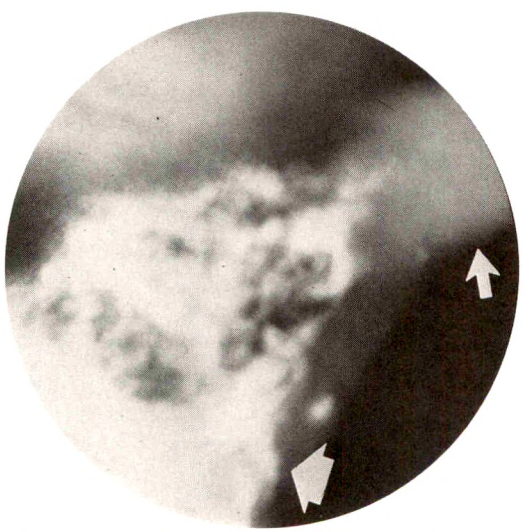


Fig. 1 (Tano, Chandler, and Machemer). Rabbit fundus after injection of fibroblast suspension (seen as gray cloud, thin arrow) and triamcinolone acetonide (white cloud, thick arrow).

When strands attached to the retina, the attachments were confined only to the area of the retinal vasculature. These strands anchored themselves onto the retinal vessels with surrounding capillary engorgement, followed by pucker formation. We observed traction retinal detachments in four of 43 control eyes (Fig. 2). The corticosteroid cloud was well preserved in the midvitreous cavity.

On the seventh day, more eyes showed attachment of strands to the medullary ray and subsequent traction on the medullary ray. Traction retinal detachments occurred in 22 of 43 control eyes, whereas only one of the 44 treated eyes showed detachments (Fig. 2). We observed prominent neovascularization growing from the attachment site into the strand in three control eyes. We found no neovascularization in the group treated with corticosteroid (Fig. 3). The corticosteroid mass was well preserved in almost all the eyes (41 of 44 eyes).

On the 14th day, vitreous strands were prominent and thick in most of the untreated eyes. Strands that had failed to anchor their free end to the medullary rays usually stopped growing or began to shrink. Traction retinal detachments occurred in 34 of 43 control eyes and in six of 44 treated eyes (Fig. 2). Many of the strands had neovascularization, appearing in a corona-like fashion or along and in the whole strand. Twenty-one of 43 control eyes showed clinically unequivocal neovascularization, whereas none of the treated eyes did (Fig. 3). The corticosteroid mass was visible in 39 of 44 treated eyes.

By day 28, the attached strands had increased in size, and pucker configurations became more prominent. Many of the localized retinal detachments enlarged, causing extensive retinal detachment. Most of the strands that had failed to reach the retinal vessels became thin in a quiet eye. There were traction retinal

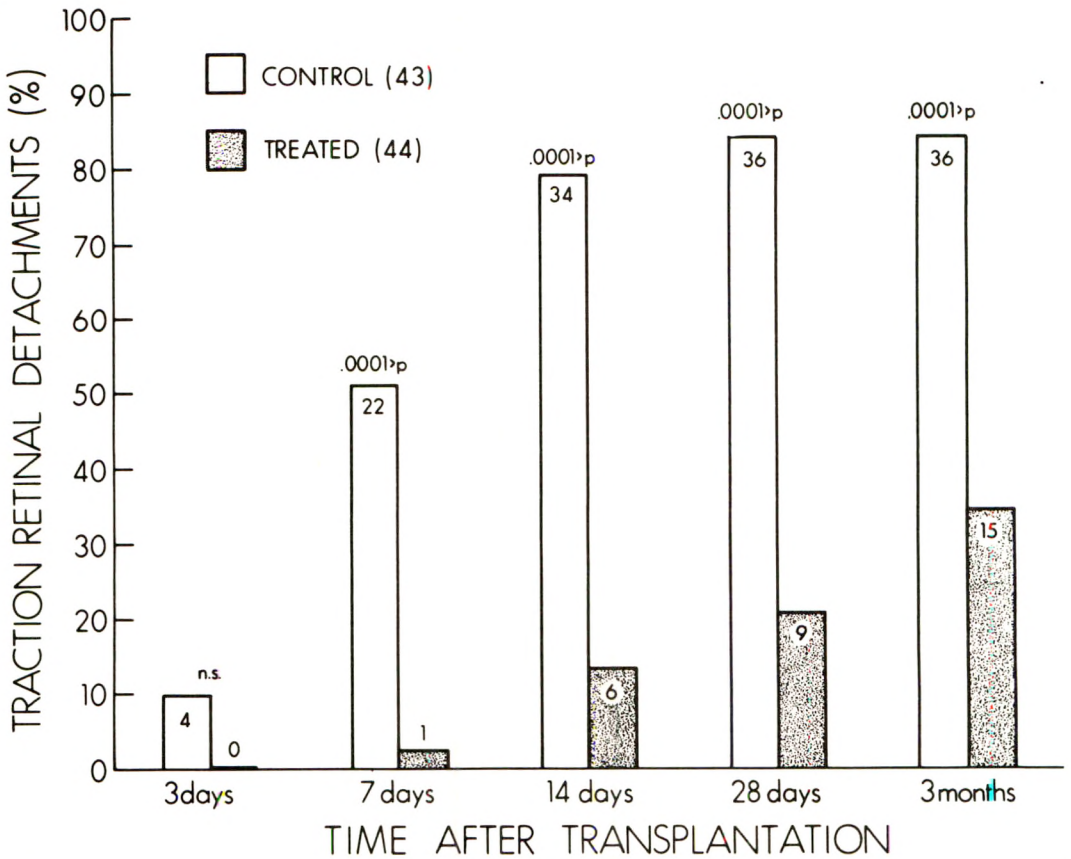


Fig. 2 (Tano, Chandler, and Machemer). Number of developing traction retinal detachments after intravitreal fibroblast injections with and without corticosteroid injection. The statistical significance of the differences is shown above the bars.

detachments in 36 of 43 control eyes (84%), but only nine of 44 (20%) treated eyes had traction retinal detachments (Fig. 2). In the control group, 25 eyes (58%) showed neovascularization; in the group treated with corticosteroid, there was no neovascularization (Fig. 3). Corticosteroid was still visible in about one half of the treated eyes (24 of 44 eyes).

Three months after autotransplantation, the eyes with traction retinal detachment showed pronounced scarring of tissue, that is, stiffer and more opaque fibrous tissue and higher traction retinal detachment. There were traction retinal detachments in 36 of 43 control eyes, whereas only 15 of 44 treated eyes had traction retinal detachments (Fig. 2). Although the number of neovascularized areas increased in the period from four weeks to three months, some neovascularized areas that had been unequivocal were covered with white fibrous tissue and seemed to show regression of vascularization. We verified the presence of the vessels in these eyes and in other eyes with possible neovascularization by examining thin sections of the fibrous strand (Fig. 4). In the control group, 31 eyes (72%) showed neovascularization, whereas in the treated group only eight eyes (18%) did (Fig. 3). By the time of enucleation, we could no longer identify a corticosteroid mass in any of the eyes.

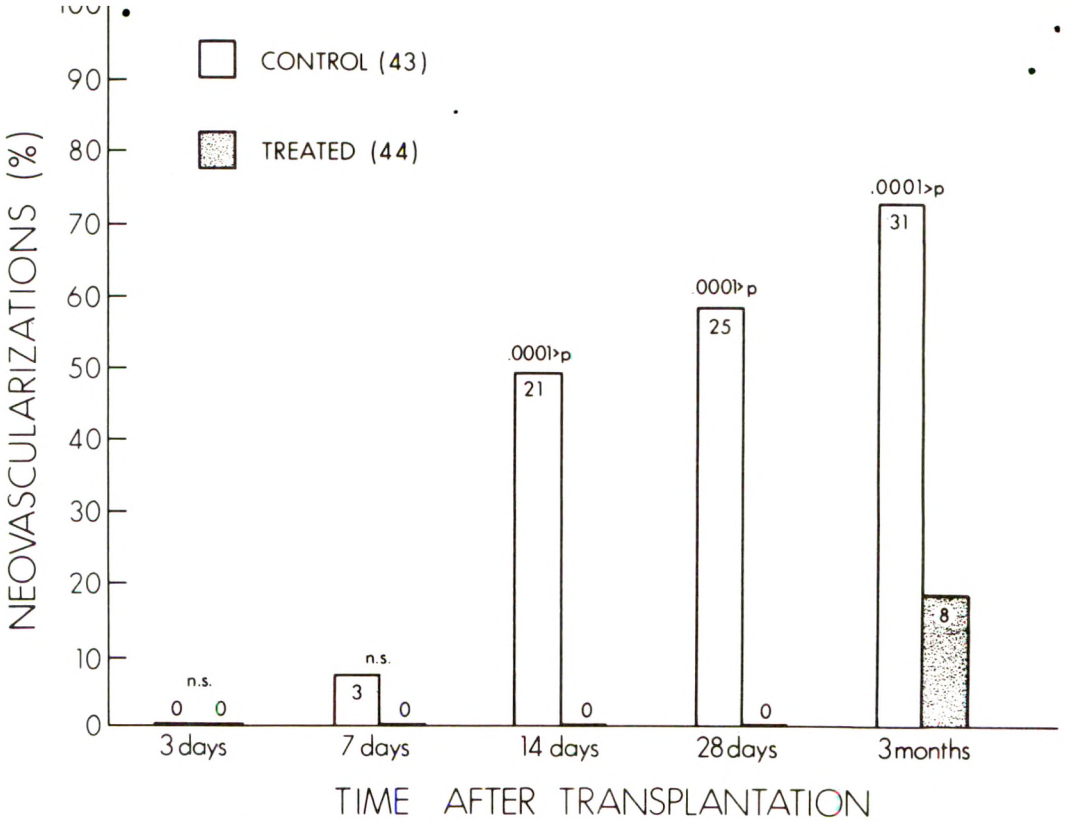


Fig. 3 (Tano, Chandler, and Machemer). Number of eyes in which retinal neovascularization occurred after intravitreal fibroblast injection with and without corticosteroid injection. The statistical significance of the differences is shown above the bars.

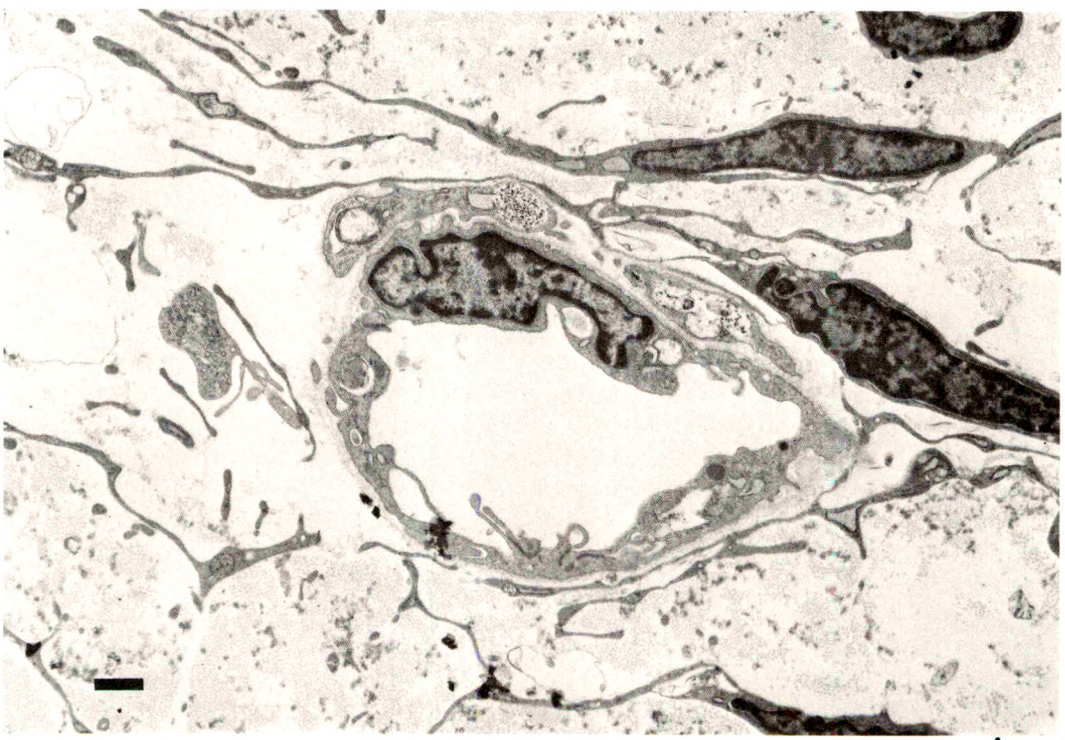


Fig. 4 (Tano, Chandler, and Machemer). Electron micrograph showing newly formed capillary with thin basal lamina surrounded by loosely packed fibroblasts and collagen (bar = 1 μ).

Discussion

In this experiment we were able to produce traction retinal detachments in 84% and neovascularization in 58% of the eyes four weeks after autotransplantation of fibroblasts. This is a much higher yield than in our previously reported experiment.[16] We attribute this increase to improved tissue culture and harvesting techniques with more viable cells being injected into the vitreous cavity than before.

Injection of triamcinolone acetonide reduced the percentage of traction retinal detachments on the 28th day from 84% to 20% (difference of proportion, $P_1 - P_2 = .633$) and that of neovascularization from 58% to 0 ($P_1 - P_2 = .581$).

These results prove that triamcinolone acetonide significantly inhibits the development of traction retinal detachment and neovascularization. If these differences of proportion are compared to those for dexamethasone alcohol,[14] the inhibition caused by triamcinolone acetonide is much greater than that caused by dexamethasone alcohol. Dexamethasone alcohol reduced the percentages of detachments from 57% to 24% ($P_1 - P_2 = .335$) ($\chi_1^2 = 5.59$; $P = .18$) and of neovascularization from 19% to 4% ($P_1 - P_2 = .148$) ($\chi_1^2 = 18.1$; $P < .00001$).

Triamcinolone acetonide inhibits fibroblast growth 21 times more than the same amount of dexamethasone alcohol and 325 times more than corticosterone.[8]

Triamcinolone acetonide remains in the eye longer than dexamethasone alcohol does because it is almost insoluable in water. We found visible traces of triamcinolone acetonide for more than 28 days, whereas dexamethasone alcohol disappeared from the vitreous cavity of rabbit eyes after 14 days.[14]

Most of the proliferation occurs near the seventh day after the injection of fibroblasts.[16] Conversely, persistent, low-grade proliferation can be observed many months after production of an experimental retinal detachment.[5] In order to evaluate the effect of corticosteroid on this extended low-grade proliferation, we expanded the experimental period in this study to three months. After this time the corticosteroid was no longer visible. The number of detachments had increased over those seen at 28 days (from eight to 15) and areas of neovascularization were visible more often (from zero to six). This indicates on-going proliferation, presumably after the cessation of corticosteroid activity. To reduce late proliferation further, therefore, a second corticosteroid injection, three to four weeks after the initial injection, may be needed. This may be particularly true in human eyes where vitrectomy and aphakia may cause early removal of the corticosteroid from the eye through the trabecular meshwork.

Neovascularization seems to be dependent on the development of strands when they touch the vascularized part of the retina. Neovascularization also decreases as fewer strands develop under the influence of triamcinolone acetonide. There seems to be more than a linear relationship between these two. No neovascularization developed despite traction detachments when triamcinolone was present. Another investigator was unable to demonstrate any deleterious side effect of triamcinolone acetonide (B. McCuen, unpublished data).

Summary

We studied the inhibitory effect of triamcinolone acetonide on experimental intraocular proliferation. Autotransplantation of fibroblasts from rabbit rump skin into the vitreous cavity resulted in intravitreal strand formation and traction retinal detachment in 36 of 43 eyes (84%) over a period of three months. A single intravitreal injection of 1 mg of triam-

cinolone acetonide inhibited fibroblast growth and significantly reduced the number of retinal detachments in 15 of 44 eyes (34%). Retinal neovascularization caused by fibrous strands coming into contact with vascularized retina was also reduced by triamcinolone acetonide (31 of 43 control eyes [72%] vs eight of 44 treated eyes [18%]). Intravitreal corticosteroid therapy may be an important adjunct to the therapy of perforating injuries and massive periretinal proliferation.

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THE USE OF RADIOACTIVE PHOSPHORUS (^{32}P) IN THE DIAGNOSIS OF OCULAR TUMORS

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In 1952 Thomas, Krohmer, and Storaasli[1] first reported the use of radioactive phosphorus (^{32}P) in the differential diagnosis of intraocular tumors. In late 1957 the clinical use of ^{32}P in suspected tumor cases was begun here. In February 1961 we reported a series of 125 cases.[2] In the ensuing 17 years we have studied 408 more cases which we report here.

The two criteria for a positive result that we used initially were those suggested by Eisenberg, Terner, and Leopold,[3] dependent on a one- and 24-hour count. First, the one-hour study had to show an increased uptake of 30% or more (lesion compared to control site), and, second, the 24-hour study had to yield a percentage uptake greater than the one-hour study.

We found that a 48-hour study yielded more significant information than the one- and 24-hour studies. In 1961[2] we published evidence to support our contention that the 48-hour data provided a single and more significant criterion for a positive result, that is, a percentage uptake of at least 65%. The validity of the 48-hour study has been substantiated by others[4-8] and is the basis for a positive result in the 408 cases reported here.

MATERIAL AND METHODS

The clinical investigation consists of a detailed history (with special reference to any previous, especially recent, ocular surgery that may lead to false-positive results), a medical examination, and a comprehensive ophthalmic examination, which, in recent years, includes ultrasonography and fluorescein angiography.

After the preliminary investigation, ^{32}P is administered intravenously as sterile isotonic sodium phosphate solution. The usual adult dose is 500 μCi of ^{32}P. (In treatment of polycythemia vera [erythremia] ^{32}P is administered in doses eight to 15 times the tracer dose used in the radioactive phosphorus [^{32}P] uptake test for ocular tumors. In more than 20 years of experience and over 500 cases we have not encountered a single case of leukemia to date.) Forty-eight hours after injection, counts of activity over the lesion and control sites are taken by using an eye probe, a small Geiger-Müller tube. The sensitive window portion of the probe is positioned on the sclera as close to the underlying uveal lesion as possible. To position the probe accurately, a ratemeter is used to determine the site of highest uptake. Scanning is especially important for the more posterior uveal lesions, which are less easily visualized than, for example, an iris lesion.

At the site of highest uptake a series of one-minute counts are taken with the probe kept stationary against the sclera, until consistent results are obtained. Similarly, a series of counts at a control site is recorded. The data obtained at 48 hours consist of C (counts per minute over the control site) and L (counts per minute over the lesion site), which are used to calculate the percentage increase in activity ($\triangle 48$) according to the following equation:

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$$\triangle 48 \text{ (in percent)} = \frac{\text{L-C}}{\text{C}} \times 100.$$

The criterion for a positive result is a percentage uptake of 65% or more at 48 hours after injection.

For anterior lesions (lesions of the iris, ciliary body, and anterior choroid) we use the external approach by placing the sensitive end of the probe in contact with the cornea or intact conjunctiva overlying the lesion after instilling an anesthetic topically. Uveal lesions posterior to the equator require a surgical approach in which conjunctiva is incised and Tenon's capsule buttonholed, permitting direct apposition of the probe face against sclera overlying the lesion. This is called the transconjunctival approach.The external approach for anterior uveal lesions includes the following: anesthetic administered topically, an outpatient procedure, and the use of a nonsurgical technique. In contrast, the transconjunctival approach for posterior uveal lesions involves an inpatient procedure, requires the use of an operating room and administration of a general anesthetic, and includes a surgical procedure. In the external approach the control site is a corresponding site in the fellow eye. In the transconjunctival approach, the control site is in the same eye, 180 degrees away from the lesion in an area free of tumor.

The dynamics for the ^{32}P uptake test depend upon the uptake by the ocular tissues of the ^{32}P and its relative concentration in the nuclear component of newly formed cells, paralleling its greater concentration in neoplastic vs normal ocular tissue.

The age and sex distribution of patients in our current series of 214 malignant tumors shows an average age of 60 years with a range of 12 to 83 years; 112 were males and 102 were females. The left eye was slightly more frequently involved, with 102 right eyes and 112 left eyes.

The location of 214 malignant tumors in our series showed a sparse incidence in the iris, only 2.8%. In the ciliary body a 12.6% incidence was noted as compared with the 10% noted by Shammas and Blodi.[9] Cases of anterior choroidal tumors in our series showed an incidence of 17.8%, compared with Shammas and Blodi's 28%.[9] The more posterior the area of uvea studied, the more the incidence of malignancy increased. Our cases of posterior segment lesions showed an incidence of 63.5% compared with Shammas and Blodi's series,[9] which showed a 59% incidence in cases of posterior segment lesions.

A breakdown of the 408 cases into five categories is shown in Table 1. Category 1, anterior segment cases, includes lesions of the iris, ciliary body, anterior choroid (up to the equator), and lesions of the conjunctiva and corneoscleral limbus as well, totalling 172 cases. Category 2, posterior segment, refers to posterior choroidal lesions (including macular and juxtapapillary cases). This was the largest category, totalling 209 cases. Category 3 comprises those 11 cases in which the lesion could not be visualized because of opaque media. Category 4, adnexal lesions, comprises nine cases of lesions of the eyelids, orbit, caruncle, plica, and fornix. Category 5 consists of seven excluded cases, and represents those studies that were incomplete because of failure of the patient to report for the completion of the test. An additional 12 cases are classified as equivocal (Table 1) because the patient did not consent to a transconjunctival approach for a posterior segment lesion, instrument breakdown occurred, or the like. The seven excluded cases and the 12 equivocal results make up 19 cases not included in the calculations of percentage error.

TABLE 1

SUMMARY OF RADIOACTIVE PHOSPHORUS ^{32}P UPTAKE TEST RESULTS IN 408 CASES

Category	No. of Cases	Test Results					
		Positive		Negative		Equivocal	Excluded Cases
		Total	False	Total	False		
1, Anterior segment	172	66	1	95	8	2	0
2, Posterior segment	209	132	1	63	4	9	0
3, Lesions not visualized	11	0	0	10	0	1	0
4, Adnexae	9	4	0	5	0	0	0
5, Excluded cases	7	0	0	0	0	0	7
Totals	408	202	2	173	12	12	7

RESULTS

In the remaining 389 cases, 204 positive results were obtained, including two false-positive results. Of 185 negative results, 12 were false-negative. The errors therefore totalled 14 of 389 cases, or 3.6% error.

A more detailed analysis of each category is shown in Table 2. For example, in Category 1, comprising the anterior segment cases, the one false-positive result and seven out of the eight false-negative results were all cases involving the ciliary body.

The ciliary body remains the most difficult area for ^{32}P uptake studies, with a marked tendency to yield false-negative results. Although our series did not show any errors in the 52 cases with iris lesions (Category 1A), the same features that tend to yield false-negative results in ciliary body cases were also apparent in these studies. The deeper, more centrally situated ciliary body lesions, characteristically not involving the pars plana, but lying deeper in the thicker pars corona, possibly contribute to the increased tendency to false-negative results. In the pars corona, between the probe and the more centrally located lesion, are the more peripheral structures of the ciliary body. Paraffin barrier absorption studies (Table 3 and Fig. 1) show decreased transmission of betas ^{32}P through the increased thickness of the paraffin barrier. Similarly, superficial uninvolved uveal tissue acts as an absorbing barrier to the betas emanating from the deeper-lying tumor, which leads to an increased tendency to false-negative results. Iris lesions that are closer to the pupillary border (Figs. 2 and 3) are located farther from the probe, and separated from the lesion by the depth of the anterior chamber as well as the corneal thickness. We

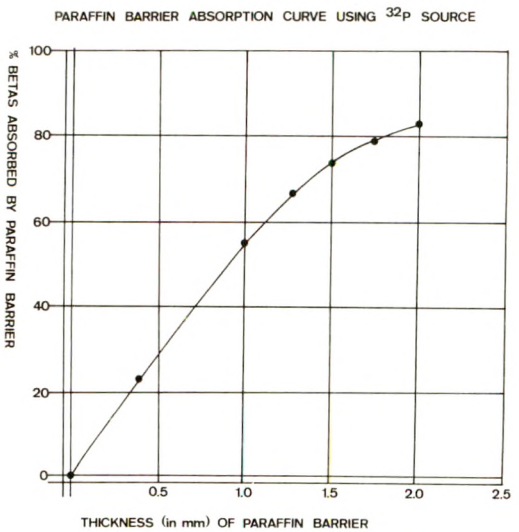


Fig. 1 (Goldberg, Kara, and Previte). Paraffin barrier absorption curve using ^{32}P source.

TABLE 2

TABULATION BY CATEGORY OF ^{32}P UPTAKE TEST RESULTS IN 408 CASES*

Category	No. of Cases	Positive				Negative				Equivocal	Excluded
		Total	U	C	False	Total	U	C	False		
1, Anterior segment											
A, Iris	52	6	0	6	0	46	37	9	0	0	0
B, Ciliary body	38	21	6	14	1	17	7	3	7	0	0
C, Anterior choroid	63	38	7	31	0	23	14	9	0	2	0
D, Bulbar conjunctiva	16	2	0	2	0	14	9	5	0	0	0
E, Palpebral conjunctiva	1	0	0	0	0	1	0	1	0	0	0
F, Corneoscleral limbus	2	0	0	0	0	2	0	1	1	0	0
Subtotal	172	67	13	53	1	103	67	28	8	2	0
2, Posterior segment											
A, Posterior choroid	188	126	13	112	1	54	24	28	2	8	0
B, Macula	18	5	1	4	0	12	9	2	1	1	0
C, Juxtapapillary	3	2	0	2	0	1	0	0	1	0	0
Subtotal	209	133	14	118	1	67	33	30	4	9	0
3, Lesions not visualized											
A, Opaque media	11	0	0	0	0	10	1	9	0	1	0
Subtotal	11	0	0	0	0	10	1	9	0	1	0
4, Adnexae											
A, Eyelids	2	1	0	1	0	1	1	0	0	0	0
B, Orbit	2	1	0	1	0	1	0	1	0	0	0
C, Caruncle	3	0	0	0	0	3	1	2	0	0	0
D, Plica	1	1	0	1	0	0	0	0	0	0	0
E, Fornix	1	1	0	1	0	0	0	0	0	0	0
Subtotal	9	4	0	4	0	5	2	3	0	0	0
5, Excluded cases											
A, Not done	7	0	0	0	0	0	0	0	0	0	7
Subtotal	7	0	0	0	0	0	0	0	0	0	7
Total	408	204	27	175	2	185	103	70	12	12	7

*U designates unconfirmed; C, confirmed.

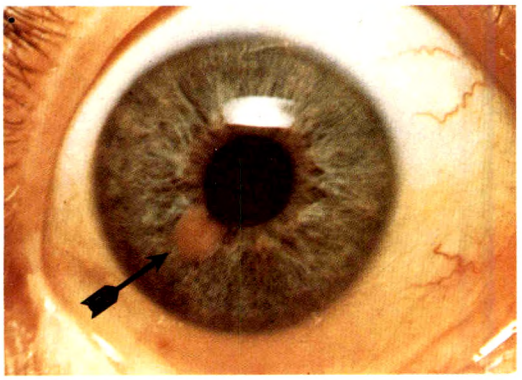


Fig. 2 (Goldberg, Kara, and Previte). External photograph showing tumor (arrow) of pupillary margin of iris.

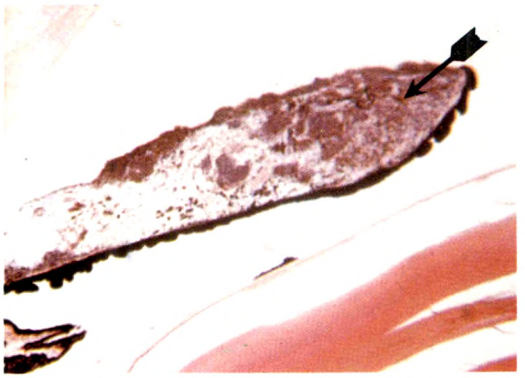


Fig. 3 (Goldberg, Kara, and Previte). Histologic section of pupillary border iris tumor (arrow) (hematoxylin and eosin, × 40).

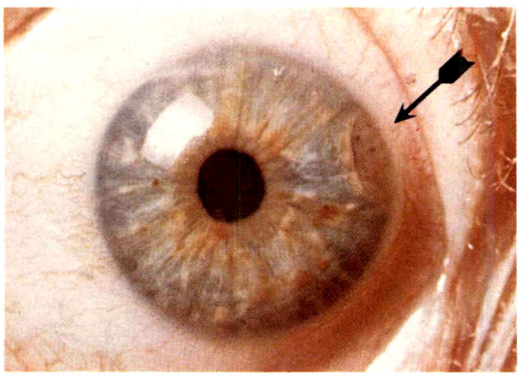


Fig. 4 (Goldberg, Kara, and Previte). External photograph showing tumor (arrow) of peripheral iris.

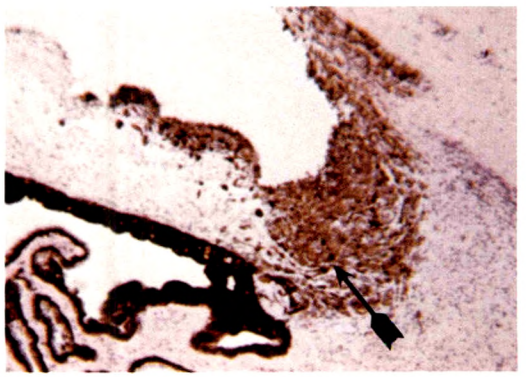


Fig. 5 (Goldberg, Kara, and Previte). Histologic section of iris tumor (arrow) invading angle and Schlemm's canal (hematoxylin and eosin, × 100).

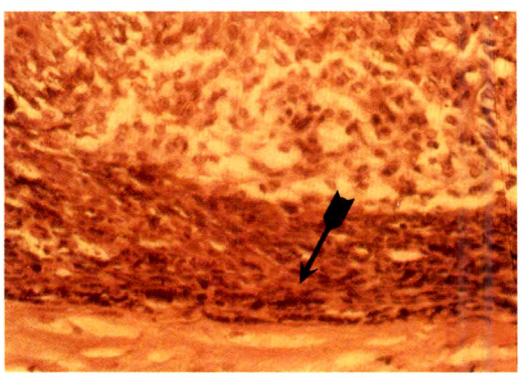


Fig. 6 (Goldberg, Kara, and Previte). Histologic section of choroidal tumor showing underlying layer of nevus cells (arrow) between malignant melanoma and sclera (hematoxylin and eosin, × 250).

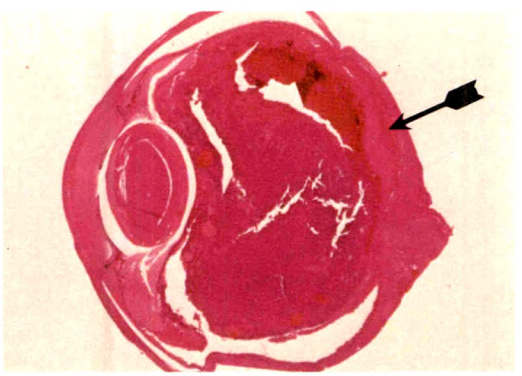


Fig. 7 (Goldberg, Kara, and Previte). Histologic section of enucleated globe showing hemorrhage (arrow) separating massive intraocular tumor from sclera (hematoxylin and eosin, × 5).

TABLE 3

TABULAR DATA OF PARAFFIN BARRIER ABSORPTION STUDIES USING ^{32}P SOURCE

Thickness of Paraffin Barrier (mm)	Counts Per Minute	Percent of Betas Transmitted	Percent of Betas Absorbed
0.00	381	100.0	00.0
0.40	295	77.0	23.0
1.00	170	44.6	55.4
1.25	128	33.6	66.4
1.50	98	25.7	74.3
1.75	80	21.0	79.0
2.00	69	18.1	81.9

usually dilate the pupil well in iris cases, bringing the lesion to a more peripheral location at which point the anterior chamber is shallower. In contrast, peripheral iris lesions (Figs. 4 and 5) are in closer proximity to the probe, with a lower incidence of false-negative results.

How does the ^{32}P uptake test compare with clinical judgment in typical iris and ciliary body cases (Table 4)? There were 26 cases of iris nevi and 18 cases clinically diagnosed (the clinical impression of the referring ophthalmologist who requested the ^{32}P study) as malignant melanoma of the iris referred for study. All 26 cases diagnosed as nevi were benign lesions by the ^{32}P uptake test, but of the 18 cases diagnosed clinically as malignant melanomas, only six were actually malignant melanomas; the remaining 12 appeared to be benign, with no contradictory data obtained by available follow-up. The clinical impression of iris malignancy was in error in two thirds of the cases referred for study. Of all cases with iris lesions, referred with a definite clinical diagnosis of nevus or malignant melanoma, 12 of 44 cases or 27% were in error.

A different situation exists in the studies of lesions of the ciliary body. Of 33 cases referred with a clinical diagnosis of malignant melanoma, 26 were malignant lesions (21 pathologically confirmed); the other seven were benign, with a clinical error rate of 21%. The ^{32}P uptake test did not improve the percentage of error in cases with ciliary body lesions. Of 38 ciliary body studies, there were eight errors, giving a test error rate of 21%, which is identical to the clinical error rate.

Of 42 cases with anterior choroidal lesions (Category 1C), referred clinically as malignant melanoma of the anterior choroid, nine appeared to be benign lesions, giving a 21% clinical error rate. The ^{32}P uptake test gave no errors in the anterior choroidal tumor studies, with 38 positive and 23 negative results (two equivocal cases not included).

In posterior segment studies, Category 2, the clinical error rate improved somewhat. Of 150 cases of posterior choroidal lesions clinically diagnosed as malignant melanoma, 24 were benign lesions, yielding a 16% clinical error rate. This may be contrasted with a 2.5% error rate obtained with the ^{32}P uptake test in cases with posterior segment lesions. These cases totalled 209, of which 200 were used in calculating the error rate; nine cases were equivocal. In these 200 cases there was one false-positive result and four false-negative results. The test error

TABLE 4

COMPARISON OF PERCENT ERROR IN CLINICAL DIAGNOSIS AND ^{32}P UPTAKE TEST RESULTS (INTRAOCULAR CASES)

Category	Clinical		^{32}P Uptake Test	
	No. of Cases	Error (%)	No. of Cases	Error (%)
1A, Iris	44	27.0	52	0.0
1B, Ciliary body	33	21.0	38	21.0
1C, Anterior choroid	42	21.0	61	0.0
2, Posterior segment	150	16.0	200	2.5

rate of 2.5%, compared favorably with the 16% clinical error rate noted previously.

There were 11 cases of Category 3, lesions not visualized because of opaque media. One equivocal case was excluded; the ten remaining cases all yielded negative results. Nine of the ten negative results have been confirmed, four pathologically and five clinically. Clinical confirmation of the benign nature of a lesion was considered to be a clinical course showing no suspicious changes in appearance and remaining free of local or metastatic growth for a period of more than five years. Although no hidden tumors were found in our present series, two unsuspected malignancies were found in Category 3 lesions studied in our 1961 series of 125 cases.[2]

The presence of an unsuspected tumor should be considered in a blind eye that shows either sudden onset of acute inflammation or an increase in intraocular pressure. The unexplained onset of uveitis or glaucoma in a blind eye with opaque media is a strong indication for a ^{32}P uptake test (as well as ultrasonography and other studies). Of 14 cases in the first series with opaque media referred for study, two cases had unsuspected tumors, an incidence of over 14%, which parallels other findings.[10]

The overall ^{32}P error rate was 3.6%, which included two false-positive results and 12 false-negative results or 14 errors in 389 cases (408 less 19 equivocal and excluded cases). Thus, if we exclude cases with ciliary body lesions, the error rate is 1.7% (six errors in 351 cases with non-ciliary-body lesions), which compares favorably with the 19% error rate noted clinically in our series. Zimmerman,[11] using Ferry's data,[12] shows the incidence of incorrect clinical interpretation leading to enucleation to be 19%, a figure identical to the clinical error made by our referral sources in diagnosing malignancy. The ^{32}P uptake test is a significant contribution to the detection of malignant ocular tumors.

DISCUSSION

Despite the overall low error rate in the ^{32}P uptake test, when the test is in error, the error is more likely to be a false-negative than it is to be a false-positive. In our combined series of 533 cases, there was a total of 16 errors of which four were false-positive and 12 were false-negative. Thus, there is a three-to-one predominance of false-negative results compared with false-positive results in our combined series. However, the present series of 408 cases shows that the ratio is even more striking

with a six-to-one predominance of false-negative results (12 false-negative results and two false-positive results).

Possible reasons for the false-negative results have been discussed previously and involve the following:

In the first group, the distance is greater between the probe face and the lesion in the ciliary body, which is characteristically less peripheral in location (those cases with ciliary body lesions: Cases 129, 283, and 403).

In a second group, the false-negative results may derive from an inactive or resting phase of tumor growth. A case that gives a negative ^{32}P result with low uptake may, in a repeat determination six months later, give a positive result with a higher ^{32}P uptake. We postulate a metabolically inactive phase in tumor growth to explain this phenomenon.

A third group of false-negative results, alluded to previously, gives a false-negative result because the tumor base apposed to the sclera contains benign nevus cells (Fig. 6), which are and remain metabolically inactive, but from which the malignant cells may have originated. Arnesen and Nornes[13] support the thesis that, as opposed to Reese's[14] original opinion, most malignant choroidal melanomas originate in a pre-existing benign nevus and that the tumors undergo a gradual change from a differentiated to a less differentiated type. The original nevus site is most apt to be in the scleral bed, which is that portion of the tumor counted by the probe.

A fourth group includes those false-negative results obtained in cases in which the tumor is shielded from the probe by intervening hemorrhage (Fig. 7), necrotic tissue (Fig. 8), or a serous fluid barrier as in retinal detachment.

A fifth miscellaneous group of false-negative results are caused by failure to scan thoroughly, thus missing a large



Fig. 8 (Goldberg, Kara, and Previte). Histologic section of globe showing tumor with necrotic portion (arrow), which could give a false-negative result (hematoxylin and eosin, ×5).

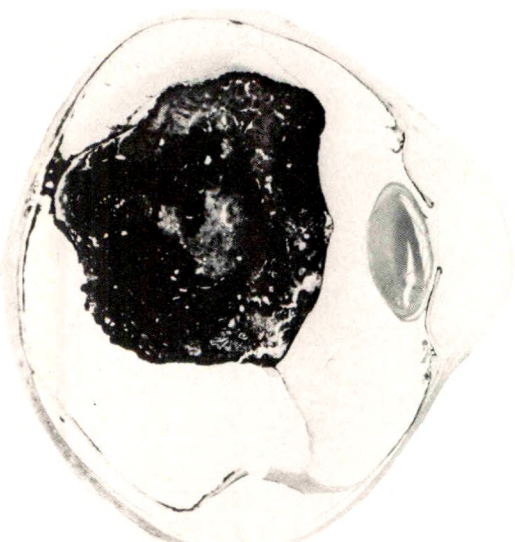


Fig. 9 (Goldberg, Kara, and Previte). Histologic section of globe showing large tumor with a small stalk that requires careful scanning (hematoxylin and eosin, ×5).

tumor suspended from a small base by a narrow stalk (Fig. 9). Similarly, a small tumor in any location requires careful scanning (Fig. 10). Cases with juxtapapillary tumors tend to give false-negative results because the optic nerve mechanically blocks the accurate apposition of the probe to the tumor site (Fig. 11) .

There were four false-negative results involving posterior segment tumors (Cases 192, 199, 233, and 381). In Case 381 the low count was attributed to destructive changes caused by radiation. The pathologic report noted hydropic changes secondary to radiation, leaving only a few viable tumor cells, thus the low count of 27%. The original lesion was metastatic from a kidney tumor, which is more amenable to radiation than malignant melanoma. In the other three posterior segment cases (Cases 192, 199, and 233) we are unable to account for the false-negative results. In each case the malignant melanoma proved to be of the spindle-cell type.

However, the false-positive results are either errors in technique, or faulty instrumentation; excluding these possibilities, false-positive results are found in inflammatory lesions that simulate neoplasm by laying down metabolically active cellular and nuclear components.[15,16] A consideration of the conditions necessary for increased uptake of ^{32}P at 48 hours is the need for a significant nuclear component in order to concentrate and retain the ^{32}P. This criterion can be satisfied by subacute or chronic inflammation, as in posttraumatic lesions or in chronic granulomata, as in Case 94 in our original series.[2]

False-positive results are less common. Shammas and Blodi[9] quoted Shields as having no false-positive results in almost 600 ^{32}P uptake tests. We have similarly found few false-positive results (two in 389 cases). For example, a false-positive result was obtained in Case 298 that showed active uveitis. In Case 517, the ^{32}P uptake test was significantly positive, 164%. The surgeon performed a cyclec-



Fig. 10 (Goldberg, Kara, and Previte). Histologic section of globe showing small tumor requiring careful scanning (hematoxylin and eosin, ×5).



Fig. 11 (Goldberg, Kara, and Previte). Histologic section showing juxtapapillary tumor. These cases tend to give false-negative results because optic nerve blocks probe apposition to the tumor (hematoxylin and eosin, ×5).

tomy; the specimen was reported pathologically as showing areas of vasodilatation. We believe that the excised tissue did not include the main lesion, which we believe to be malignant. Characteristically, the uvea adjacent to a melanoma shows vasodilatation and we believe that the surgeon removed a portion of such tissue adjacent to the main lesion, which remains unresected. If our hypothesis is correct, there should be a local recurrence, and this case is being followed up.

The results obtained in the 408 additional cases support our contention that the ^{32}P uptake test is useful in the diagnosis and management of ocular tumors. It is a nondestructive, at times noninvasive, test, easily performed by trained personnel, and it is inexpensive. It provides valuable information not available from fluorescein angiographic or ultrasonographic studies. It supplements ophthalmoscopy, trans- and retro-illumination, and other clinical techniques.

Some investigators have failed to consider inflammation secondary to recent surgery or trauma and primary inflammatory disease. These conditions, which may simulate malignancy, cause misinterpretation of raw ^{32}P uptake test results and lead to unjustified criticism of the ^{32}P uptake test.

Gass[17] noted that enucleations have been performed with some false-positive results in eyes devoid of malignant tumors. In our 533 cases, we have found false-positive results in only four cases. Enucleation was performed in two of these four cases, and in each of these two cases the decision was not based on ^{32}P findings alone. Gass[17] tends to reserve the ^{32}P uptake test primarily for those cases that we describe as eyes with opaque media (tabulated in Category 3, lesions not visualized). He presupposes greater accuracy of clinical judgment by the experienced observer not using the ^{32}P uptake test. Our studies do not support his hypothesis.

Summary

The radioactive phosphorus (^{32}P) uptake test used in ocular tumor evaluation in 408 cases achieved an overall percentage error rate of 3.6%. This compared favorably with the clinical error rate of 19.2% overall. The least accuracy was obtained in cases with ciliary body lesions.

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Ophthalmic miniature

The diplopia of the drunken man may help us towards a diagnosis in other cases, in the adult subject. In the tipsy diplopist nobody hesitates to say that the pathological mischief is in the brain. The liquor has got into the head; mediately or in propria persona.

That it is substantially present in the vessels of the brain is not improbable. In the case of a gentleman, who voluntarily encountered death with an over-dose of aqua vitae. I smelt the brandy in the brain itself.

Robert Hull, Cursory Notes on *The Morbid Eye*,
London, Longman, Orme, Brown, Green and Longman, 1840

ULTRASTRUCTURE OF TRACTION RETINAL DETACHMENT IN RHESUS MONKEY EYES AFTER A POSTERIOR PENETRATING OCULAR INJURY

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Traction retinal detachment secondary to penetrating trauma or as a complication of rhegmatogenous retinal detachment is the major cause of failure in vitreous and retinal reattachment surgery. Studies in experimental animal models and on specimens from human eyes undergoing vitreous surgery have helped delineate the pathogenetic mechanisms in vitreoretinal traction. These observations have emphasized the role of intraocular cellular proliferation in forming transvitreal and periretinal membranes.[1-4]

We have developed a reproducible model of traction retinal detachment secondary to a posterior penetrating eye injury in the rhesus monkey.[5,6] This experimental model is characterized histologically by intravitreal cellular proliferation with the formation of cyclitic, transvitreal, epiretinal, and retroretinal membranes.[6] By electron microscopy, we found proliferating cells with the characteristics of both fibroblasts and smooth muscle (myofibroblasts). Myofibroblasts are the contractile cells found in healing wounds and are considered responsible for wound contraction.[7] We propose that traction retinal detachment in our rhesus monkey model is produced by cellular contraction in a process analogous to the wound healing response and to wound contraction occurring elsewhere in the body.

In this article, we describe the electron microscopic findings that define the cellular response in the intraocular tissues of the monkey eyes. Additionally, we report the findings of autoradiographic studies designed to clarify the origin of proliferating cells within these eyes.

MATERIAL AND METHODS

As previously described,[5] we subjected rhesus monkey eyes to a penetrating wound, using a standard incision 8 mm long and 3.5 mm posterior and parallel to the corneoscleral limbus through the pars plana. We excised prolapsed vitreous and carefully closed the wound with interrupted sutures of 8.0 silk, using microsurgical techniques. After wound closure, we performed indirect ophthalmoscopy and examined the area of the wound and peripheral retina by scleral indentation. We injected autologous blood (0.5 ml) through a 25-gauge needle which we inserted through the wound under ophthalmoscopic control into the midvitreous.

We enucleated a series of 20 monkey eyes at intervals ranging from two days to four months.

For electron microscopic evaluation, we injected 0.2 ml of Karnovsky's fixative (glutaraldehyde 2.5% and paraformaldehyde 2% in 0.1 M cacodylate buffer, pH 7.2) through the pars plana into the vitreous cavity immediately before enucleation. We then enucleated the eye, excised a central corneal button, and immersed the eye in this fixative for 24 hours at 4°C. We opened some eyes

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circumferentially at the equator and sectioned others sagittally through the optic nerve and midpoint of the wound. We photographed areas of interest. Small blocks of tissue were excised and post-fixed in 2% phosphate-buffered osmium tetroxide, dehydrated in a graded series of alcohols, and embedded in epoxy resin. We stained 1-μ sections with toluidine blue and examined them by light microscopy. We further trimmed selected blocks, then sectioned and stained them with uranyl acetate and lead citrate, and examined them with an electron microscope.

For radioautography studies, we injected 0.1 mCi of tritiated thymidine in saline intravitreally two hours before enucleation. The eyes were fixed in 4% paraformaldehyde. We prepared autoradiographs by standard dipping techniques as outlined by Rogers,[8] using Kodak NTB-2 emulsion diluted 1:1 with distilled water on both 5-μ paraffin and 1-μ epoxy resin sections. We routinely exposed the autoradiographs in light-tight boxes for 15 to 18 days at 4°C. Slides were developed in Kodak D-19 at 18°C, fixed, and dried. We stained the paraffin and epoxy resin autoradiographs with toluidine blue, and examined them by both light- and dark-field microscopy.

Results

The vitreous—Along the inner aspect of the wound, in the tissue ingrowing from the wound and within the vitreous, we encountered typical fibroblasts which were stellate or spindle-shaped with long cytoplasmic processes. The cell nuclei were oval and elongated and contained one or more prominent nucleoli. The cytoplasm contained large mitochondria with irregular cristae and extensively developed and dilated rough endoplasmic reticulum and a prominent Golgi apparatus. These cells were loosely packed and embedded in large amounts of extracellular matrix composed of masses of collagen fibrils. These fibroblasts were present by one week after injury and could still be identified after four months.

Within the vitreous, we found other stellate or spindle-shaped cells with features of fibroblasts and smooth muscle cells. We identified these cells as myofibroblasts. They had irregularly shaped, convoluted nuclei and cytoplasmic filaments arranged in thin bundles of parallel filaments 6 to 8 nm in diameter, typical of contractile proteins in smooth muscle cells. The filaments were usually interspersed with small electron-dense bodies and were located just beneath the cell membrane (Fig. 1). A few of these filaments extended for the entire length of the cell and occasionally short bundles of filaments appeared to extend through the cell membrane at the long, narrow end of a cytoplasmic process. The cytoplasm of these cells was also packed with dilated cisternae of rough endoplasmic reticulum. Intercellular connections of the desmosome type were present, and we occasionally found a basal lamina (Fig. 1). Myofibroblasts were most commonly present at from two to six weeks after injury and were most often embedded in an extracellular matrix of collagen-like fibrils.

Proliferation of the pigmented and nonpigmented ciliary epithelium also occurred at the site of the penetrating wound and at the ora serrata. As early as four days after injury, a mound of proliferating nonpigmented ciliary epithelial cells was present at the ora serrata. These cells incorporated radioactive thymidine. Most of these proliferating cells had rounded nuclei and abundant clear cytoplasm, but there were often several layers of pigment-containing cells at the mound. By electron microscopy, the proliferating, nonpigmented ciliary epithelial cells were large and of varying size

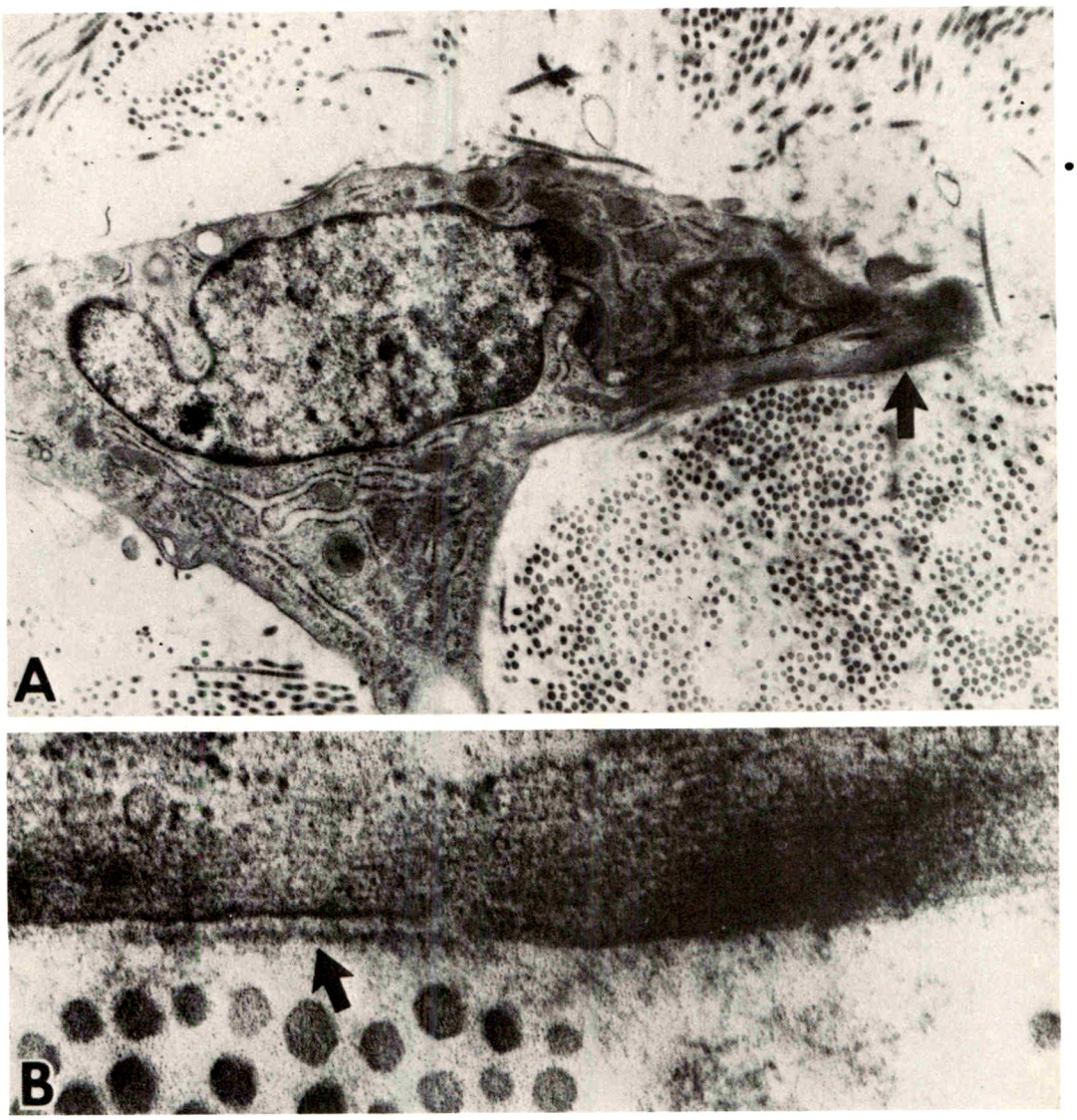


Fig. 1 (Cleary, Minckler, and Ryan). Top, Intravitreal fibrous tissue adjacent to wound from monkey eye at six weeks after injury. A stellate cell embedded in collagen fibrils (63 nm diameter and 48 nm periodicity) has prominent rough endoplasmic reticulum, a convoluted nucleus with deep traverse folds, and peripheral bundles of cytoplasmic filaments (arrow) (× 13,230). Bottom, Enlargement of the area of cytoplasmic filaments. Basal lamina-like material is also present (arrow) (× 76,500).

and shape. Their electron-dense cytoplasm was packed with fine filaments and contained smooth endoplasmic reticulum and occasional pigment granules. Some of these cells formed clusters with a rosette-like configuration of densely packed cells with interdigitation of fine cell processes in the center and a smooth surface between apposing cells joined by zonulae adherens. The proliferating ciliary epithelial cells seemed to have migrated into the vitreous cavity, contributing to the fibrous ingrowth from the wound (Fig. 2). They also extended along the surface of the detached posterior hyaloid.

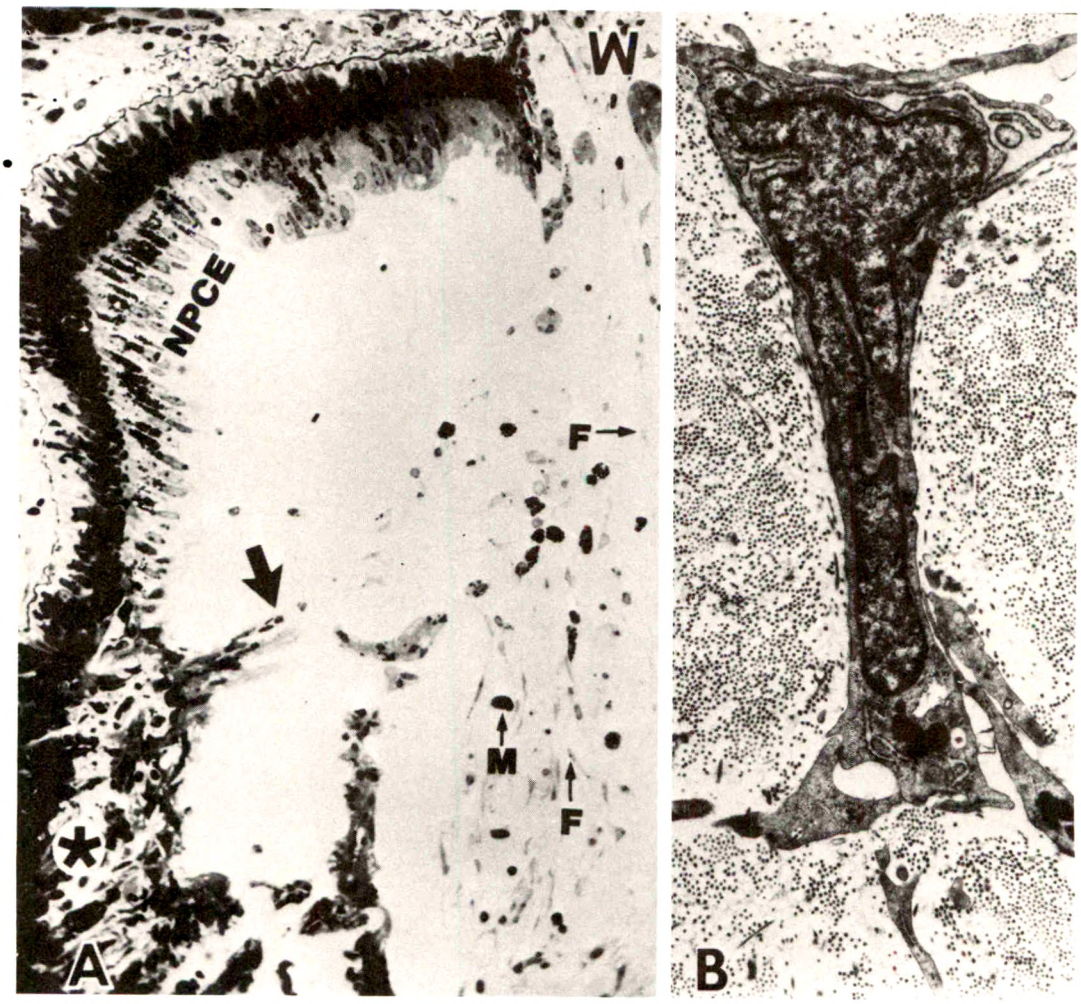


Fig. 2 (Cleary, Minckler, and Ryan). Left, Wound (W) and adjacent pars plana in a rhesus monkey eye enucleated seven weeks after injury. The nonpigmented ciliary epithelial cells (NPCE) appear to have proliferated and to blend (arrow) with the fibroblast-like cells (F) which are in continuity with the wound. Macrophages (M) containing pigment granules are scattered throughout the intravitreal fibrous tissue. Many of the nonpigmented ciliary epithelial cells (NPCE) normally contain pigment. There may be proliferation of the pigmented ciliary epithelial cells at the ora serrata (*) (toluidine blue, ×201). Right, An individual cell from the intravitreal fibrous tissue from adjacent section. The nucleus is convoluted, rough endoplasmic reticulum is prominent, and pigment granules are probably present in the cytoplasm, suggesting origin from the ciliary epithelium (×8,650).

Within the vitreous, we also found cells which contained non-membrane-bound, round pigment granules in their cytoplasm (Fig. 2). These pigment-containing cells appeared to be derived from proliferation of pigmented ciliary epithelium, both at the edge of the penetrating wound and at the ora serrata (Fig. 2). Some of these cells were spindle-shaped and had elongated nuclei, whereas others were irregularly shaped and had indented nuclei. These cells had long, thin cytoplasmic processes and contained well-developed, rough endoplasmic reticulum. They also had the characteristic bundles of intracytoplasmic filaments and

the intercellular junctions seen in myofibroblasts.

In the intravitreal fibrous tissue from six to eight weeks after the injury and thereafter, we found mostly irregularly shaped spindle or stellate cells with long cytoplasmic processes and folded or indented nuclei. A few of these cells with the characteristics of myofibroblasts contained parallel filaments, 6 to 8 nm in diameter, whereas others contained irregularly tangled filaments 12 nm in diameter. In general, intracytoplasmic filaments were difficult to demonstrate in these intravitreal cells. The intercellular spaces contained prominent bundles of collagen-like fibrils.

In the rhesus monkey model, detachment of the posterior vitreous occurs typically about two weeks after injury.[5] Specimens from the detached posterior hyaloid contained vitreous collagen-like fibrils 13 nm in diameter without discernible periodicity. Cellular debris was also present and, in some specimens, there were spindle-shaped cells with the characteristics of fibroblasts. We also found cells with features of nonpigmented ciliary epithelium. In light microscopic sections, continuity with the proliferating nonpigmented ciliary epithelium cells at the ora serrata could be identified. Additionally, in one eye the posterior hyaloid contained a discrete wrinkled membrane, which by electron microscopy had a smooth surface on its vitreous aspect and an irregular contour on its retinal aspect (Fig. 3). We identified this wrinkled membrane as a segment of the inner limiting membrane of the retina, stripped from the surface of the retina during detachment of the posterior hyaloid.

We found several different types of collagen fibrils by electron microscopy in the vitreous of the rhesus monkey eyes. Most common were fine unbanded filaments about 13 nm in diameter, which were similar to native vitreous collagen. Also present were fibrils with diameters of 20 to 25 nm and a periodicity of 15 to 20 nm. Occasionally, we found fibrils with diameters of 60 to 64 nm and a periodicity of 48 to 64 nm. These different collagen fibrils were present not only in the vitreous cavity at the inner aspect of the wound, but elsewhere in the vitreous and in the epiretinal membranes.

The retina—Over the peripheral retina at about six to eight weeks after injury, we found epiretinal membranes which were a few cell layers thick. These membranes contained fibroblastic-type cells with long cytoplasmic processes. The cell nuclei were oval and the cytoplasm contained dilated rough endoplasmic reticulum. In these thin epiretinal membranes, some of the spindle-shaped cells had ultrastructural characteristics of myofibroblasts, with convoluted nuclei and bundles of intracytoplasmic filaments about 6 to 8 nm in diameter. In some of these cells, the microfilaments lay directly at the cell membrane and appeared to send extensions into the extracellular space at the edge of a cytoplasmic process.

Thick epiretinal membranes had formed over the peripheral and posterior retina in the rhesus monkey eyes at about three months after injury. They were associated with full-thickness retinal folds in detached retina. These thick epiretinal membranes contained some spindle-shaped fibroblastic-type cells, but the predominant cell type had a convoluted and indented nucleus characteristic of a myofibroblast (Fig. 4). The cytoplasm contained rough endoplasmic reticulum, but only a few of these cells had bundles of intracytoplasmic filaments.

Epiretinal membranes over both the peripheral and posterior retina contained another cell type with features suggesting derivation from retinal glia. In these cells, the cytoplasm contained little besides

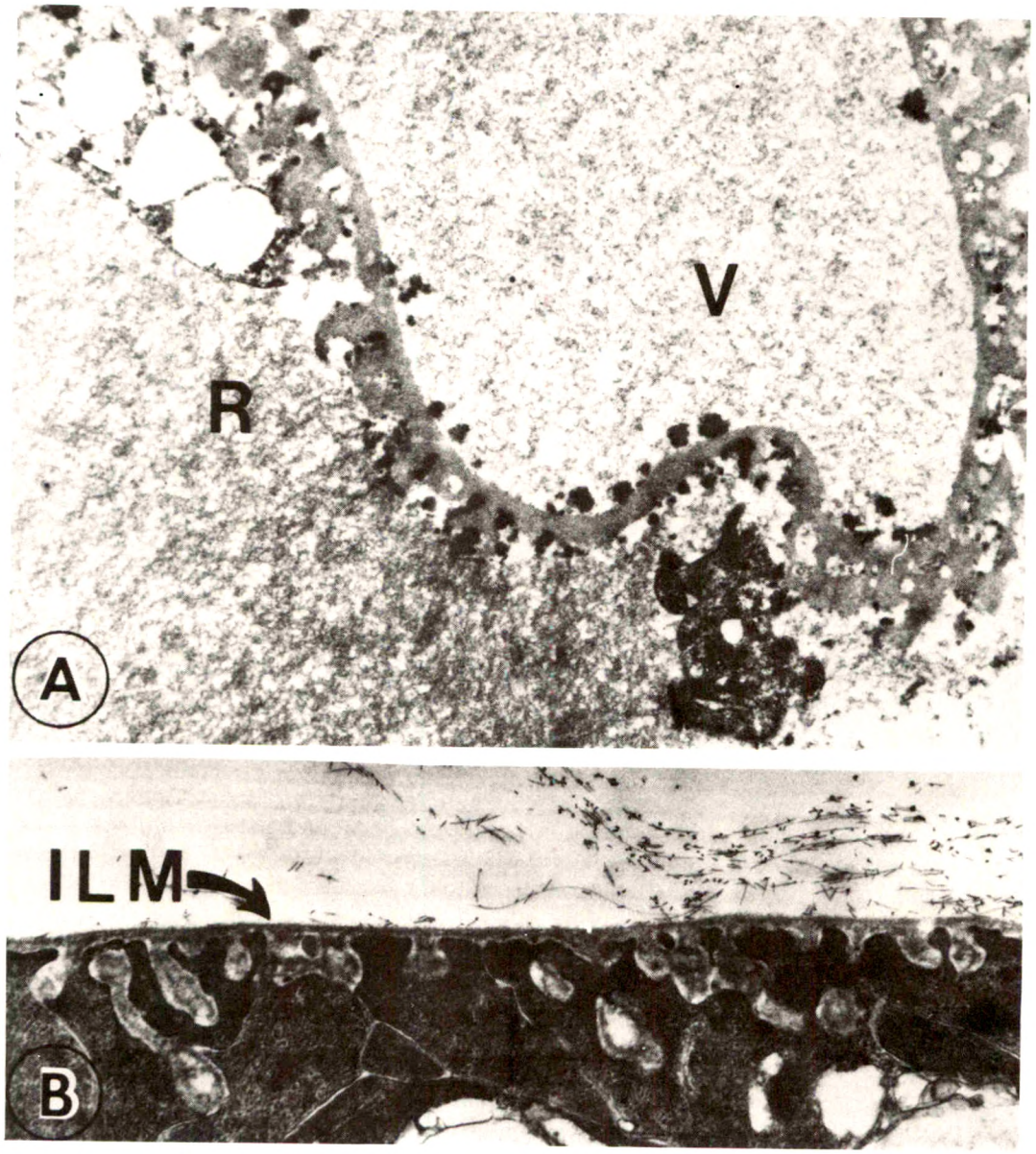


Fig. 3 (Cleary, Minckler, and Ryan). Top, Vitreous from a rhesus monkey eye obtained 14 days after injury includes the posterior edge of the detached vitreous. The membrane-like structure probably represents a detached segment of the internal limiting membrane and is smooth on the vitreous side (V) and irregular in contour on the retinal side (R). Debris, possibly cell fragments, is present on the retinal side. The dense granular material along the membrane is probably iron, freed from degenerating erythrocytes (× 15,000). Bottom, The internal limiting membrane (ILM) and adjacent retina from the periphery of a normal control eye. The internal limiting membrane is smooth on the vitreous side and irregular on the retinal side where it fills gaps between Müller cell processes (× 9,900).



Fig. 4 (Cleary, Minckler, and Ryan). Epiretinal membrane and underlying retina from a rhesus monkey eye ten weeks after injury. The nuclei of the cells forming the epiretinal membrane are markedly irregular in orientation with prominent infolding of the nuclear membranes. Collagen-containing material (C) is interspersed between cells. There is prominent folding of the internal limiting membrane (ILM) and iron-laden macrophages (MAC) lie just beneath the internal limiting membrane. The inner retina consists only of glial cell processes (G) in this area (× 2,016).

loosely packed filaments and microtubules oriented longitudinally (Fig. 5). Cell processes were densely packed and cell nuclei were very few. Electron microscopic examination showed a definite break in the inner limiting membrane of the retina through which glial cell processes extended and formed bridges connecting the retina to the overlying epiretinal membrane (Fig. 5). These glial cell processes were joined to each other and to adjacent intraretinal glial cells by desmosomes (Fig. 6).

Macrophages were present in the epiretinal membranes, along the inner limiting membrane of the retina, and within the inner retina. We found deposits of electron-dense material within these macrophages identical in location to iron-positive material in light microscopic sections. This same electron-dense material became aggregated along the vitreal aspect of the inner limiting membrane and could also be found within the inner nerve fiber layer. Macrophages were also aggregated along the vitreal aspect of the inner limiting membrane and occasionally appeared to be in transit through the inner limiting membrane into the retina (Fig. 7). We did not find residual breaks in the inner limiting membrane which could be attributed to the passage of macrophages, but in many specimens in which macrophages were present in the inner nerve fiber layer we noted complex infoldings of the overlying inner limiting membrane and also vesicles containing basal lamina-like material (Fig. 8).

Retroretinal membranes were less common and were not seen until about ten weeks after injury. Retroretinal membranes were connected to the outer retina, apparently fused to the outer nuclear layer through defects in the plane of the external limiting membrane (Fig. 9). In the retroretinal membranes, we found cells probably of glial origin with abundant cytoplasm, containing loosely packed filaments oriented longitudinally (Fig. 9). Cell processes were densely packed, and desmosomes and zonulae adherens existed between cells. Also present in these retroretinal membranes were cells with the typical characteristics of retinal pigment epithelial cells. These cells had fine apical villi and elliptical cytoplasmic pigment granules and were connected to each other by junctional complexes (Fig. 9).

Discussion

The precise pathogenetic mechanisms responsible for traction retinal detachment are poorly understood. Changes in collagen linkage in the vitreous or in newly formed fibrous tissue in epiretinal membranes may produce shrinkage which results in traction on the retina.[9-11] On the other hand, cellular contraction in transvitreal or periretinal membranes is more likely to be the critical factor producing vitreoretinal traction and traction retinal detachment.[1-4] In our experimental animal model, the pathophysiology of traction retinal detachment shared many features with the normal processes of wound healing and, in particular, with the mechanisms responsible for wound contraction.

The wound healing response consists of an inflammatory reaction followed by a cellular phase with fibroplasia and the production of collagen. These processes lead to wound contraction and scar formation. Wound healing may be controlled by chemical factors, but is facilitated by the presence of a fibrin matrix that provides orientation for the invasion of fibroblasts that lay down collagen.[12] Collagen synthesis or strong covalent bonding between collagen fibers is not required for wound contraction.[13,14]

New interest in wound contraction was created by the discovery of previously unrecognized multipurpose cells that combine the electron microscopic features of fibroblasts and smooth mus-

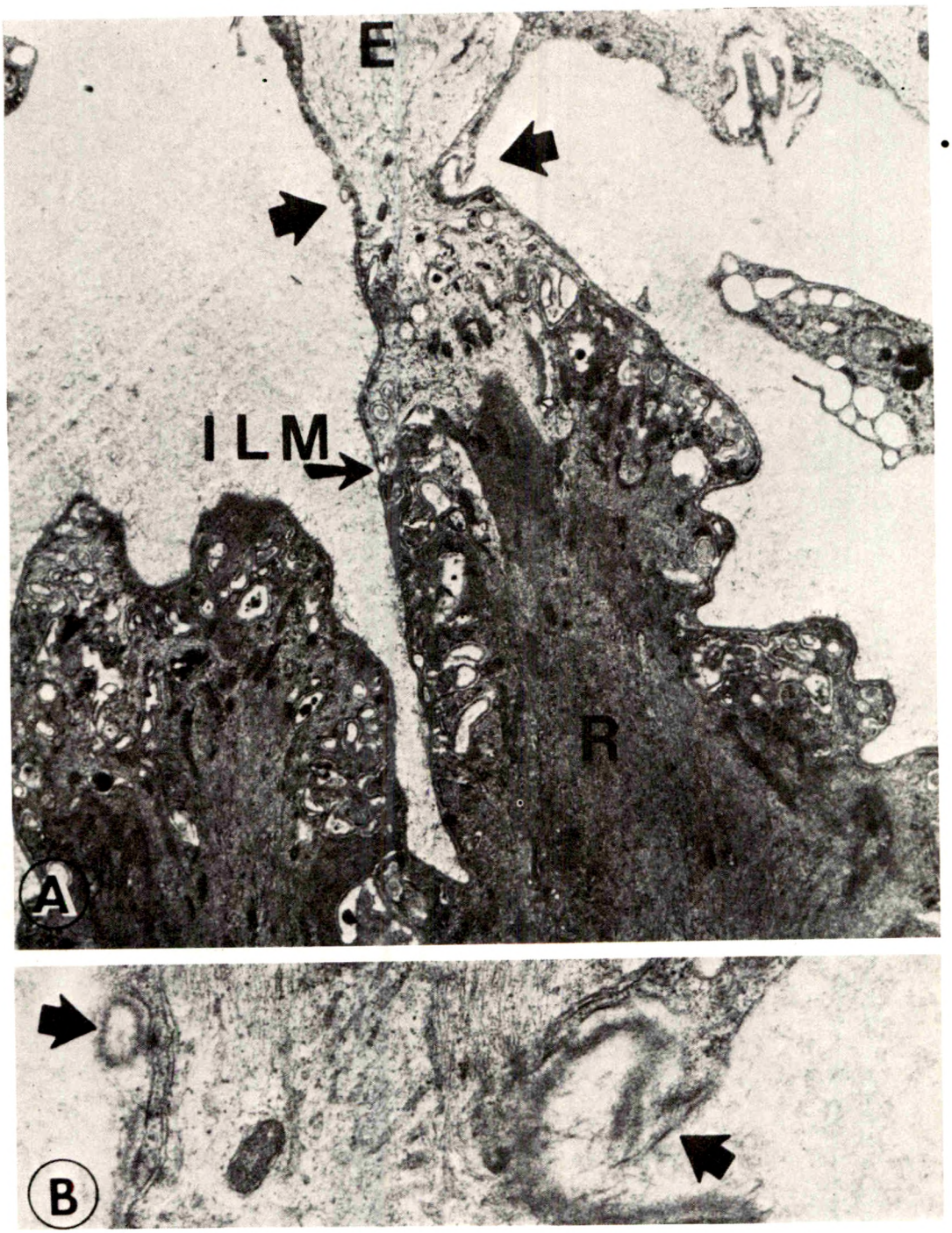


Fig. 5 (Cleary, Minckler, and Ryan). Top, Rhesus monkey eye ten weeks after injury shows an area of bridging between the retina (R) below and an epiretinal membrane (E) above. Continuity between the retina and epiretinal membrane occurs through a gap in the internal limiting membrane (ILM), which is apparent along the tissue bridge (large arrows) (× 6,300). Bottom, Higher magnification of the area along the tissue bridge, demonstrating a break in the internal limiting membrane with apparent recoil of its edges on either side (arrows) (× 30,600).

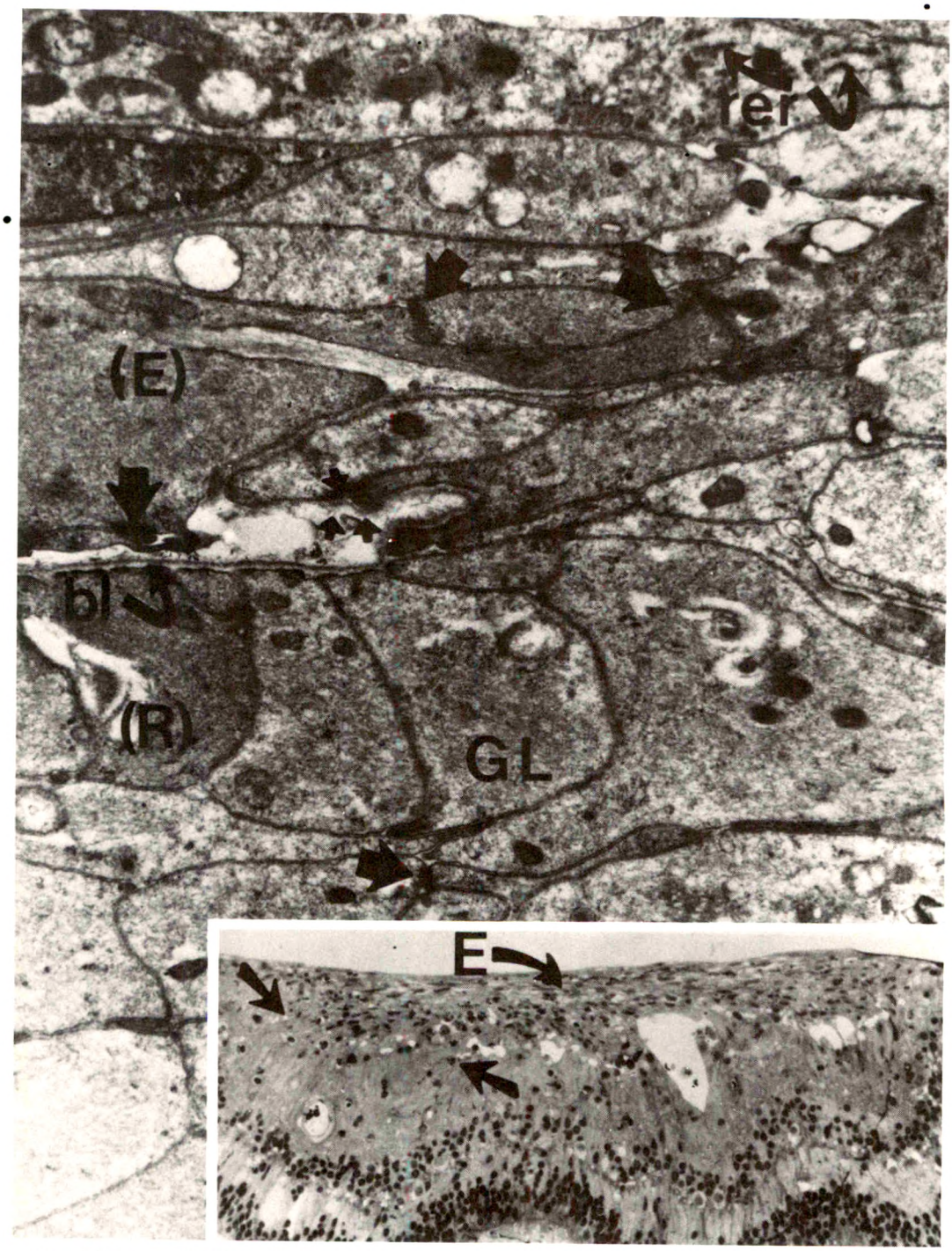


Fig. 6 (Cleary, Minckler, and Ryan). Rhesus monkey eye 12 weeks after injury shows one edge (small arrows) of a relatively large defect in the internal limiting membrane where the basal lamina (bl) is recoiled. There is continuity between glial cells (GL) of the retina (R) below and similar cells in the epiretinal membrane (E) above. Desmosomes (large arrows) are noted both between the glial cells in the retina and between cells in the epiretinal membrane. Epiretinal membrane cells adjacent to the internal limiting membrane strongly resemble glia while more superficial epiretinal membrane cells appear fibroblastic with spindle-shaped nuclei and prominent dilated rough endoplasmic reticulum (rer) (× 15,000). Inset, A 1-μ section of the same area of the retina and epiretinal membrane (E). The epiretinal membrane is continuous with the retina through a large gap in the internal limiting membrane corresponding to the area between the two arrows on the left (toluidine blue, × 160).

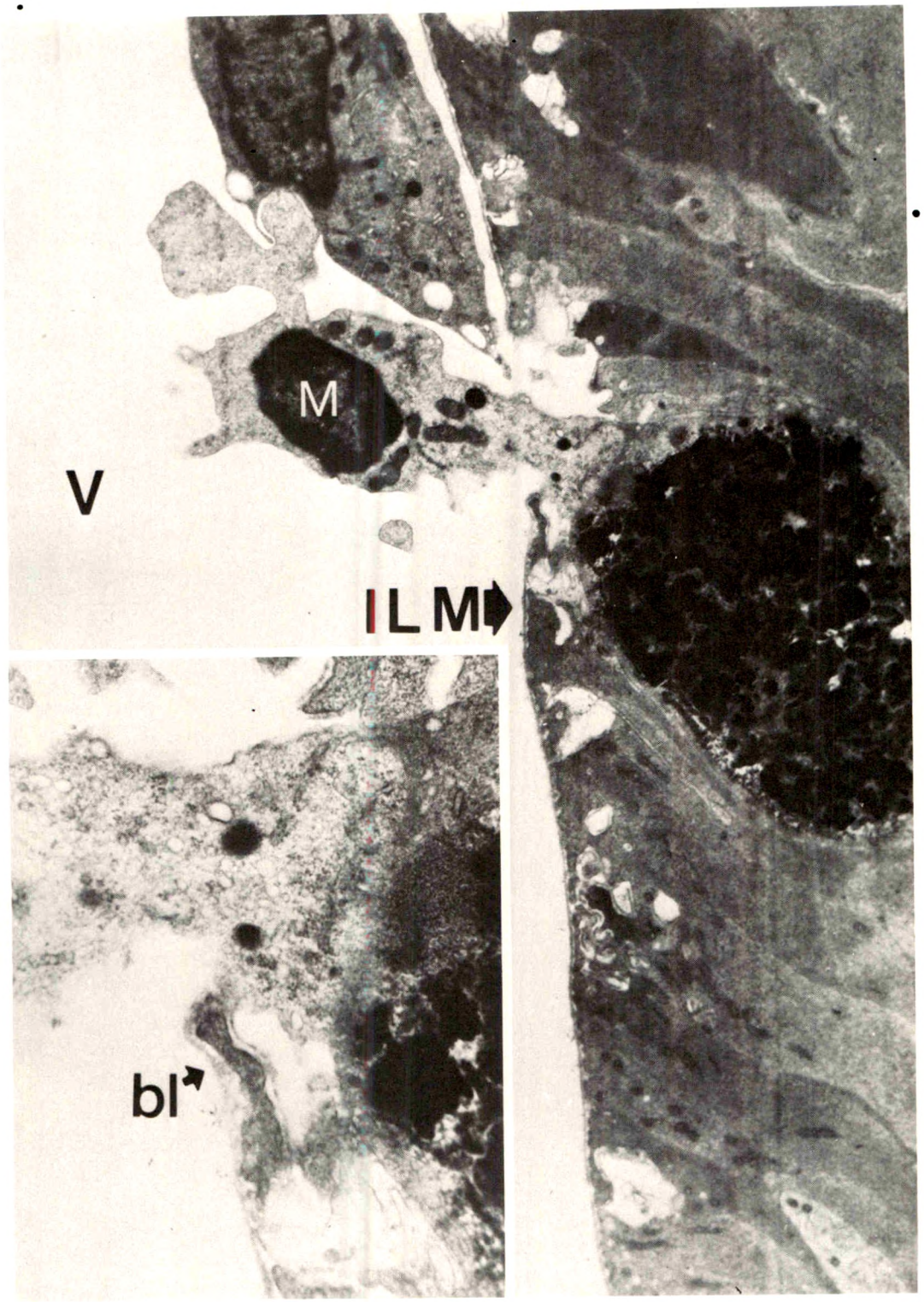


Fig. 7 (Cleary, Minckler, and Ryan). Rhesus monkey eye six weeks after injury. A macrophage (M), probably iron-laden, appears to be crossing a gap in the internal limiting membrane (ILM). The cell nucleus is still in the vitreous cavity (V) and the cell was probably in the process of entering the retina (× 8,800). Inset, Higher magnification of one edge of the gap in the internal limiting membrane shows that the basal lamina (bl) appears to be coiled back from the cell membrane of the macrophage (× 24,000).

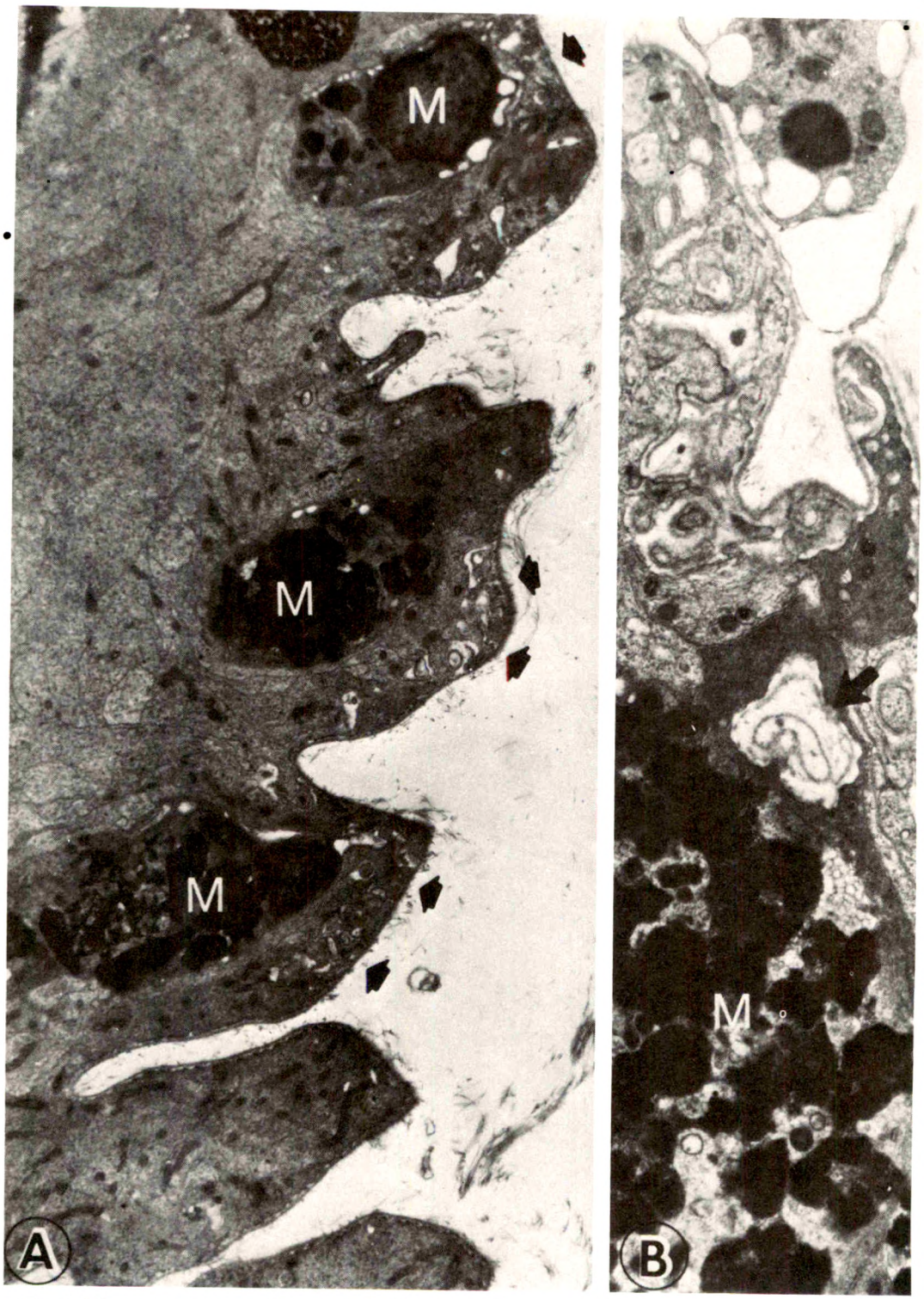


Fig. 8 (Cleary, Minckler, and Ryan). Left, Rhesus monkey eye enucleated eight weeks after injury. The inner retinal surface is wrinkled and three macrophages (M) are present in adjacent folds. Complex infolding and vesicle formation is apparent just under the internal limiting membrane overlying each macrophage (arrows), and is not apparent where retinal folds do not contain macrophages (× 6,300). Right, Another area of the same eye, showing basal lamina-like material (arrow) in vesicles or possibly complex infoldings of the internal limiting membrane. One area (arrow) probably demonstrates basal lamina-like material within the cytoplasm of a glial cell process or in the cytoplasm of an adjacent macrophage (M) (× 19,100).

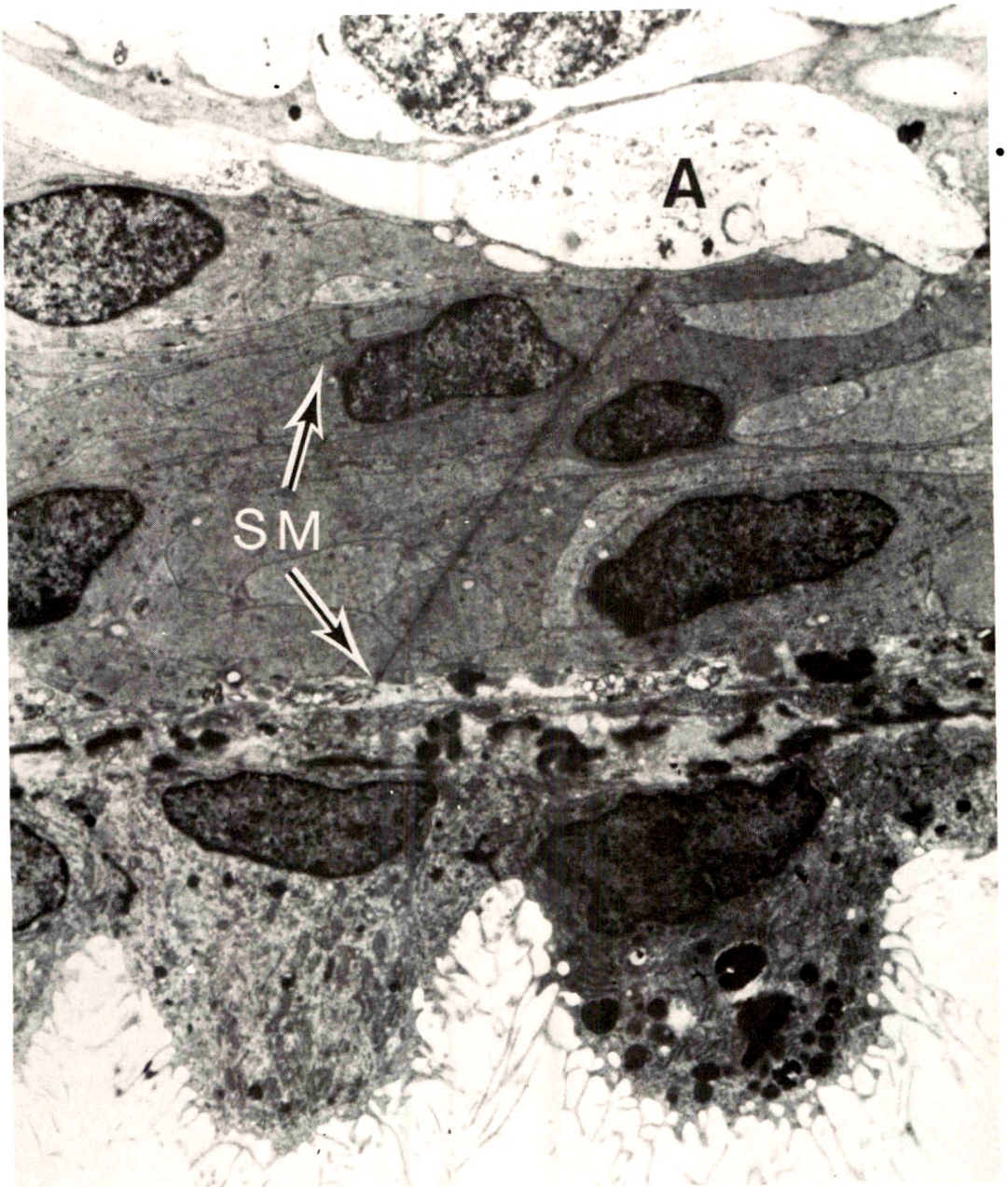


Fig. 9 (Cleary, Minckler, and Ryan). Rhesus monkey eye 12 weeks after injury shows a retroretinal membrane (SM) lined by cells resembling retinal pigment epithelial cells with prominent apical villi and melanin-like pigment granules. These cells presumably migrated into this position from adjacent retinal pigment epithelial cells. Cells to which the retinal pigment epithelial-like cells attach appear glial with electron-dense cytoplasm relatively devoid of organelles. Degenerating axons (A) are present above.

cle cells, the so-called myofibroblasts.[7] These cells contain intracytoplasmic filaments composed of actinomyosin, which provides a mechanism for cell contraction.[15] Myofibroblasts are considered responsible for the contraction of healing wounds.[7] They have also been found in pathologic tissues in a variety of human fibrocontractive disorders.[16-19]

In this study, we identified typical myofibroblasts in the intravitreal fibrous tissue and in epiretinal membranes. We also found cells that had the characteristics of myofibroblasts, but contained pigment granules in their cytoplasms. These pigment-containing cells were probably derived from the pigmented ciliary epithelium. Cells with precisely similar morphologic characteristics and thought to be derived from the retinal pigment epithelium have been identified in the vitreous and epiretinal membranes in monkey eyes with experimental retinal detachment[1] and in human eyes with massive periretinal proliferation.[4] The presence of intracytoplasmic filaments in retinal pigment epithelial cells is well known.[20] In both monkey and human eyes, retinal pigment epithelial cells may undergo metaplasia into fibroblast-like cells.[1,4]

In this study, the precise origin of the myofibroblast-type cells was often difficult to determine, and it is interesting that recent studies indicate more plasticity among differentiating cells than previously realized.[21] Wiggert and associates[22] demonstrated in tissue culture that chick retinal pigment epithelial cells can express themselves as fibroblasts. From the state of mature fibroblasts, they can be induced to return rapidly to the phenotype of mature retinal pigment epithelial cells, including the production of pigment. This remarkable change can be produced by simply altering the media with various factors. It is tempting to speculate that alterations in the extracellular environment may have a similar effect of fundamental importance in vivo as well.

In this study, we found proliferation of the nonpigmented ciliary epithelial cells in the injured monkey eyes. By light microscopy, the nonpigmented ciliary epithelial cells appeared to be elongated and a mound of proliferating cells was visible at the ora serrata. These cells appeared to contribute to the cellular proliferation within the vitreous and could also proliferate along the detached posterior hyaloid. By electron microscopy, they were remarkably similar in appearance to the Müller cells of the sensory retina, which are developmentally related.[23]

The epiretinal membranes contained cells with the characteristics of both fibroblasts and myofibroblasts. In the epiretinal membranes associated with full-thickness retinal folds, most cells had the convoluted nuclei characteristic of myofibroblasts. The irregular configuration and distorted shape of the cell nuclei provide indirect evidence of cell contraction. Nuclear indentations and folds have been successfully correlated with cellular contraction in several other systems.[24-26] We had difficulty, however, in demonstrating the characteristic bundles of intracytoplasmic filaments in these contracted cells, but, as Rudolph and associates[27] noted in experimental skin wounds, when wound contraction is established, the characteristic intracytoplasmic filaments of myofibroblasts are much more difficult to identify. This inability to demonstrate intracytoplasmic filaments may be caused by fragmentation of the filaments by osmium tetroxide during preparation for electron microscopy,[28] or by a reorganization of actin filaments taking place within the cell cytoplasm and maintaining the cell in its contracted state.[27] These cells do contain

actin and a change in the configuration or distribution of the actin filaments within the cytoplasm may explain our difficulty in demonstrating filaments in contracted cells.

The epiretinal membranes also contained cells that were definitely glial in origin. They resembled precisely glial cells found in monkey and human eyes with rhegmatogenous retinal detachment[2,3] and in human eyes with epiretinal membranes.[10,29-34] Moreover, glial cell processes extended through breaks in the inner limiting membrane of the retina to form bridges of tissue connecting epiretinal membranes to the underlying retina.

The variability of cellular elements found in the fibrous ingrowth from the wound and in the vitreous in the injured eyes is not surprising, considering the many layers of tissues traversed by the penetrating wound. We identified many typical myofibroblasts and also cells that were remarkably similar, but possibly derived from the pigmented ciliary epithelium. We believe that the presence of such contractile cells within the vitreous and in epiretinal membranes may be responsible for vitreoretinal traction and contraction of epiretinal membranes and development of traction retinal detachment in the injured monkey eyes.

These findings in the rhesus monkey model form the basis for an understanding of the mechanisms responsible for vitreoretinal traction and traction retinal detachment after a penetrating eye injury (Fig. 10). In the injured eye, the inflammatory response may be heightened by various factors such as vitreous hemorrhage, vitreous lens admixture, or the presence of a reactive intraocular foreign body or of infection. As the inflammatory process subsides, intraocular cellular proliferation occurs and gives rise to cyclitic or transvitreal membranes and to epiretinal and retroretinal membranes. The proliferating cells include fibroblasts and myofibroblasts and cells derived from the nonpigmented and pigmented ciliary epithelium and retinal glia.

During the cellular proliferation, the vitreous becomes condensed anteriorly with reorientation of vitreous fibrils incarcerated in the penetrating wound. These alterations in the structure of the vitreous may provide a more effective

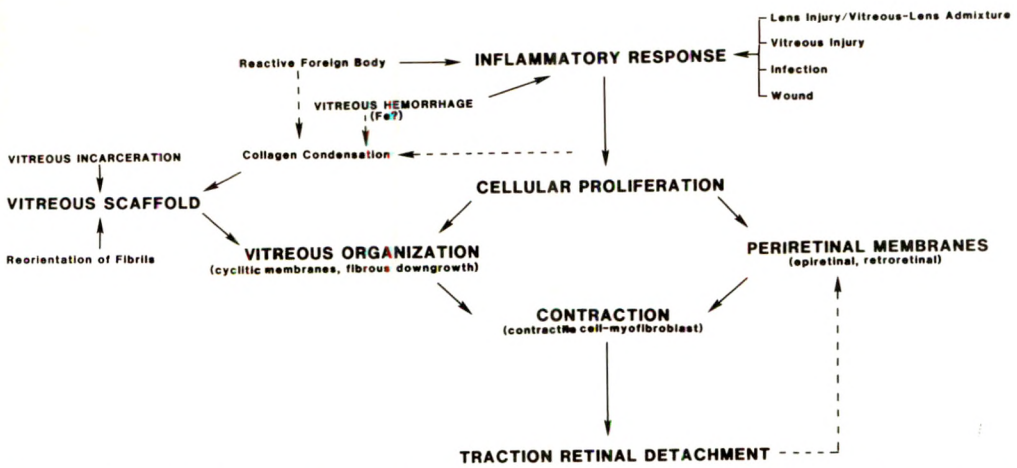


Fig. 10 (Cleary, Minckler, and Ryan). Schematic diagram of the probable mechanisms of traction retinal detachment after a penetrating eye injury.

scaffold for intravitreal cellular proliferation. Although some have placed great emphasis on shrinkage of the vitreous collagen as the mechanism producing traction, we believe that the extracellular matrix and collagen fibers are only the mediators of traction forces generated by cells.

Knowledge of the vitreoretinal anatomy is vital to our understanding of the mechanisms producing vitreoretinal traction and traction retinal detachment. In the primate eye, separation of the vitreous from the posterior retina readily occurs,[5,6] and a complete detachment of the posterior hyaloid precludes any significant anteroposterior vitreous traction. The vitreous, however, remains tightly adherent to the retina in the region of the vitreous base and, thus, when there is a penetrating scleral wound and vitreous incarceration, the effects of traction are exerted primarily on the peripheral retina.

The intraocular cellular proliferation produces transvitreal, cyclitic, epiretinal, and retroretinal membranes. In these membranes, myofibroblast-type cells contract. Cellular contraction is transmitted as traction to the retina by cell-to-cell and cell-to-vitreous connections or by glial bridges that connect the epiretinal membranes to the retina. The result of this traction is retinal detachment, which may be followed and complicated by further periretinal proliferation.

It seems likely that the invasion and proliferation of contractile cells within the vitreous and along the surfaces of the retina provide a mechanism for traction retinal detachment in a variety of conditions, including penetrating ocular trauma and massive periretinal proliferation. Possibly in the future we may be able to alter the behavior of these contractile cells by the use of pharmacologic agents. Corticosteroids may prevent or delay cellular proliferation,[35] or other pharmacologic agents may prevent cellular contraction.[36,37] Although the study of these pharmacologic agents is fraught with many difficulties, the investigative effort is more than justified by the magnitude of the problem confronting the clinical ophthalmologist. Any new therapeutic approach that may modify the growth or the behavior of these cells within the vitreous or in epiretinal membranes will offer a tremendous addition to the therapeutic armamentarium of the clinician in the treatment of complicated traction retinal detachment.

Summary

We studied the ultrastructural characteristics of an experimental model of traction retinal detachment secondary to a penetrating eye injury in the rhesus monkey. Cells with the characteristics of myofibroblasts were present in the intravitreal fibrous tissue and in epiretinal membranes. These cells were probably derived from many sources, and were capable of contraction through the mechanism of intracytoplasmic contractile proteins such as actin or myosin.

The morphologic observations supported our hypothesis that traction retinal detachment is a cell-mediated event analogous to the processes of wound healing and wound contraction. The force of contraction is mediated by cell-to-cell and cell-to-vitreous connections and by glial bridges that connect epiretinal membranes to the retina.

These ultrastructural studies, which provide insight into the pathogenesis of traction retinal detachment in the experimental animal model, may well have significant clinical relevance.

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RETINOPATHY AFTER TILORONE HYDROCHLORIDE

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Weiss, Weinberg, and Regelson[1] reported that oral administration of tilorone hydrochloride, an experimental antitumor agent, produced clinical and histopathologic ocular changes in two patients. A retrospective analysis of 14 cancer patients who had received tilorone HCl in 1974 disclosed that three patients had similar ophthalmic findings. Examination showed diffuse epithelial clouding of the cornea, sometimes associated with subepithelial infiltrates. Histologic abnormalities included cloudy swelling and intracytoplasmic inclusions in the corneal and conjunctival epithelium. There were similar inclusions in the lymphocytes. Transmission electron microscopy disclosed the presence of myelinoid bodies in the conjunctiva, cornea, and lymphocytes. Gas chromotography and mass spectrophotometry demonstrated that tilorone HCl was present in the cornea and conjunctiva.[2] Results of the remainder of the ophthalmic examinations were unremarkable. We now report the development of retinopathy in the two patients with the previously described keratopathy.

CASE REPORTS

Case 1—Because her condition did not respond to irradiation and various antineoplastic agents, a 34-year-old woman with mycosis fungoides began taking 250 mg of tilorone HCl twice daily (8.1 mg/kg/day). A preliminary ophthalmic examination showed a visual acuity of 6/6 (20/20) in both eyes. Results of color vision, biomicroscopic, ophthalmoscopic, and Goldmann perimetry tests were normal. Two months later, the patient complained of blurred vision at near and far, occurring 20 to 30 minutes after each dose of tilorone HCl, as well as the presence of blue halos around point light sources. Visual acuity remained 6/6 (20/20) and J1+ in both eyes. The biomicroscope showed subtle clouding of the corneal epithelium and anterior stroma (Fig. 1). Results of the remainder of the ophthalmic examination were normal. The dosage of tilorone HCl was decreased to 250 mg/day (4.1 mg/kg/day). One month later the patient denied all ophthalmic complaints but the corneal changes persisted. Her skin lesions, which had initially regressed, had now stabilized in size and number. The medication was then discontinued.

During the next month the skin lesions enlarged. She denied having any visual difficulties, but biomicroscopic findings in the cornea persisted. The dosage of tilorone HCl was increased to 250 mg twice daily. One month later, the blurred vision and blue halos recurred. Biomicroscopic examination disclosed an increase in the density of the subepithelial opacities. Conjunctival scraping and biopsy showed cloudy swelling of the epithelium and cytoplasmic inclusions.[1] Transmission electron microscopy showed myelinoid bodies. Wright stain and electron microscopy of a peripheral blood smear showed

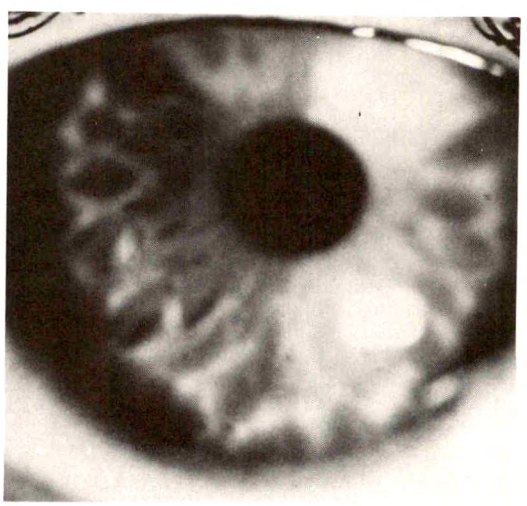


Fig. 1 (Weiss and associates). Case 1. Diffuse subepithelial clouding of the cornea.

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intracytoplasmic lymphocytic inclusions. The dosage schedule was changed to 750 mg twice a week.

Nine weeks later, she complained of intermittent flashing white lights, unrelated to the time of drug administration. Visual acuity and biomicroscopic findings were unchanged. We found no vitreous body or retinal abnormalities. The dosage of tilorone HCl was increased to 750 mg three times a week. The patient complained of foggy vision at the next two ophthalmic examinations, two and three months later, but there was no change in the ocular findings. The skin lesions were unchanged.

She independently decreased her tilorone HCl dosage to 750 mg twice weekly. At the next examination, three months later, she complained of tunnel vision and reduced night vision and noted a 9-kg weight loss during the preceding two months. Her skin tumors had increased in size and number. Visual acuity was 6/6 (20/20) and J1+ in both eyes. The subepithelial infiltrates in the corneas were denser and manifested a prominent whorl pattern. Corneal sensation was intact. Ophthalmoscopy showed fine pigment mottling of the peripheral fundus and macula and mild arteriolar attenuation in each eye. The foveal reflex was present bilaterally. Angiography confirmed the arteriolar narrowing, but no retinal pigment epithelial defects were visible. Although the patient had previously responded correctly to the 20 AOH-R-R color plates, at this time she was unable to identify plates 2 and 3 with either eye. Goldmann perimetry demonstrated marked constriction of the peripheral visual fields (Fig. 2). We performed conjunctival and corneal biopsies and scrapings. Light microscopy confirmed the previous findings of cloudy swelling of the epithelium with intracytoplasmic inclusions. Electron microscopy disclosed the presence of myelinoid bodies, as seen earlier. Similar intracytoplasmic inclusion and myelinoid bodies were demonstrated in biopsies of normal and abnormal skin and in peripheral blood lymphocytes.[1] Latex-bead and sheep cell rosetting techniques showed these inclusions in both T and B cell lymphocytes. Flash and 30′ pattern visual-evoked potentials were normal. The photopic electroretinogram (ERG) response was absent. The electro-oculogram (EOG) ratios of the light peak to dark trough was bilaterally attenuated. Tilorone HCl was again discontinued.

Four weeks after she stopped taking tilorone HCl, the patient complained of occasionally seeing "iridescent lines," like "lint on a black background." Visual acuity and biomicroscopic and ophthalmic findings were unchanged. Goldmann perimetry suggested a slight bilateral increase in the visual fields (Fig. 2). Repeat electrophysiologic testing, including a scotopic ERG, disclosed results similar to those seen before.

Case 2—A 50-year-old woman began using 750 mg of tilorone HCl twice weekly (averaging 2.5 mg/kg/day) after a brain biopsy disclosed progressive multifocal leukoencephalopathy and papova-sized virions by electron microscopy. Tilorone HCl was prescribed because of its reported antiviral

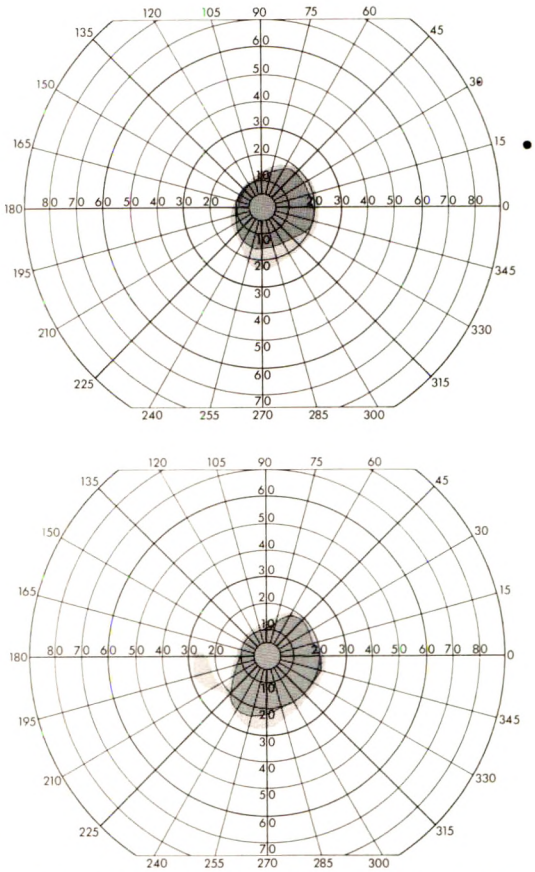


Fig. 2 (Weiss and associates). Case 1, left eye. Top, Marked constriction of the visual field. Bottom, Slight increase in the visual field three weeks after tilorone HCl was discontinued.

action, ability to induce interferon, and capacity to impede allograph rejection. Immune suppression with azathioprine was stopped and prednisone dosage reduced to 20 mg/day. Her medical history included nine years of chronic renal failure until the successful transplantation of a cadaver kidney 27 months earlier, corticosteroid-induced diabetes mellitus, and bilateral cataract extractions.

When she began taking tilorone HCl, the patient had experienced 12 months of progressive debilitation that included a right-sided hemiplegia and right-sided gaze palsy. She was, nonetheless, alert and fully responsive. An examination showed near visual acuity to be J2 in the right eye and J5 in the left eye. Color plate discrimination had been impaired more in the left eye than in the right eye. The corneas were grossly clear and normal in appearance by portable slit-lamp examination. Ophthalmoscopic examination with the pupils dilated showed only a chorioretinal scar in the superior midperiphery of the left eye. Visual fields were carefully assessed by

confrontation because of the likelihood of involvement in progressive multifocal leukoencephalopathy and were quite full. Two similar ophthalmic examinations during the next two months gave normal results.

After three months of tilorone HCl therapy, there was a slight corneal haze about the corneoscleral limbus of the left eye. During the next two years the patient complained of mild glare, pain, tearing, and burning about the eyes. Visual acuities that fluctuated between 6/6 (20/20) and 6/12 (20/40) in both eyes were attributed to her corneal condition.

A diffuse haze gradually developed, peripheral to central, more in the left eye than in the right eye. After six to eight months a whorl pattern appeared, more prominent near the corneoscleral limbus.[1] Corneal sensation remained normal. After 14 months of tilorone HCl therapy, we found the visual fields reduced to 20 to 25 degrees in all quadrants by confrontation to hand motion. We found no appreciable changes in the posterior pole.

Laboratory data was within normal limits except for the regular appearance of inclusion bodies in the lymphocytes and toxic granulations in erythrocytes of the peripheral smear. Because of the apparent arrest of progressive multifocal leukoencephalopathy and stable normal renal function, she continued taking tilorone HCl for 31 months. The total dosage was 189 g at the time it was discontinued. Subsequent conjunctival and corneal biopsies showed cloudy swelling of the epithelium with cytoplasmic inclusion and myelinoid bodies by electron microscopy, and evidence of tilorone HCl by gas chromatography and mass spectrophotometry.[1]

After 16 months without tilorone HCl, the corneal findings greatly diminished. Best corrected visual acuity was 6/7.5 (20/25) in both eyes. Color plate discrimination remained impaired in both eyes. Visual fields expanded; peripheral isopters by Goldmann perimetry were slightly suppressed but probably within the normal range for individuals with aphakia. Ophthalmoscopy showed a peripheral arteriolar attenuation with a fine pigment mottling at the macula (Fig. 3). The ERG and EOG were moderately attenuated with a normal visual-evoked potential. Tilorone HCl was still detectable in the patient's urine.

Results of electrophysiologic testing—In both patients, the amplitudes and latencies of the principal peaks of the visual-evoked potentials were within the 2.5 S.D. limits for the normal population as established in our laboratory (Fig. 4). Visual-evoked potential waveforms also conformed to the normal W shape.

In Patient 1, we could not distinguish ERG photopic or scotopic responses from background potentials. In Patient 2, the amplitudes of the photopic A waves were 18 μV in each eye, whereas the B waves measured 60 μV in the left eye and 68 μV in the right eye. The scotopic ERG showed a small or absent A wave, which is not abnormal under these conditions. The scotopic B waves measured 55 μV in the left eye and 78 μV in the right. These measurements were about 50% below



Fig. 3 (Weiss and associates). Case 2, left eye. Left, Mild arteriolar attenuation. Right, Fine pigment mottling of the macula.

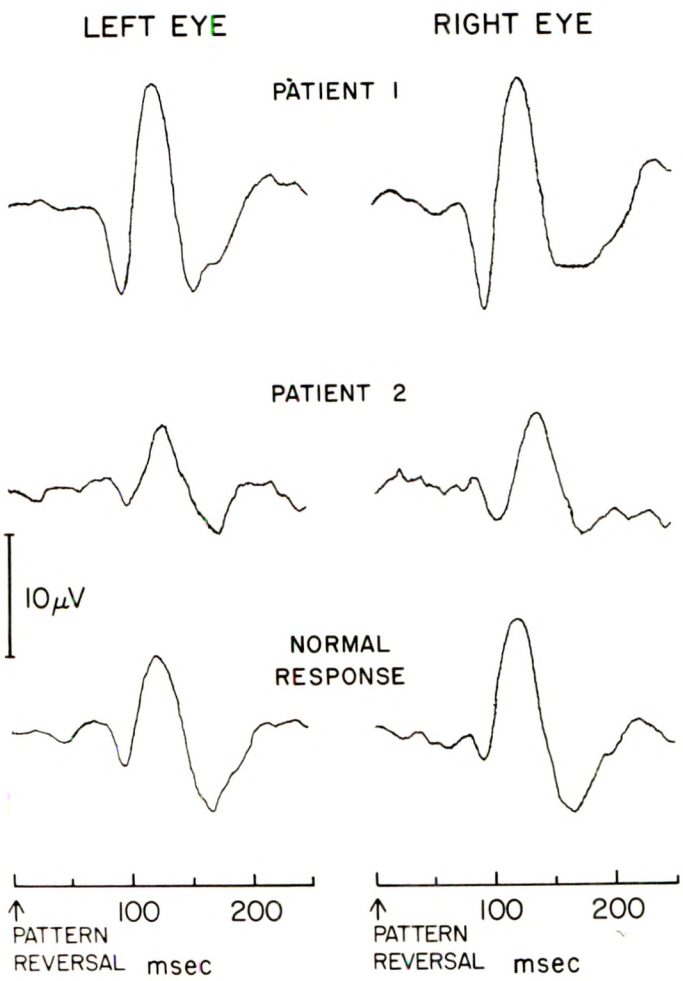


Fig. 4 (Weiss and associates). Pattern-reversal visual-evoked potentials recorded from both patients show normal amplitude and latency of the principal peaks. Their waveforms are also comparable to those found in normal subjects in our laboratory.

the 2.5 S.D. lower limit for the normal population established in our laboratory (Fig. 5). The waveform and the implicit times were normal.

The amplitude time course of the corneal standing potential, determined by EOG, is plotted in Figure 6. A clinically reliable variable is the EOG ratio (R) of the peak response under bright illumination to the lowest response (trough) obtained in darkness. We performed and interpreted this test in standard fashion.[3,4] Arden and Barrada[3] considered an EOG ratio of less than 1.85 abnormal. Accordingly, the EOG response in each eye of both our patients was attenuated.

Discussion

Though eyelid involvement in mycosis fungoides is not unusual,[5] intraocular involvement is rare.[6] Foerster[7] described a patient with mycosis fungoides who developed a chronic nongranulomatous inflammatory reaction and vitreous opacities in a quiet eye 21 years after nonpenetrating trauma. Franceshetti[8] reported cotton-wool spots in the retina of a patient with mycosis fungoides, but thought they were caused by retinopathy and not the presence of tumor cells. Sawyer and associates[9] reported severe visual loss secondary to photoreceptor degeneration

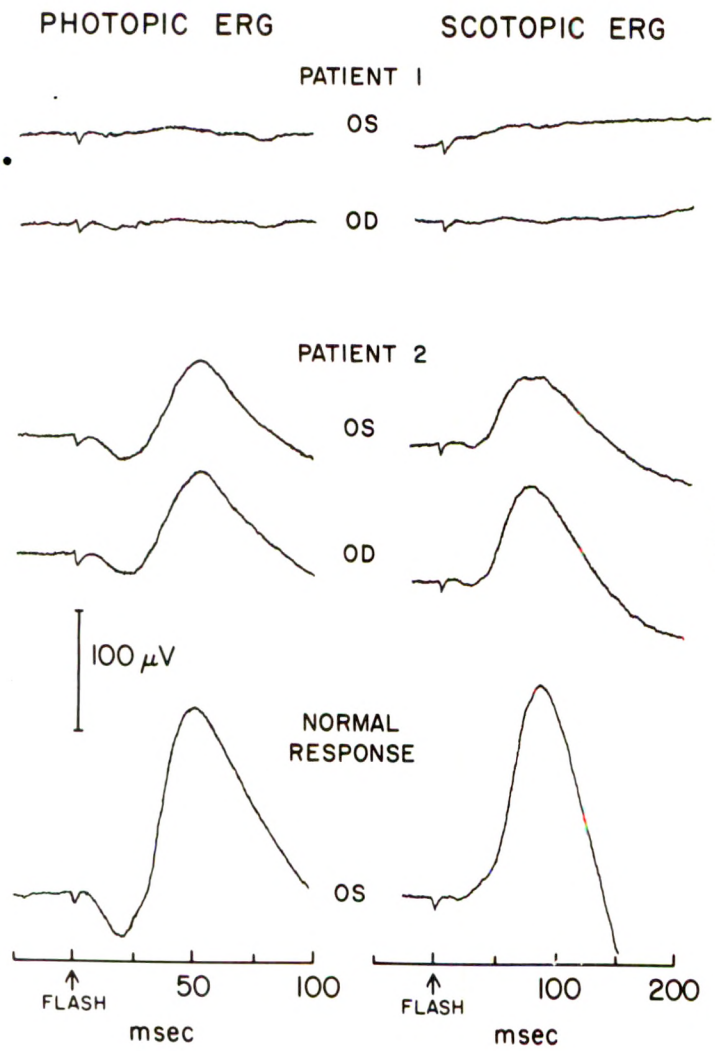


Fig. 5 (Weiss and associates). Photopic and scotopic responses are absent in Patient 1 and abnormally reduced in amplitude in Patient 2.

in three patients with highly anaplastic tumors. The retinal arterioles were narrow, and the ERG was extinguished in the one patient who had this study. One patient demonstrated a mild pigmentary disturbance. Pathologic examination disclosed total degeneration of the photoreceptors with widespread loss of cells. They suggested a remote effect of these tumors on the retina as the cause. Such effects occasionally occur with highly anaplastic tumors, particularly of the lung, but, to our knowledge, have not been reported with lymphomas. Postchiasmatic visual deficits are common in patients with progressive multifocal leukoencephalopathy,[10] but retinal involvement does not occur. The disease is a viral infection of oligodendrocytes which are normally not present in the retina. Thus, it appears that our patients' primary disease processes rarely involve the eyes.

A tilorone HCl-induced toxic retinopathy appears to be a more plausible explanation for the findings in our two patients because of the relationship between dosage and symptoms. Oral administration of

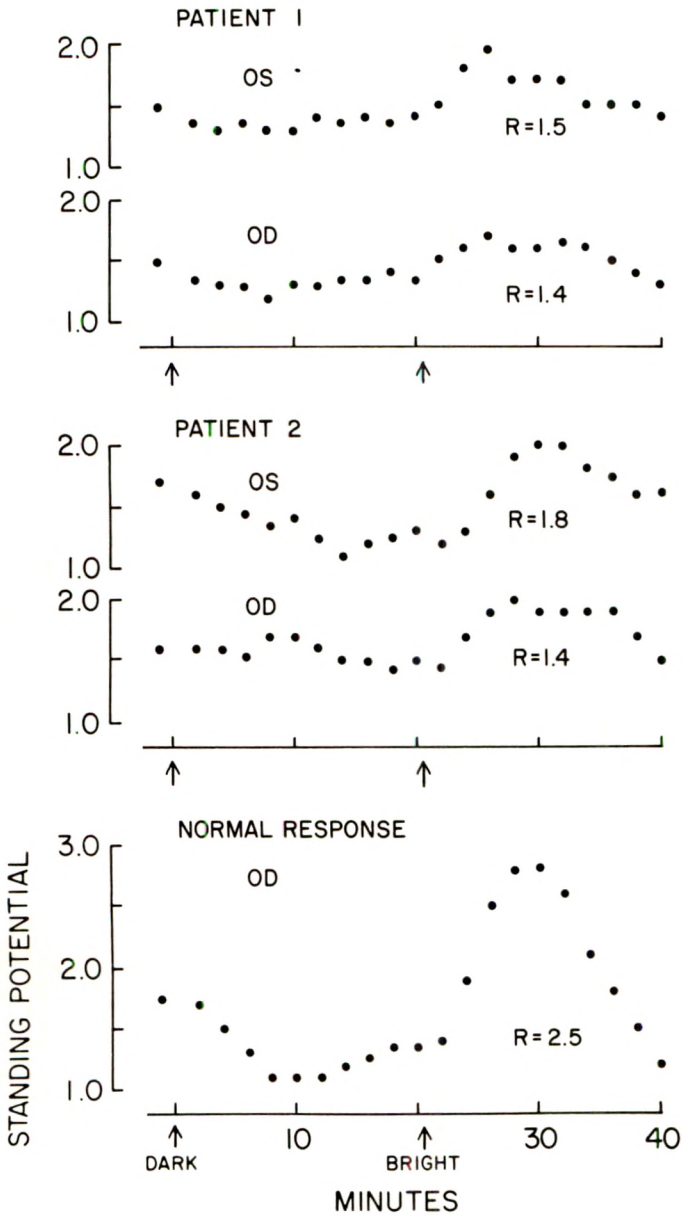


Fig. 6 (Weiss and associates). Electro-oculograms recorded from each eye of both patients show poorly defined dark troughs and low-amplitude light peak responses. Accordingly, their EOG ratios (R), defined as the amplitude ratio of the peak to the trough, are less than the lower limit of a normal response (1.85).

tilorone HCl to rats for six months caused the appearance of myelinoid bodies in the ganglion cells of the retina and cornea and in the intracytoplasmic inclusion bodies in the lymphocytes.[11]

In a previous article, we discussed the similarities between tilorone and chloroquine.[1] Further work had demonstrated that the two drugs have many more features in common: (1) long-lasting tissue-binding affinity[12,13]; (2) dose-duration-related reversible keratopathy[14]; (3) production of myelinoid bodies[15] in the cornea, conjunctiva, and skin; and (4) dose-duration-related retinopathy shown by retinal pigment epithelium mottling,

arteriolar narrowing, and abnormal ERG and EOG. These changes may be reversible, depending on the drug dosage and duration of treatment.[14,16-18] Both tilorone (rats) and chloroquine (monkeys)[19] affect the ganglion cells of the retina.

Chloroquine's role as an antioxidant may explain its affinity for tissues with active free radical scavenging mechanisms. We hypothesize that tilorone HCl is also an antioxidant; this may account for its effects on the cornea, retina, and skin which resemble those of chloroquine.

Since chloroquine retinopathy is reversible in the early stages, presumably the one induced by tilorone HCl is also. This makes early detection imperative. A reduction of a patient's EOG ratio is believed to be a more reliable indicator of chloroquine retinopathy than the amplitude of the ERG,[20] and Van Lith, Mak, and Witnands[21] suggested that a baseline EOG be performed before antimalarial chemotherapy. Given the similarity in chemotoxicity, a baseline EOG should also be obtained when tilorone HCl therapy is considered. Carr, Gouras, and Gunkel[22] demonstrated that perimetric threshold testing to red light is also a sensitive indicator of early retinal damage. Because of the clinical similarities between chloroquine and tilorone HCl, techniques for early detection of chloroquine retinopathy should be applied to detect possible tilorone HCl retinopathy.

Summary

Two patients, a 34-year-old woman and a 50-year-old woman, received tilorone HCl, an experimental antitumor drug. After taking the drug orally (total dose, 152 g), the first patient developed corneal subepithelial infiltrates and toxic retinopathy characterized by fine pigment mottling of the peripheral fundus and macula with mild arteriolar narrowing. Although visual acuity was 6/6 (20/20) throughout treatment, Goldmann perimetry showed marked peripheral constriction of the visual fields and results of an electroretinogram and an electro-oculogram were abnormal.

After taking the drug orally (total dose, 189 g), the second patient developed corneal subepithelial infiltrates, severe bilateral arteriolar narrowing, and mild pigment mottling of the macula. ERG and EOG were moderately attenuated. Visual fields by Goldmann perimetry were within normal limits.

Tilorone HCl, like chloroquine, may be an antioxidant that affects the free radical scavenging mechanism of the retinal pigment epithelium. Extensive testing should be done on all patients taking tilorone HCl in order to detect the initial manifestations of retinopathy.

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SCANNING ELECTRON MICROSCOPY OF TRABECULECTOMY SPECIMENS FROM EYES WITH OPEN-ANGLE GLAUCOMA

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The cause of increased intraocular pressure in open-angle glaucoma is generally believed to be decreased outflow of aqueous humor. Anatomic studies of the outflow channels in human eyes with glaucoma have provided little information about the cause of this abnormality.[1-8] Recent transmission electron microscopic studies have suggested that there is an increased accumulation of extracellular material in the area near the inner wall of Schlemm's canal.[2,4,5] Others have pointed out that little is known about the presence and amount of such extracellular components in the normal, aging angle.[6,9] One recent examination of trabeculectomy specimens from cases of open-angle glaucoma described an apparent obstruction of the pores of the trabecular meshwork in the zone closest to the anterior chamber.[10] While this had not been observed in any previous study, it is a finding of potential importance. If confirmed, the nature of this obstructing material might provide information on outflow abnormality in glaucoma.

We examined 13 trabeculectomy specimens from patients with open-angle glaucoma by scanning and transmission electron microscopy to evaluate the patency of the intertrabecular pores.

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MATERIAL AND METHODS

We included trabeculectomy specimens only from cases of primary open-angle glaucoma with visual field loss but no history of trauma or surgery (Table). One of us (H. A. Q.) obtained all the specimens at surgery, using an identical procedure in each instance. After preparing a conjunctival and scleral flap at the superior corneoscleral limbus, the surgeon made a knife puncture (Ziegler) in clear cornea, avoiding a major loss of aqueous humor. Immediately thereafter, he performed the trabeculectomy with a razor blade, removing a piece of tissue measuring approximately 1.5 × 3 mm. Histologic examination later confirmed that 13 of 15 specimens contained peripheral cornea, Schwalbe's line, and trabecular meshwork. The excision was rarely made posterior to scleral spur. The specimen was grasped with fine forceps in only one location and was immediately immersed in 1% glutaraldehyde and 4% formaldehyde buffered in phosphate at pH 7.2, with 1 g of sucrose per 100 ml. No more than 15 seconds elapsed between removal of the specimen and fixation. We washed each specimen briefly in 0.05 N cacodylate buffer and postfixed it in 1% osmium tetroxide in cacodylate; we then dehydrated it in acetone. It was critical-point dried, and coated with gold palladium. After scanning microscopy, several specimens were rehydrated and embedded in epoxy resin. We prepared ultrathin sections for transmission electron microscopy after staining them with uranyl acetate and lead citrate.

TABLE

SUMMARY OF CLINICAL DATA

Patient, Sex, Age (yrs)	Duration of Glaucoma (yrs)	Involved Eye	Therapy	Other Ocular Diseases
1, F, 65	4	Left	Pilocarpine, timolol, epinephrine, acetazolamide	Pseudoexfoliation
2, F, 79	15	Right	Pilocarpine, timolol, epinephrine, acetazolamide	Macular degeneration
3, M, 72	9	Left	Pilocarpine, epinephrine, acetazolamide	Fuchs' dystrophy
4, M, 17	2	Left	Pilocarpine, epinephrine, acetazolamide	———
5, M, 33	6	Both	Pilocarpine, timolol, acetazolamide	———
6, M, 50	1	Both	Pilocarpine, timolol, acetazolamide	———
7, F, 58	7	Both	Pilocarpine, timolol, acetazolamide	———
8, M, 69	7	Right	Pilocarpine, epinephrine, acetazolamide	Cataract
9, F, 67	1	Left	Pilocarpine, timolol, acetazolamide	———
10, F, 47	15	Right	Pilocarpine, timolol, epinephrine, acetazolamide	———

RESULTS

In 13 trabeculectomy specimens from ten patients, there was enough trabecular meshwork for examination. In two other specimens obtained during the same time period, no meshwork was visible, owing either to damage or anterior excision. None of the 13 trabecular meshwork pieces had material obstructing the pores at the anterior chamber surface (Figs. 1

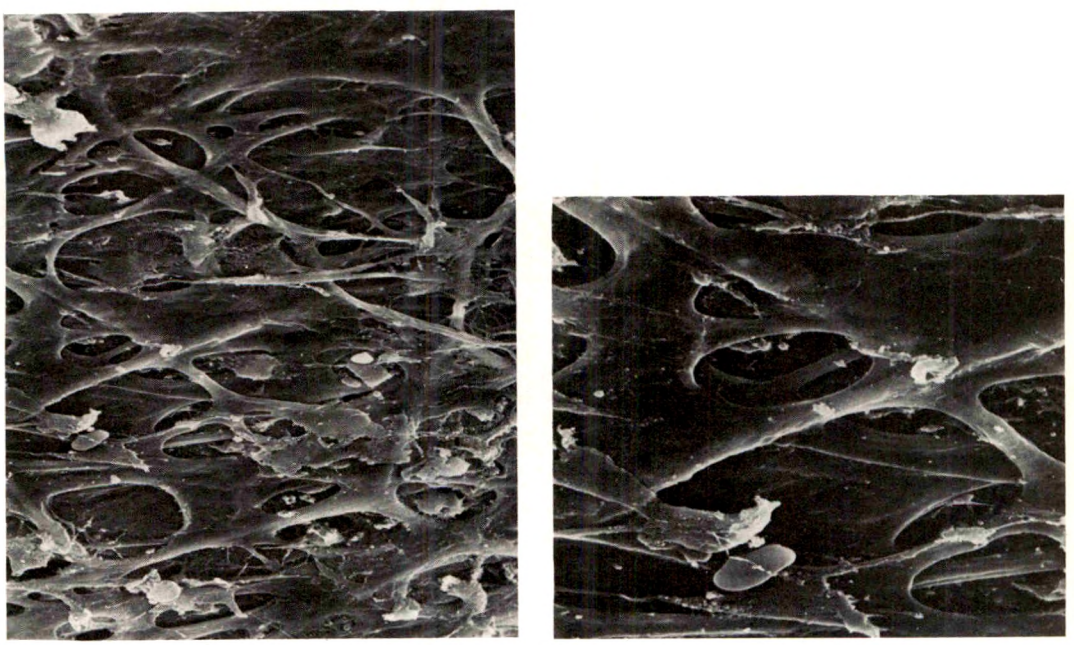


Fig. 1 (Quigley and Addicks). Patient 8. Left, Trabecular meshwork of a 69-year-old man with open-angle glaucoma viewed from anterior chamber side. While there is a small amount of extracellular debris, most pores appear open (× 1,300). Right, Higher-power view of normal uveal trabeculum of same specimen (× 2,600).



Fig. 2 (Quigley and Addicks). Patient 7. Trabecular meshwork with open pores and a small amount of material, chiefly red blood cells, deposited on it (× 1,800).

to 5). In three specimens, we saw red blood cells and fibrin, caused by hemorrhage at the time of excision, on the inner surface (Figs. 2, 3 and 5). In each specimen, the normal trabecular structure was easily visible. The innermost sheets



Fig. 4 (Quigley and Addicks). Patient 4. The pores are clearly open through at least three layers of trabecular sheets (× 1,800).

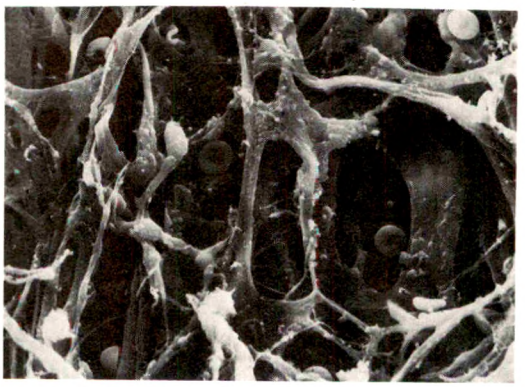


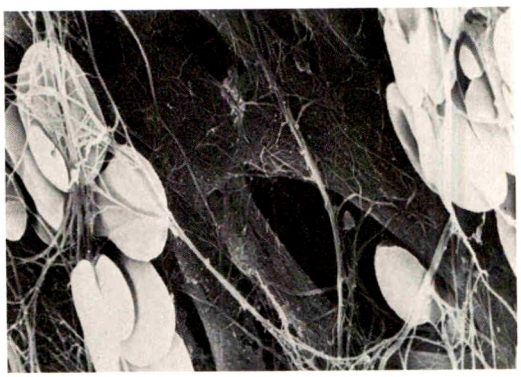
Fig. 3 (Quigley and Addicks). Patient 2. Normal-appearing trabeculum from a 79-year-old woman with glaucoma (× 1,600).

(uveal meshwork) had a round shape, while those in the outer meshwork, seen through the inner pores, were broader and flatter (corneoscleral meshwork). Even in the one case of pseudoexfoliation syndrome, open pores were present, with no visible extracellular accumulation of material in the inner meshwork.

Light and transmission electron microscopy of the re-embedded trabeculectomy specimens disclosed no material obstructing the inner trabecular pores (Fig. 5, right). The fine structure of the meshwork was not perfectly preserved after the secondary embedding.

Discussion

We were unable to confirm the finding of Chaudhry and associates[10] that the pores of the inner trabecular meshwork are obstructed in trabeculectomy specimens from eyes with open-angle glaucoma. We took great care to maintain optimal tissue preservation by careful handling of the specimens and immediate fixation. Our fixative differed only in minor details from that of the previous report. The surgical methods used in obtaining the specimens in their series may have been different. The patient populations might have differed some-



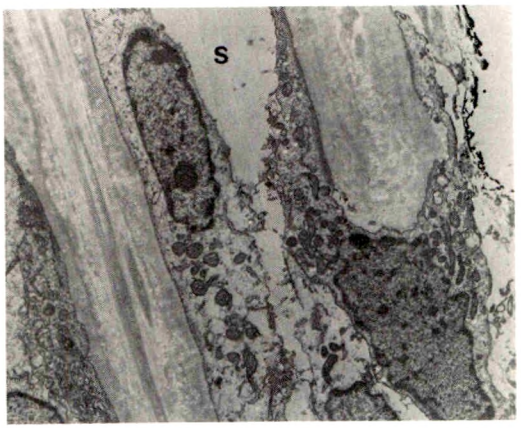


Fig. 5 (Quigley and Addicks). Patient 6. Left, Red blood cells are trapped in a network of fibrin, but this does prevent an adequate view through the trabecular pores (× 5,300). Right, Transmission electron micrograph of same specimen. Connective tissue sheets of the trabeculum are covered by endothelial cells with normal, open intertrabecular spaces (S). The anterior chamber is at right, indicated by dense black gold-palladium coating from scanning microscopy preparation (× 11,300).

what; however, this seems an unlikely explanation of the difference in results. Extracellular materials may be removed by the several exposures to preparation solutions involved in electron microscopy. While it is possible that our techniques led to a washing away of the material observed by Chaudhry and coworkers, it seems equally likely that some material not present in the living eye became deposited on the trabeculum during their tissue preparation. Further examination of similar material by our laboratories and others seems indicated. Should such obstructing material be found, it should be examined by histochemistry and transmission electron microscopy.

Summary

We examined 13 trabeculectomy specimens from ten patients with open-angle glaucoma by scanning electron microscopy. The pores of the inner trabecular meshwork were not obstructed by extracellular material in all specimens, a finding contrary to a recently published report.

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PERIORBITAL SCLERODERMA ASSOCIATED WITH HETEROCHROMIA IRIDIS

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Linear scleroderma has clinical features similar to progressive hemifacial atrophy (Romberg's syndrome); often a clear distinction between the two entities is not made.[1-3] The two processes, however, appear to be distinguishable clinically and histopathologically.[4] We describe two patients who had biopsy-proven linear scleroderma (morphea) that involved periorbital tissues. Both patients had an associated pigmentary abnormality of the anterior segment of the eye. One had heterochromia iridis and a markedly asymmetric pigmentary glaucoma, and the second had heterochromia iridis.

CASE REPORTS

Case 1—When first examined in 1940 at the age of 14 years, the patient had a best corrected visual acuity of 6/6 (20/20) with a −1.25 sphere in each eye. There were no ocular abnormalities.

In 1945, the patient developed a gradual loss of cilia, almost complete one year later, from the outer half of the right upper eyelid and a corresponding area of slight depigmentation of the adjacent skin. The lower border of right upper tarsal plate was atrophic. In 1946 the margin of the right upper eyelid was inflamed; there was mild palpebral conjunctival congestion. The cornea remained normal.

In 1947, an imbedded corneal foreign body was removed from the right eye. Sodium sulfadiazine was instilled and the eye covered with a patch overnight. Despite subsequent treatment with penicillin drops, the cornea became secondarily infected and a severe corneal ulcer developed. The patient was hospitalized and treated with penicillin, sodium sulfadiazine, streptomycin, and fever therapy. The cornea healed slowly and an avascular scar about 2 mm in diameter developed just inferonasal to the corneal center. The best corrected visual acuity in the right eye was 6/12 (20/40).

The margin of the right upper eyelid continued to resorb, and over the following years a "coup de sabre" lesion developed over the right temporal region. The patient consulted a dermatologist on several occasions. A skin biopsy specimen showed localized scleroderma.

In 1957, examination showed normal fundi but the intraocular pressure was 25 mm Hg in the right eye and 20 mm Hg in the left eye. He was not seen again until 1962, when he noticed a marked drop in the visual acuity of his right eye (hand movements). Intraocular pressure was 50 mm Hg in the right eye and 21 mm Hg in the left eye. The right visual field showed marked constriction and nasal loss; the left visual field was normal. The right optic disk showed glaucomatous cupping; the left optic disk was normal. There was heterochromia with the right iris appearing lighter in color than the left. Gonioscopy showed the right trabecular meshwork to be grade 4 pigmented and the left trabecular meshwork to be grade 3 pigmented.[5] The midperiphery of the right iris showed continuous transillumination while the left iris showed only scattered transillumination. Tonographic outflow values were 0.09 in the right eye and 0.16 in the left eye. Results of a water provocative test were positive in the right eye.

The diagnosis was pigmentary glaucoma in the right eye and the pigment dispersion syndrome in the left eye. Pilocarpine HCl, 4%, initially controlled the intraocular pressure in the right eye. With time, the intraocular pressure increased in the left eye, and the eye was treated with 1% pilocarpine HCl. The optic disk and field in the left eye remained normal (Fig. 1).

In 1978, the intraocular pressure had increased bilaterally. The patient began using an ophthalmic mixture of pilocarpine HCl and epinephrine bitartrate (P6E1) in the right eye and 2% pilocarpine HCl in the left eye. Several weeks later he developed a temporal retinal detachment in the left eye associated with a horseshoe tear. A scleral buckling procedure successfully reattached the retina. We have controlled the patient's intraocular pressures with 4% pilocarpine HCl in the right eye only.

Case 2—In 1953 when he was 5 years old, the patient had tinea capitis and he received local therapy, with no radiation. He had been struck in the head with a golf club in 1952 and had also sustained two other head injuries, the sites of which were unknown, and which were apparently unrelated to the present problem. The patches of alopecia continued to appear atrophic, but considerable regrowth of hair occurred. The partially bald spots then began to fuse, and a furrow began to form on the right side of the scalp. The furrow became continuous with a scar on the right upper eyelid.

By 1955, sufficient retraction occurred to distort the right upper eyelid. A small new furrow began to

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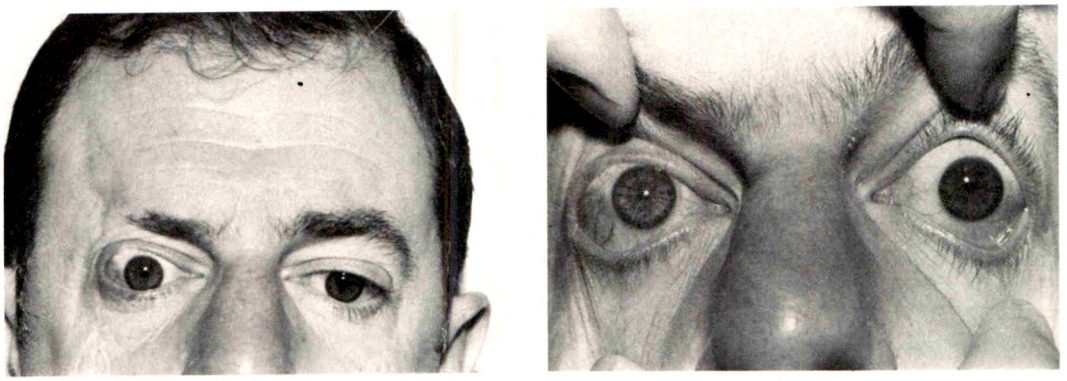


Fig. 1 (Stone and Scheie). Patient 1 at 47 years of age. Left, The coup de sabre lesion and atrophy of the right upper eyelid. Right, Heterochromia, with the right eye having a lighter iris.

develop on the left side of the scalp and continued to increase in length. A biopsy of the scalp showed atrophy of the overlying epidermis and fibrosis, edema, and chronic inflammation of the underlying dermis. Hair follicles and skin appendages were absent except for several small, distorted cell clusters within the superficial dermis. Homogeneous acidophilic collagen bundles and a few thick-walled vessels were present. These findings were consistent with scleroderma, morphea type.

Within the next two years, blepharoptosis of the left upper eyelid developed and became almost complete. He also had 18-diopter exotropia and 30-diopter hypotropia in the left eye.

Medical and neurological examinations gave normal results, except for the changes in the scalp and eyes. X-rays of the skull, chest, and esophagus showed normal findings. A complete blood cell count and urinalysis were within normal limits. An electroencephalogram showed showers of high-voltage, abnormal slow waves bilaterally, 4 to 6 per second, and bursts and showers of high-voltage slow waves, 3 to 4 per second, with hyperpnea. His neurologist felt that these electroencephalographic changes were nonspecific and of no clinical importance.

In June 1962, the patient underwent a 5-mm recession of the inferior rectus muscle of the left eye. Two months later, he had a palpebral superioris levator muscle resection in this eye with good results. In 1967, an incision was made in the upper tarsal plate of the right eye to lower the retracted right upper eyelid.

The patient was next seen 12 years later when he reported that the blepharoptosis of the left eye had gradually recurred over the past few years. We noted a marked blepharoptosis. A definite heterochromia was also apparent, the right iris being lighter in color than the left. There were no discrete foci of transillumination. Gonioscopy showed both angles to be wide open and nonpigmented (Fig. 2).

Discussion

Localized scleroderma, or morphea, is generally confined to the skin. The lesion



Fig. 2 (Stone and Scheie). Patient 2 at the age of 31 years. Left, Coup de sabre lesion is more prominent on the right. The right upper eyelid is retracted and there is heterochromia. The left eye is exotropic and hypertropic; blepharoptosis has recurred. Right, The iris of the right eye is lighter.

occurs most frequently in the lower extremities, but can occur elsewhere. It generally appears as an acute, edematous, infiltrative lesion involving the entire thickness of the dermis, progressing to sclerosis and atrophy of the skin and its appendages. Associated hair loss occurs when the scalp or eyelids are involved. The single lesion is generally well circumscribed. The skin often adheres to the subcutaneous tissues. Lesions in the paramedian or frontoparietal region of the scalp produce the typical coup de sabre deformity, a linear, depressed plaque with hyperpigmented borders, telangiectases, and alopecia that may extend from the scalp to the neck.[4]

The histologic findings of localized scleroderma depend upon the age of the lesion at the time of the biopsy. In late stages, as in our patients, hair follicles and sweat glands are lost and sclerosis of intradermal collagen and thickening of dermal vessel walls with luminal narrowing are found. Trauma, as in Case 2, is sometimes cited as a provocative factor.[4]

In contrast, progressive hemifacial atrophy generally causes a more diffuse change. The skin tends to remain soft and does not adhere to underlying tissues, and localized hair loss is a less prominent feature.[4] Also, the elastic tissue is preserved.[6]

In addition to skin and eyelid changes, ocular findings associated with localized scleroderma include iritis, sector iris atrophy,[1] retinal hemorrhages,[7] corneal epithelial opacities,[8] decreased corneal sensation, phthisis bulbi with uveitis and cataract, ipsilateral neurocytoma of the optic nerve,[9] and paresis of ocular muscles, particularly to the superior and medial recti.[1,9] Our Patient 2 showed marked involvement of extraocular muscles and also had paresis of the palpebral superioris levator muscle of the left eye.

Heterochromia has previously been associated with progressive hemifacial atrophy,[10,11] but not with localized scleroderma.[9] Because a clear distinction between these two entities is not always made, cases of localized scleroderma with heterochromia may have been incorrectly reported as progressive hemifacial atrophy. In Patient 1 the pigment dispersion problem was bilateral, although the morphea lesion was unilateral. The pigment dispersion was more severe, however, on the side with the scleroderma lesion. Aqueous humor dynamics were more abnormal on the involved side as shown by the earlier increase in intraocular pressure, by the more impaired outflow facility on early tonography, and by the greater difficulty of controlling intraocular pressure. The eye on the involved side also developed glaucomatous visual field loss. In Patient 2, who had bilateral morphea lesions, heterochromia developed during the 27-year follow-up period.

Summary

Two patients had biopsy-proven linear scleroderma, associated with a pigmentary abnormality of the anterior segment of the eye. One patient had heterochromia iridis, atrophy of the upper eyelid, a typical coup de sabre lesion, and a markedly asymmetric pigmentary glaucoma. The second patient had bilateral scalp involvement, paresis of the extraocular muscles, blepharoptosis, and heterochromia iridis.

Acknowledgment

Louis Jaffe, M.D., provided the medical history of Patient 2.

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THE KERATOPATHY OF CROHN'S DISEASE

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A significant number of patients with Crohn's disease (granulomatous ileocolitis) have ocular problems.[1] The corneal and external ocular manifestations reported in Crohn's disease include recurrent conjunctivitis, marginal corneal ulcers, keratitis, blepharitis, dry eye, scleritis, and episcleritis.[1-8]

Previous reports have focused on acute, inflammatory corneal problems or scleritis and corticosteroid-responsive marginal ulcers.[6] A static or slowly progressive keratopathy has been briefly described.[8] In this report we describe four patients with a keratopathy that we have seen only in patients with Crohn's disease. Inflammatory bowel disease was diagnosed before the finding of their corneal disorder in three patients. In the fourth patient, characteristic corneal findings were so suggestive of Crohn's disease that, although he was otherwise asymptomatic, examination by an internist and upper gastrointestinal radiographic studies led to the diagnosis of Crohn's disease.

CASE REPORTS

Case 1—A 39-year-old man receiving treatment for Crohn's disease had no unusual findings at an ocular examination in September 1969. In March 1971, we noted a corneal abnormality. Visual acuity was R.E.: 6/6 (20/20) and L.E.: 6/5 (20/15), with small (0.3 to 0.5 mm in diameter), rounded elevations of the corneal epithelium and subepithelial infiltrates, located primarily 2 to 3 mm inside the entire corneoscleral limbus in both eyes. We were not sure whether the subepithelial infiltrates represented epidemic keratoconjunctivitis or if they were part of a previously undescribed keratopathy.

The central corneal has remained clear and the patient has remained asymptomatic. Since December 1971, the only changes have been flattening and coalescence of infiltrated and elevated areas and the development of new elevated thickenings. In January 1973, Schirmer tests showed 18 mm of wetting in both eyes and possible staining of the cornea over the lesions. A serum carotene determination at that time was 65 mg/100 ml (normal, 50 to 300 mg/100 ml). He began taking vitamin A orally; in February 1973, we thought that the corneal lesions were somewhat improved. However, by July 1973 he stopped taking vitamin A because we believed that the corneal lesions were not changing or resolving. When we last examined the patient in February 1980, visual acuity was R.E.: 6/5 (20/16) and L.E.: 6/6 (20/20). The findings remained basically unchanged (Fig. 1) and the patient was asymptomatic.

Case 2—This 53-year-old woman had had Crohn's disease since the age of 46 years and had been taking prednisone systemically for three months. Her ocular history was unusual only because the ophthalmologist who had examined her regularly during the previous 17 years had never mentioned that there was anything wrong with her corneas. Nine months previously, she had noted a "sandy feeling" in both

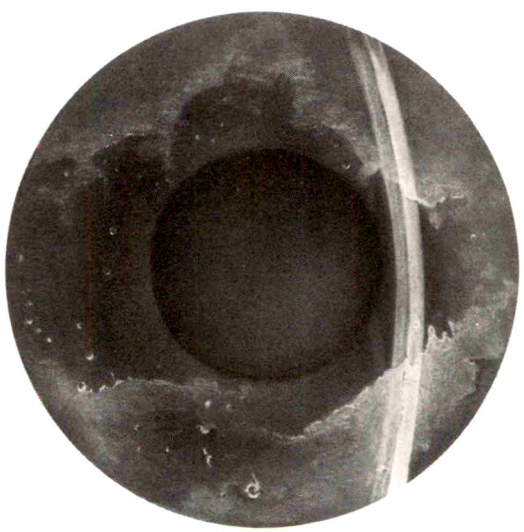


Fig. 1 (Knox, Snip, and Stark). Case 1. Diffuse corneal scarring and punctate infiltrates, February 1980.

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Fig. 2 (Knox, Snip, and Stark). Case 2. Diffuse corneal scarring, November 1973.

eyes and was examined by her ophthalmologist who at first diagnosed a keratitis and treated her with eyedrops for approximately one month without obtaining relief. She was referred to several other ophthalmologists who advised artificial tears and then was referred to one of us (D.L.K.) for examination.

On examination, in November 1972, corrected visual acuity was 6/6 (20/20) in each eye. The only unusual features were peripheral opacities in the corneas (Fig. 2). Both corneas were symmetrically hazy anteriorly with densities greater below than above. We noted no nodularity, vessels, or staining, and the epithelium was intact. There was possible thickening of the epithelium in the limbal areas. Schirmer testing with topically applied anesthesia was 0 mm of wetting at five minutes. There was no Rose-Bengal staining of the cornea. Our impression was that she had a keratopathy of unknown origin, similar to that seen in Case 1, and dry eyes, probably related to her Crohn's disease. Her serum carotene level, tested to evaluate a possible relationship to vitamin A, was 82 mg/100ml (normal, 50 to 300 mg/100 ml).

When we examined her again on Jan. 5, 1973, she had normal visual acuity, foreign-body sensation of the right eye, no apparent change in her cornea, and minimal tear film. We prescribed artificial tears, 5,000 units of vitamin A, and 500 units of vitamin D per day.

She returned on Jan. 15, 1973, with her eyes feeling less sticky. The tear film was better and Schirmer tests with topically applied anesthesia showed 15 mm of wetting in the right eye and 11 mm of wetting in the left eye at five minutes. In March 1973, we thought that there was a slight clearing of infiltrates. Schirmer tests repeated at that time showed 15 mm of wetting in the right eye and 5 mm of wetting in the left eye. Serum carotene levels had risen to 106 mg/100 ml. In April 1973, she stopped taking the vitamins.

In May 1974, visual acuity was 6/8 (20/25) in her right eye and 6/6 (20/20) in her left eye. Schirmer tests showed 12 mm of wetting in the right eye and 24 mm of wetting in the left eye at five minutes. We believed the corneas were unchanged except for a possible new area of involvement in the right eye. She has not returned for further examination.

Case 3—This 34-year-old woman had had Crohn's disease since she was 29 years old, four years before her ocular findings were noted. About two months before we examined her, her ophthalmologist found some "interesting corneal changes" during a routine examination. She was asymptomatic and had not been examined for many years previously. She was referred here for diagnosis in June 1977.

On examination her best refracted visual acuity was 6/5 (20/15) in each eye. She denied having any visual symptoms. At that time her intestinal symptoms were controlled by prednisone, 15 mg every other day. Schirmer tests with topically applied anesthesia showed 20 mm of wetting in the right eye and 25 mm of wetting in the left eye at five minutes. The only unusual finding involved the corneas.

On slit-lamp examination she had subtle subepithelial nodular opacities of the superior and inferior corneal stroma (Fig. 3). These were present only in the subepithelial tissue and there was no apparent epithelial involvement. There was no corneal staining and the patient was asymptomatic. The central palpebral optical axis was spared.

Case 4—This 37-year-old man seemed to be in good general health when he saw his ophthalmologist who noted bilateral corneal infiltrates and referred him for diagnosis. When first examined in March 1979, he told us that some abnormalities in his corneas had been noted two years previously. Since that time he had had occasional irritation which was helped by Lacri-Lube. There were no other unusual findings

On examination, his best corrected visual acuity was 6/9 (20/30) in both eyes. His conjunctiva and sclera were white and quiet. The corneas showed subepithelial scarring and reduplication of basement membrane inferiorly with minimal changes in the more central and superior aspects (Fig. 4). The corneas did not stain. His tear function was normal.

We believed that his corneal changes warranted a medical examination for possible Crohn's disease. He was referred to his internist, who, at the patient's insistence, requested upper gastrointestinal and small bowel X-rays. They disclosed extensive Crohn's disease involving two 20-cm lengths of the mid and distal ileum. Since that time he has been observed.

Discussion

Although ocular abnormalities in Crohn's disease have occurred in as

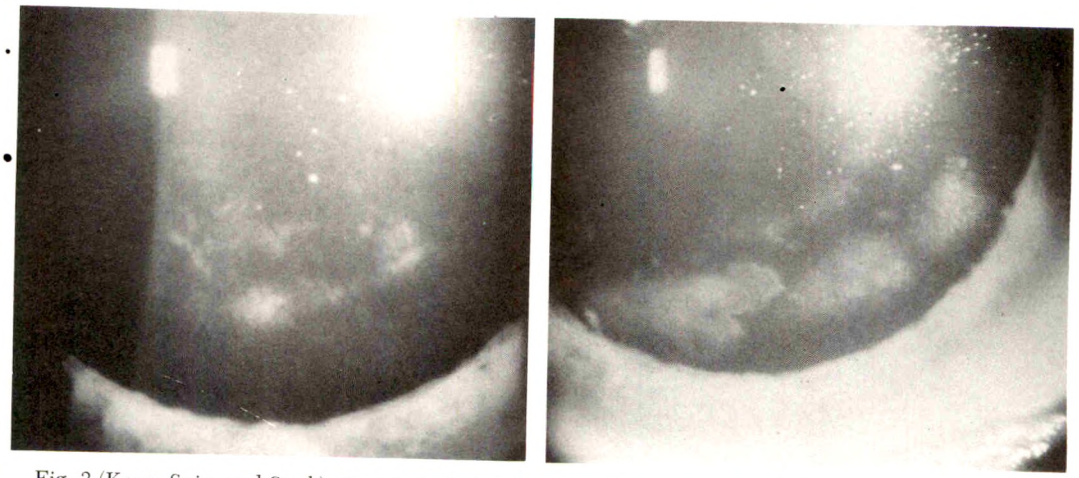


Fig. 3 (Knox, Snip, and Stark). Case 3. Left, Subtle corneal scarring in the right eye. Right, Overt corneal scarring in the left eye.

many as 6.3% of patients in one large series,[1] the distinct keratopathy described here is uncommon. We have made no attempt to establish the incidence or prevalence of this keratopathy in patients with Crohn's disease. These four cases represent the total number that we have recognized during 17 years of interest in ocular manifestations of systemic disease (D.L.K.).

At this time there is no histopathologic evidence of the nature of this corneal lesion. We have chosen not to biopsy these lesions. In Case 2, symptoms of dry eye and reduced tear production as measured by Schirmer test responded to

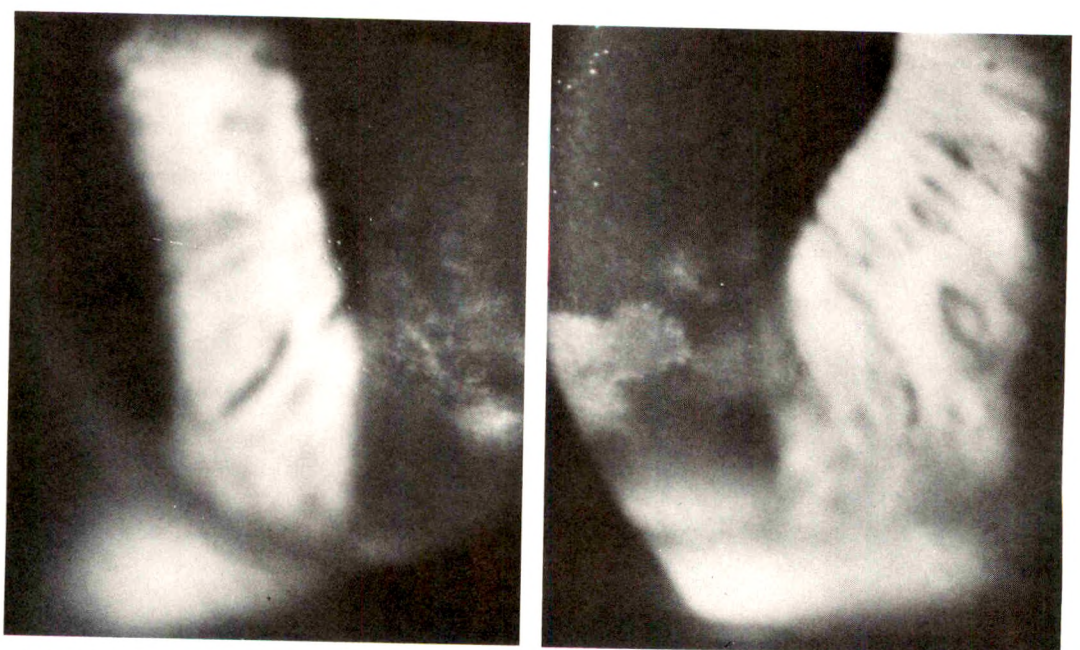


Fig. 4 (Knox, Snip, and Stark). Case 4. Left, Subtle corneal scarring in the right eye. Right, Overt corneal scarring in the left eye.

orally administered vitamin A, but the corneal lesions remained unchanged. In Case 1, vitamin A therapy did not change the corneal lesions.

We cannot explain this keratopathy, particularly since we do not have histopathologic data to support the clinical findings. Since arthritis and psoriasis are often complications of Crohn's disease, it is possible that some type of autoimmune or hypersensitivity inflammatory change in the peripheral cornea may be responsible for the biomicroscopic findings. The infiltrates seem to be of two types: the first is an epithelial or subepithelial infiltration and elevation seen in the anterior cornea as small gray, pale dots; the second type is deep lamellar nebulous subepithelial infiltrates or scarring. These seem to be more marked inferiorly, but appear superiorly as well. Possibly the first type represents an acute inflammatory phase and the later scarring is secondary.

This keratopathy is basically a benign manifestation of Crohn's disease. It has not interferred with vision, but is a distinct clinical sign.

SUMMARY

A distinctive subepithelial keratopathy developed in four adult patients with Crohn's disease (granulomatous ileocolitis). Small, bilateral, symmetrical subepithelial, and anterior proliferative infiltrates and scarring were present 2 to 3 mm inside the entire corneoscleral limbus. Vision was not affected because the lesions spared the central cornea. The mechanism is not known and the lesion should be regarded as a distinct clinical sign of Crohn's disease.

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ALKALINE TEAR pH IN OCULAR ROSACEA

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Ocular rosacea is a common yet frequently undiagnosed condition.[1] Dermatologic manifestations may precede ocular involvement, but ocular findings were observed before skin changes in 20% of the 99 cases reported by Borrie.[2]

Acne rosacea is a chronic hyperemia of the facial skin, usually involving the nose, central forehead, and upper cheeks. It is characterized by erythema, telangiectasia, papules, pustules, hypertrophic sebaceous glands, and, in the most advanced form, rhinophyma. The main ocular manifestations include blepharitis, conjunctivitis, keratitis, and, more rarely, episcleritis. If left untreated, rosacea keratitis can progress steadily toward corneal melting with vascularization and stromal scarring as the end result.

The cause of ocular rosacea is unknown. Although tetracycline is usually successful in treating both the ocular and dermatologic aspects of the disease,[3,4] improvement does not seem to be related to the antibacterial effect of the drug.[5,6] Tetracycline does not cure rosacea; its action is purely suppressive.[7]

Associations between acne rosacea and other conditions have been made.[8,9] Of particular interest is the long-established correlation between rosacea and hypochlorhydria.[10,11]

The diffusely hyperemic conjunctiva of one patient with ocular rosacea led one of us (M.B.A.) to inoculate that patient's tears into one of his own eyes. The investigator's eye burned immediately and became injected. This finding led to a study of possible changes in tear pH values in ocular rosacea. We report herein the tear pH values of seven patients with untreated, active ocular rosacea and compare them to the tear pH values in tetracycline-treated ocular rosacea, other ocular disorders, and normal eyes.

SUBJECTS AND METHODS

The study included 44 normal, healthy volunteers, 20 patients with ocular disorders other than rosacea, seven patients with untreated, active ocular rosacea, and five patients with tetracycline-treated ocular rosacea. The total number of eyes was 142.

We used the following clinical criteria for the diagnosis of ocular rosacea: conjunctival injection with blepharitis or keratitis, or both, plus the dermatologic presence of malar erythema, telangiectasia, and hypertrophic sebaceous glands. We graded injection intensity on a scale of 0 to 3+ (0 = no redness, 1+ = mild redness, 2+ = moderate redness, and 3+ = severe redness). Patients were examined by at least two additional investigators who independently confirmed the diagnosis.

We obtained pH measurements with

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pH paper and with a microcombination glass pH probe immersed in the tear lake in the inferior cul-de-sac. We compared the two measuring techniques statistically, based on 50 pH values obtained from patients with ocular disorders other than rosacea and from normal subjects.

The data were analyzed by a mixed model analysis of variance to separate the total variation into components attributable to differences between groups, persons, and eyes. Individual *t*-tests were then performed to compare each pair of groups.[12]

Results

The mean tear pH values of the four groups tested were: normal eyes, 7.0 ± 0.20; other ocular disorders, 7.0 ± 0.18; active ocular rosacea, 8.0 ± 0.32; and tetracycline-treated ocular rosacea, 7.0 ± 0.34. The tears of patients with untreated, active ocular rosacea (Table 1) were significantly more alkaline than those of normal subjects ($P < .001$), patients with other ocular disorders ($P < .001$) (Table 2), or patients with tetracycline-treated ocular rosacea ($P < .001$).

After being treated with tetracycline (250 mg four times a day), patients showed a return to normal in lacrimal pH values as well as in clinical appearance. There was no significant difference in tear pH values between tetracycline-treated patients and normal subjects ($P > .20$).

The mean difference between the two measuring techniques was 0.07 (standard error = .029). This difference was significant ($.01 < P < .02$).

Discussion

Our study of 81 normal eyes has established the normal human tear pH range (6.5 to 7.6) and mean value (7.0 ± 0.20).[13] Only the group with active ocular rosacea demonstrated tear pH values outside the normal range. As the tear pH values of additional patients with other ocular disorders are monitored, values outside of the normal range may be found.

The significantly more alkaline tear pH of patients with untreated, active ocular rosacea may indicate the presence of some factor responsible for the irritation and ulcerative propensity of this condition. Both the clinical appearance and increased level of tear pH respond favorably to tetracycline, but the mechanism by which the drug acts to decrease tear pH values is unknown.

Both measuring techniques in this study were used in vivo; we did not transport the tears from the cul-de-sac. Carbon dioxide losses caused by tear transport were thereby avoided. Even

TABLE 1

Tear pH in untreated, active ocular rosacea

Patient No., Sex, Age (yrs)	Injection Intensity	Tear pH	
		R.E.	L.E.
1, M, 24	3+ both eyes	8.0	7.8
2, F, 49	1+ both eyes	8.0	8.0
3, M, 68	2+ both eyes	8.5	8.5
4, M, 64	3+ both eyes	8.0*	8.0*
5, M, 57	1+ R.E.; normal L.E.	8.0	7.2
6, M, 38	1+ R.E.; normal L.E.	7.8	6.4
7, F, 65	2+ both eyes	7.2	8.0

*By electrode.

TABLE 2

TEAR pH IN OTHER OCULAR DISORDERS

Clinical Diagnosis	No. of Patients	Tear pH	
		R.E.	L.E.
Staphylococcal conjunctivitis	1	7.0	7.0
Viral conjunctivitis	2	7.2	7.2
		7.1	7.2
Viral conjunctivitis with corneal erosion	1	7.0	7.4
Chemically induced conjunctivitis*	1	7.0	6.8
Iritis	2	6.6	6.6
		6.8	6.8
Corneal abrasion	1	7.0	7.0
Superior limbic keratoconjunctivitis	1	7.2	7.4
Salzmann's nodular dystrophy	1	6.9†	7.1†
Giant papillary conjunctivitis			
Soft contact lens	1	6.8	6.8
Hard contact lens	2	7.0	7.0
		7.0	7.0
Psoriasis; undergoing ultraviolet treatment	2	7.0	7.0
		7.0	6.8
Keratoconjunctivitis sicca	1	7.0	7.0
Herpetic limbitis	1	7.0	7.0
Folliculosis	1	—	7.0†
Mycosis fungoidis	1	6.8†	6.8†

*Conjunctivitis caused by chemical irritant of neutral pH.
†By electrode.

though we found a slight difference between the two techniques, the mean difference of 0.07 was small compared to differences of 1.0 unit in pH values between patients with untreated, active ocular rosacea and the other groups tested. The small difference caused by technique cannot explain the significant differences reported in this study. Indicator paper can be used as a reasonably accurate measure of tear pH values (a correction factor of +0.07 should be applied to obtain the equivalent electrode reading). We suggest the use of pH paper to confirm the diagnosis of advanced, untreated, active ocular rosacea.

SUMMARY

We compared the tear pH values of 44 normal, healthy volunteers, 20 patients with ocular disorders other than rosacea, seven patients with untreated, active ocular rosacea, and five patients with tetracycline-treated ocular rosacea. The group with untreated, active ocular rosacea had significantly more alkaline tear pH values than the other groups tested. In patients with tetracycline-treated ocular rosacea, tear pH values were not significantly different from those of normal subjects.

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NOTES, CASES AND INSTRUMENTS

PERSISTENT CORNEAL EROSION SECONDARY TO TARSAL ADRENOCHROME DEPOSIT

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The topical use of epinephrine has been associated with a variety of ocular[1,2] and systemic[3] side effects. Pigmentation resulting from epinephrine breakdown products involved the cornea,[4-9] conjunctiva,[10-13] soft contact lenses,[14,15] and prostheses.[16] Obstruction of the punctum[17] and nasal lacrimal ducts[18] has also occurred with the use of topical epinephrine. To our knowledge, this is the first reported case of persistent corneal erosion secondary to an exposed tarsal adrenochrome deposit.

CASE REPORT

A 64-year-old man with a 14-year history of medically treated open-angle glaucoma had a one-month history of intermittent foreign-body sensation in the right eye. His ocular symptoms coincided with an oral, presumably herpetic vesicle. His only ocular medication was a mixture of 1% pilocarpine and 1% epinephrine which he had applied topically four times a day for the previous 13 years.

On ocular examination, the patient had a superotemporal epithelial defect diagnosed as a herpetic ulcer. He was treated with vidarabine ophthalmic ointment, idoxuridine, gentamicin ointment, and 1% cyclopentolate HCl. Over the ensuing month the erosion persisted and the patient was referred here.

When we examined him, his best corrected visual acuity was R.E.: 6/24 (20/80) and L.E.: 6/6 (20/20). There was moderate bulbar conjunctival injection in the right eye; the left eye was uninflamed. External examination disclosed buried conjunctival adrenochrome deposits bilaterally in the inferior cul-de-sacs. Examination of the everted right upper eyelid disclosed an exposed, partially extruding, adrenochrome deposit along the temporal one third of the tarsus (Figure). Slit-lamp examination showed a superotemporal wedge-shaped corneal epithelial defect in the right eye with no surrounding edema or infiltrate. There was a moderate amount of cell and flare in the anterior chamber. Results of the rest of the examination were within normal limits.

We anesthetized the conjunctiva with a topical anesthetic and easily expressed an adrenochrome deposit measuring 3.0 × 1.5 × 1.0 mm from the superior tarsal conjunctiva. All previous medications were discontinued. We applied chloramphenicol ointment and a pressure patch to the right eye for 24 hours. The patient returned three days later; he had no symptoms and the erosion was healed. His visual acuity in the right eye had returned to 6/6 (20/20).

To identify the nature of the presumed adrenochrome deposit, we dissolved it in 1.25 M potassium hydroxide and ran it through an ultraviolet spectrophotometer. We identified two absorption peaks at approximately 28.3 nm and 29.5 nm. These peaks correspond with the absorption characteristics of an indole ring.[19] We also ran epinephrine through the ultraviolet spectrophotometer and found characteristic absorption peaks at 26 nm and 28 nm. The spectrophotometric characteristics of the presumed adrenochrome deposit strongly suggested that it was a polymer of epinephrine.

DISCUSSION

The fundamental structure of adrenochrome is an indole ring. It is an intermediate polymer in the oxidation of epinephrine to melanin. The auto-oxidation of epinephrine is accelerated by an alkaline environment as well as by copper, tyrosinase, and xanthine oxidase, all found in human tissues.[20] Ultraviolet light also hastens the formation of adrenochrome from epinephrine[20]; this may explain why the inferior conjunctiva is the most common site of adrenochrome formation.[10]

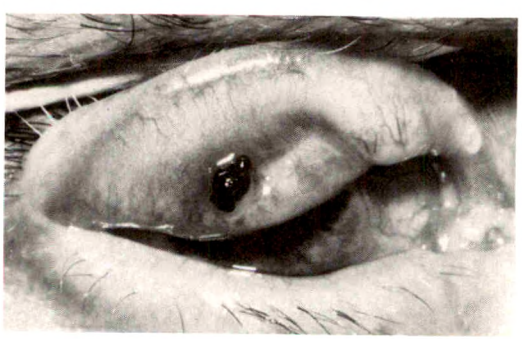


Figure (Pardos, Krachmer, and Mannis). The everted right upper eyelid shows the exposed adrenochrome deposit along the temporal one third of the tarsus.

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Most conjunctival adrenochrome deposits are encapsulated by squamous epithelium.[21] The offending adrenochrome deposit in this case was not completely covered, allowing the initial nidus of adrenochrome to increase in size with continuing direct exposure to epinephrine.

The upper tarsus is the second most common site of adrenochrome deposits.[10] In this case, its juxtaposition to the superotemporal corneal erosion and the erosion's rapid resolution after removal of the adrenochrome strongly suggests a causal relationship.

Ophthalmologists should consider the possibility of an exposed adrenochrome deposit in patients taking glaucoma medications who have a corneal foreign-body sensation or corneal erosion.

Summary

A 64-year-old man with open-angle glaucoma had used topically applied epinephrine for 13 years. He developed a foreign-body sensation and persistent superotemporal corneal erosion as a result of an adrenochrome deposit on the upper tarsus. Removal of the deposit ended the corneal erosion and returned visual acuity to normal.

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CENTRAL SEROUS RETINOPATHY IN A 7-YEAR-OLD GIRL

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Central serous retinopathy typically begins with the sudden onset of mild

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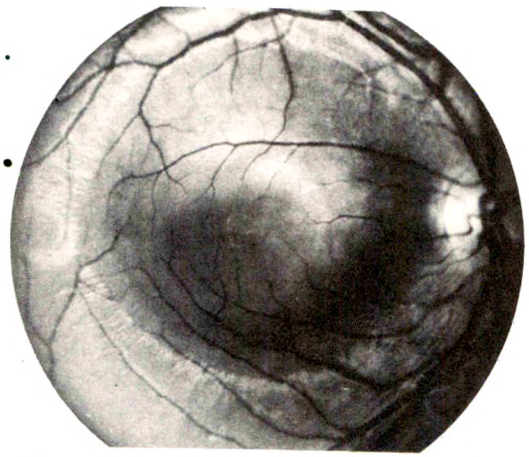


Fig. 1 (Fine and Owens). Right eye. Serous detachment of sensory retina at initial examination. Visual acuity was 3/60 (10/200).

visual loss and metamorphopsia.[1,2] Visual acuity in the affected eye may be reduced 1 or more lines on the Snellen chart, and the patient almost always sees distortion of the parallel lines of the Amsler grid. The diagnosis is based on a serous detachment of the sensory retina and confirmed with fluorescein angiography, which shows early-phase focal dye leakage and late-phase dye pooling under the sensory retina.

Resolution of the serous retinal detachment generally occurs in one to several months. Applying argon laser photocoagulation to the focal area or areas of leakage demonstrated on the angiogram may hasten resolution.[3-5] Whereas most patients with central serous retinopathy are 20 to 45 years old, we recently studied a 7-year-old girl with this condition.

Case report

A 7-year old girl had been treated for intermittent extropia since January 1978. Visual acuity had been recorded at 6/7.5 (20/25) in each eye. On Sept. 7, 1979, she complained of reduced visual acuity in the right eye. An ophthalmologist in the local emergency room found that visual acuity was R.E.: 6/60 (20/200) and L.E.: 6/6 (20/20). He also noted a macular lesion in the right eye. She was referred here.

On Sept. 11, 1979, visual acuity was R.E.: 3/60 (10/200), and L.E.: 6/6 (20/20). Neither pinhole nor refraction techniques could improve the visual acuity. The only ocular abnormality found was a serous detachment of the sensory retina, 3 disk diameters in size, in the right eye (Fig. 1). Fluorescein angiography showed many areas of early-phase focal leakage through the retinal pigment epithelium and late-phase pooling of dye under the sensory retina (Fig. 2). There was no suggestion of a choroidal neovascular membrane. The angiogram of the left macula was normal. No specific treatment was recommended.

Two weeks later, visual acuity had improved to 6/12 (20/40), and there was significant reabsorption of

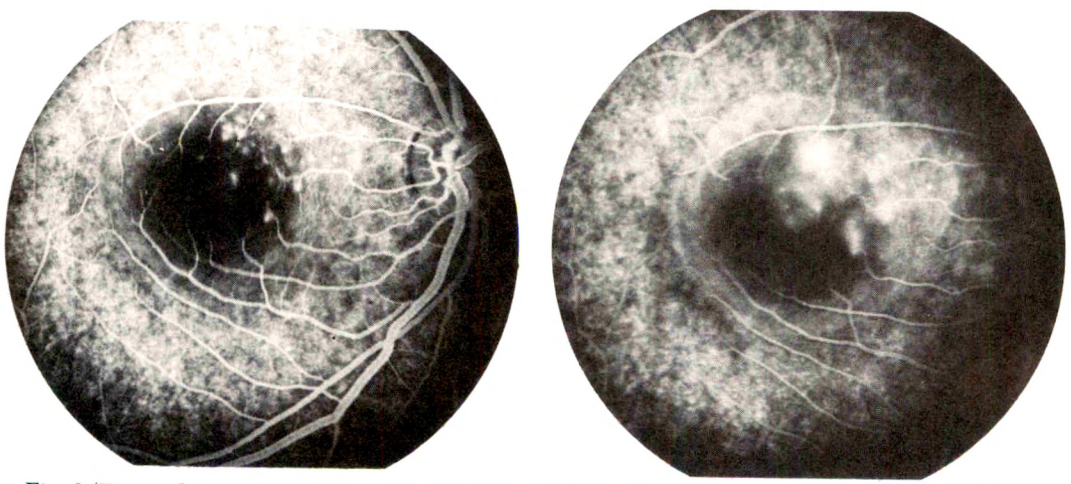


Fig. 2 (Fine and Owens). Right macula. Left, Early phase shows many areas of focal dye leakage through retinal pigment epithelium. Right, Late phase shows pooling of dye under sensory retina.

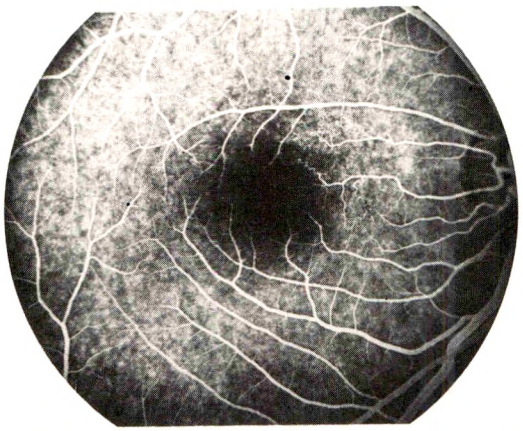


Fig. 3 (Fine and Owens). Right eye, one month after initial examination, has subtle retinal pigment epithelial transmission defects in area of previous sensory retinal detachment.

the subretinal fluid. Two weeks later (one month after the initial examination) visual acuity remained 6/12 (20/40) and no subretinal fluid was visible on biomicroscopic examination of the macula. The pigment epithelium in the macula appeared mottled, however, in the area corresponding to the previous serous detachment. Fluorescein angiography at this time showed only subtle retinal pigment epithelial transmission defects through the area of the previous sensory retinal detachment (Fig. 3). Two months after the initial examination, visual acuity had returned to 6/7.5 (20/25) and the sensory retina was flat.

Discussion

There were two unusual features to this case. The first was the patient's age. The second was the angiographic finding of many areas of focal dye leakage through the retinal pigment epithelium. Because of these unusual features, we considered the possibility of a focal choroiditis, possibly related to a pre-existing or coexisting viral infection. There was no history of a preceding upper respiratory infection or other systemic disease, and the patient was otherwise well and afebrile. Additionally, there were no other findings in either eye suggesting intraocular or extraocular inflammation. Because of the unusual features in this patient, she was examined at two-week intervals until the sensory retinal detachment completely resolved.

Although these features were unusual for a 7-year-old patient, the typical ophthalmoscopic appearance and the prompt resolution of the sensory retinal detachment led us to conclude that this was central serous retinopathy.

Summary

A 7-year-old girl experienced sudden loss of central visual acuity in one eye. Ophthalmoscopy disclosed a serous detachment of the sensory retina, and fluorescein angiography showed many areas of focal dye leakage through the pigment epithelium. Visual acuity returned to 6/7.5 (20/25), and the subretinal fluid was reabsorbed completely over the next two months. To our knowledge, this is the youngest patient with central serous retinopathy ever described.

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CENTENARY OF CREDÉ PROPHYLAXIS

On June 1, 1880, Professor Karl Siegmund Franz Credé first instilled a solution of 1.6% silver nitrate into the eyes of a newborn. During the previous five months, there had been 187 infants born at the Leipzig Lying-In Hospital. Of these, 14 developed ophthalmia neonatorum. Between June 1 and Dec. 8, 1880, there were 200 infants born; only one of these developed ophthalmia neonatorum.[1] Although Credé's method of prophylaxis of gonorrheal infections of the conjunctiva was widely confirmed, the toll of unnecessary blindness continued. In 1887, Lucien Howe prompted the American Ophthalmological Society to recommend legislative measures for obligatory Credé prophylaxis. Twenty years later, however, ophthalmia neonatorum was responsible for one quarter of the children in schools for the blind and 10% of the total blind population.[2] Eventually the National Society to Prevent Blindness was able to obtain the necessary legislation in every state.

Credé was born in Berlin in 1819 and received his schooling there. He graduated in medicine in 1842. After five years of postgraduate study, he joined the staff of the Obstetrical Clinic in Berlin and in 1850 became Privat-dozent. In 1856 he was appointed Professor of the Faculty of Medicine of the University of Leipzig and director of the university clinic and polyclinic for obstetrics and gynecology. He wrote several textbooks and monographs, and was editor of the Monatsschrift für Geburtskunde from 1853 to 1869 and of the Archiv für Gynakologie, which he founded, from 1870 to 1892.[3]

This centenary of the Credé prophylaxis is of interest because the Center for Disease Control of the Public Health Service[4] and the Committee on Drugs, Committee on Fetus and Newborn, and Committee on Infectious Disease of the American Academy of Pediatrics[5] have revised their recommendations for prevention of gonococcal ophthalmia neonatorum. The recommendations now state that "ophthalmic ointment or drops containing tetracycline or erythromycin or a

1% silver solution" are effective and acceptable. Some clinicians have theorized that silver nitrate prophylaxis immediately after delivery may impair maternal infant-bonding by reducing eye contact. Additionally, silver nitrate frequently causes a conjunctivitis that may be inaccurately implicated when a microbial infection is the cause.

The Committees of the American Academy of Pediatrics agree that the prophylactic agents should not be removed from the eye after instillation. They believe, although confirming data are not available, that delaying prophylaxis for up to one hour after birth will not affect the efficacy and should facilitate initial maternal-infant attachment. Infants born by cesarean section should also be treated. In addition to prophylaxis, infants born to mothers with clinically apparent gonorrhea should receive intravenous or intramuscular aqueous crystalline penicillin G in a single dose of 50,000 units for full-term infants or 20,000 units for low-birthweight infants.

Infants with clinical evidence of ophthalmia or complicated (disseminated) gonococcal infection should be hospitalized in isolation and managed with wound and skin precautions or secretion precautions for 24 hours after initiation of treatment with crystalline penicillin G. Two doses of 25,000 units/kg of body weight should be given intravenously each day for seven days. The eyes should be irrigated with saline. The Committee states that topical antibiotics are superfluous when systemic antibiotic therapy is given. In this, the Committee on Ophthalmia Neonatorum of the National Society to Prevent Blindness, chaired by John W. Chandler, M.D., of the University of Washington, believes that much higher concentrations of antibiotic can be provided by topical instillation of tetracycline ointment or erythromycin ointment.

The emergence of chlamydial infections as a major cause of conjunctivitis in the newborn emphasizes the value of local treatment with erythromycin or tetracycline in minimizing chlamydial conjunctival infections. Some believe, however, that the 1% silver nitrate solution properly instilled and not flushed from the eyes is prophylaxis against chlamydial infections also. Indirect evidence suggests that this is true for infants who develop extraocular chlamydial infections such as pharyngitis or pneumonitis and do not have a concurrent conjunctivitis or a history of such a conjunctivitis.

Thus, 100 years after Professor Credé's remarkable discovery, although the gonococcus is still with us, careful physicians will make certain that infants receive appropriate prophylaxis.

FRANK W. NEWELL, M.D.

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CORRESPONDENCE

Letters to the Editor must be typed double-spaced on 8½ × 11-inch bond paper, with 1½-inch margins on all four sides, and limited in length to two manuscript pages.

Orbital Carcinoid Tumor

Editor:

We read with interest the article, "Orbital carcinoid tumor metastatic from the colon" (Am. J. Ophthalmol. 89:636, 1980), by J. A. Rush, R. W. Waller, and R. J. Campbell. Other cases have been

described. Honrubia and associates[1] described a patient with bilateral orbital metastases and increased urinary levels of 5-hydroxyindoleacetic acid. Kulvin and Sawchak[2] had a case with an orbital metastasis from a bronchial carcinoid. Seven cases have been described where the tumor was metastatic to the choroid or optic nerve.[3-10] In all these cases there was either a bronchial or mediastinal primary carcinoid tumor. In five cases enucleation was undertaken or recommended,[4,6,8-11] particularly because the carcinoid tumor is reportedly resistant to radiotherapy. Although good responses to radiation therapy have been found more recently, none of the patients with functional (5-hydroxytryptophan-producing) tumors or carcinoid syndrome responded.[12] This may be because 5-hydroxytryptophan is radioprotective.[13] Even the response to radiation therapy of bone tumors secondary to carcinoid tumor occurred in a nonfunctioning tumor.[14,15]

We recently examined a woman who died of cancer. Her urinary level of 5-hydroxyindoleacetic acid was 350 mg/24 hrs. Two years previously she had had an orbital metastasis to the inferior rectus muscle from an unknown primary site. She had been treated with external beam radiation (^{60}Co, 4,000 rads in 17 doses over 34 days) with good response of proptosis and recovery of eye movements, although she unfortunately had an intraocular hemorrhage after the biopsy. When she developed carcinoid syndrome years later, histologic findings were reviewed and carcinoid tumor diagnosed. At autopsy she had an ileal primary tumor with liver, mesenteric, and bilateral breast secondary sites.

Although we do not know if her orbital secondary tumor had a high concentration of 5-hydroxytryptophan, she clearly had a 5-hydroxytryptophan-producing tumor. To our knowledge, the long delay between the appearance of an orbital secondary deposit and development of carcinoid syndrome has not been described before. Our experience, and that of Rush, Waller, and Campbell, suggests that orbital secondary deposits from carcinoid tumor should be treated with external beam radiation rather than enucleation, and that even 5-hydroxytryptophan-producing tumors can respond in extrahepatic secondary sites.

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Reply

Editor:

Drs. Harris and Montgomery relate their successful experience using radiation therapy in the treatment of an orbital carcinoid tumor. Their case differed from ours in two ways: (1) the primary tumor was in its usual site, the ileum; and (2) the symptoms of the orbital metastases antedated the discovery of the intestinal tumor, which was clinically undetected until the carcinoid syndrome developed two years later.

We noted with interest that their patient, like ours before her, responded favorably to radiation therapy directed to the orbital mestastases. Our patient is alive and well 12 months after completion of treatment. Long-term follow-up, however, is needed before successful comparison of orbital radiation therapy can be made with results of previous treatment modes cited by Drs. Harris and Montgomery. Radiation therapy for these orbital metastases is a cosmetically superior treatment and one which will, we hope, provide survival rates comparable to those of exenteration in patients who have an orbital carcinoid.

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Chloral Hydrate Sedation in Children

Editor:

We read with great interest the article, "Chloral hydrate sedation as a substitute for examination under anesthesia in pediatric ophthalmology" (Am. J. Ophthalmol. 89:560, 1980), by G. F. Judisch, S. Anderson, and W. E. Bell. We believe that certain inconsistencies should be pointed out, however.

The authors referred to excitement and delirium as side effects of chloral hydrate. Although recognized as rare side effects of chloral hydrate, excitement and delirium are more commonly seen in patients who are in stage II of general anesthesia.[1] Although the authors claimed to avoid the problems associated with a general anesthetic by heavily sedating their patients, they instead created what appears to be an inadequate and potentially dangerous level of general anesthesia.

Judisch, Anderson, and Bell used terms such as induction, awakening, and airway obstruction. These terms all imply general anesthesia. Airway problems, laryngospasm, and aspiration do not normally occur in the awake or mildly sedated patient, but can occur in the oversedated patient. All of the side effects mentioned are considered dangerous by anesthesia personnel. They are more likely to occur if stage II of general anesthesia (delirium and excitement) is prolonged, as the authors' description implied. Should a patient vomit during this critical period, airway problems in the form of obstruction or laryngospasm can occur, as well as life-threatening aspiration pneumonia.[2]

The authors did not say whether their patients fasted before being sedated, although we are certain that the authors intended to include this. Increased intragastric volumes and pressures create an increased risk of vomiting or regurgitation with excessive sedation, and life-threatening sequelae may ensue. The authors reported a 15% incidence of vomiting. They did not report temporal relationships or any radiographic confirmation of lack of complications.

Judisch, Anderson, and Bell stated that it is necessary to observe the patient who is subject to effects of the drug. We completely concur. This can be done

more safely in the controlled environment of an operating room where trained personnel can recognize and treat problems associated with sedation, and where electrocardiographic apparatus is available for monitoring. Statistical data demonstrate that the risks associated with a properly administered general anesthetic are minimal.[3]

What the authors suggest is not a substitute for general anesthesia but an unsafe general anesthetic given in an uncontrolled atmosphere. The authors exceeded the manufacturer's suggested hypnotic dose of chloral hydrate by a twofold margin.[4] Although anesthesia personnel would gladly aid in the search for a more desirable agent, we cannot sacrifice the safety of the patient. We desire not only a safe and comfortable environment for the patient, but an optimal setting for the surgeon.

We noted that this study was performed in a university setting. Our fear is that other practitioners may emulate this technique. The questionably low incidence of complications reported does not preclude catastrophic results in the future, particularly in less well controlled surroundings. We feel the authors have in this instance substituted a potentially dangerous situation for a recognized safe environment.

We urge the authors to continue their search for the ideal sedative for use in outpatient ophthalmologic procedures.

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Reply

Editor:

Before acquiring experience with chloral hydrate, we shared most of the concerns expressed by Drs. Alpert and Salazar. Consequently, most of the questions they raised were addressed directly or indirectly in our article.

The observations concerning our vocabulary seem more semantic than substantive. We are uncertain as to the exact location of the boundaries between adequate sedation, oversedation, and stage II of anesthesia. As emphasized in our report, chloral hydrate provides absolutely no analgesia, nor is it ever suggested for that purpose.

Most of the vomiting we observed occurred shortly after the ingestion of the chloral hydrate, was presumably secondary to gastric irritation, and occurred well before sedative effect of the chloral hydrate appeared. We have not had patients fast, but have found drug administration easier in infants if they are a little hungry, as opposed to recently satiated.

We are well aware of the risks of giving any drug, whether aspirin or a general anesthetic. We have continued to use chloral hydrate as described in our article. The nature and incidence of the side effects has remained unchanged in several hundred more patients.

Drs. Alpert and Salazar voice concerns which are appropriate and significant. We do wonder, though, if they have had any personal experience with chloral hydrate as outlined in our paper. Further, we wonder if they fully appreciate the large number of children who require computed axial tomographic scans, irradi-

ation therapy, bone marrow examinations, and various ophthalmologic evaluations? Few facilities have the capacity to provide anesthesia in all these instances, to say nothing of the time/expense factors. If their observations are based on conjecture, reasonable as these may be, we would encourage personal clinical evaluation of chloral hydrate, knowing that they would report their findings regarding its safety and efficacy if they were at variance with ours.

G. F. JUDISCH, M.D.
W. E. BELL, M.D.
Iowa City, Iowa

BOOK REVIEWS

Radiant Energy and the Eye. By Sidney Lerman. New York, Macmillan Publishing Co., Inc., 1980. Hardcover, 321 pages, references, index, 167 black and white figures. $45

The discipline of ophthalmology has long needed a book codifying our knowledge of the effect of radiant energy on the eye. This volume admirably fills this need and more than justifies the author's wish "to summarize and correlate our current knowledge of physical and biological elements as it relates to the known and potential hazards of radiant energy on the eye".

Unlike any body organ except the skin, the eye is constantly showered with both natural and artificial radiant energy which it either absorbs or transmits. Consequently, its tissues are admirably suited for research. The researcher seeking new basic knowledge or the clinician searching for a cure for ocular ailments can observe and study the interplay of radiant energy and biological systems. This volume contains the results of such studies, from classical studies such as Verhoeff and Bell's demonstration of thermal retinal damage from visible light to the recent work of Ham and his coworkers which incriminates visible light, particularly the blue component of the spectrum, as causing solar retinitis by photochemical rather than thermal effects.

The book is divided into six chapters. Chapter 1 discusses the basic principles of radiant energy. Chapter 2 deals with ocular anatomy, histology, physiology, and biochemistry, and Chapter 3 ultraviolet radiation and its effect on the structures of the eye. This chapter is of particular interest to clinicians because of the relationship of lens aging and nuclear cataract to ultraviolet radiation. Chapter 4 treats the effects of visible radiation on the eye and includes an excellent discussion of photocoagulation, lasers, and laser safety criteria. Chapter 5 deals with infrared, microwave, and radiofrequency radiation and the effects of electricity on the eye. The last chapter is devoted to the effects of ionizing radiation on the eye.

The book is written in a lucid, flowing style, is easily comprehended, and is completely devoid of padding. The many black and white illustrations and line drawings are excellent and the tables and diagrams are well designed. Each chapter has a summary and a comprehensive bibliography with cited references. The index is somewhat abbreviated but seems adequate. On page 4 there is an error, probably typographical, in the footnotes defining E in ergs/mm^2 rather than correctly as ergs.

This is an unusually good book which should appeal to student, researcher, and clinician not only as a reference book but also as a basic textbook. However, the book is not parochial and it should be of interest to anyone interested in the bio-

logical effects of radiant energy. The author is to be commended for a job well done.

DUPONT GUERRY III

Manual of Ocular Diagnosis and Therapy. Edited by Deborah Pavan-Langston. Boston, Little, Brown and Co., Publishers, 1980. Paperbound, 479 pages, index, 33 black and white figures. $12.95

The editor's preface says that this manual is directed to almost anyone with any interest in ophthalmology. The manual covers a wide range of clinical problems and treats them in a most effective manner. The manual does have something for everyone.

There are 15 chapters and two appendices. The writing was done by 12 contributors including the editor. Among the topics covered are the techniques of ocular examination and diagnostic tests, ocular drug toxicity, glaucoma, the cornea, pediatric ophthalmology, the retina and vitreous, cataract, trauma, the orbit, neuro-ophthalmology, strabismus, refraction, and systemic manifestations of ophthalmologic diseases. The first appendix is a formulary listing common ophthalmic drugs and their dosages. The second appendix covers antibiotics used in ophthalmology with their routes of administration and dosages.

This manual is really a primer of ophthalmology. It is written in outline form and is surprisingly complete. Although most of the book will interest only ophthalmologists, there are many items of value to any physician.

For example, the paragraphs on vitreoretinal degeneration discuss juvenile retinoschisis, the Goldmann-Favre syndrome, and Wagner's vitreoretinal degeneration and would interest only the ophthalmologist. However, the entire chapter on neuro-ophthalmology would be valuable to the family practitioner, the neurologist or neurosurgeon, and the medical student as well as to the ophthalmologist.

The book uses a few simple drawings and figures, most of which are helpful to the reader. There are no references, reflecting the fact that the book is a collection of the methods and opinions of the various contributors. However, these contributors are knowledgeable ophthalmologists and the information they impart to the reader will prove helpful in most clinical situations.

The desire of the editor to create a handbook which could serve as an aid in the evaluation of a particular patient has been reasonably well achieved. This type of manual is a difficult undertaking and one could not expect such a book to fulfill its objectives totally. Because the manual takes a systems approach rather than a signs and symptoms approach, the reader has to know where to look in order to find helpful information. Thus, when treating a patient with a red eye, the emergency room physician might have difficulty in locating the correct clinical diagnosis since there is no summary of problems which can cause a red eye. Furthermore, this commonly used clinical term is not listed in the index.

In short, Pavan-Langston and her contributors have provided a concise, easily read manual of ophthalmology. I would place it in the textbook category. As most such texts do, it contains more information than is wanted or needed by the average medical student or non-ophthalmic physician; however, it can serve as a valuable reference book for these individuals as well as an up-to-date overview of the field for ophthalmic residents and ophthalmologists.

PAUL R. LICHTER

Insight Into Eyesight. The Patient's Guide to Visual Disorders. By Paul E. Michelson. Chicago, Nelson Hall Publishers, 1980. Hardcover, 269 pages, introduction, index, 80 black and white figures. $16.95

This book gives the layman more than a basic understanding and an introduction to ocular physiology, routine examination, the more common diseases and disorders of the eye, and proper eye care.

Chapters 1 and 2 are devoted to details of the ocular specialties and the roles of the ophthalmologist, optometrist, and optician. A detailed description of the ocular examination discredits any myths that it is anything but a painless and routine procedure.

Chapter 3 discusses the various parts and functions of the eye and the reflexes which cause us "to see" as we do. Later in the book, the author explains irregularities in the shape of the eye and the corrective measures and optical aids available to improve visual acuity.

Glaucoma, cataracts, and the effect of diabetes on the eye each receive an entire chapter. The discussion of normal aging of the eye and weakening of the focusing power of the lens provides a logical sequence of what to expect in later years and stresses the importance of evaluating our individual visual requirements.

Lengthy descriptions of the conditions existing within the eye that ultimately cause loss of vision are quite clinical at times, and might have been as equally effective had they been treated more simply.

The book can help the layman ask his ophthalmologist the "right" questions about ocular problems and decide if he is being given every option available for correcting that particular problem.

Overall, this book is a valuable guide to the understanding of the eye, symptoms of ocular disorders, and the importance of ongoing ophthalmic care.

CHRISTA A. SCHNEIDER

One Pair for a Lifetime. By Richard E. Simmons and Donald A. Keller. Wesleyville, Ohio, Robin Enterprises, 1979. Hardcover, 164 pages, index, 123 black and white figures.

As a layman, I regret that this cleverly titled book was not available 20 years ago when our 18-month-old daughter had surgery for marked esotropia. I would have saved myself many qualms and my sympathetic ophthalmologist and patient husband endless questions. The beautifully clear, simple yet eloquent illustrations by Donald Keller and the conversational style of the text, written entirely in question-and-answer format by Dr. Simmons, would have given me an immediate understanding of the implications and impact of the procedures.

Among the 20 chapters included in this very practical book are the following: the parts of the eye and how it works; examination of the eye (what tests are done, when a child should be examined, the difference between an optometrist and ophthalmologist, and the like); myopia; spectacles, bifocals, and contact lenses; amblyopia; muscle imbalance; tearing; red eyes and injuries; nystagmus; faulty development; learning and visual perception problems; and habits, headaches, and other symptoms. There is a useful chapter on definitions, but I suspect that the chapter on systemic diseases and drugs is directed more toward general practitioners than parents who may be slightly confused by the terminology and syndromes.

It is obvious that Dr. Simmons' extensive experience with parents provided the questions that form the skeleton of

the text: "Does sitting too close to the TV hurt the eyes?" "Is reading in a poor light harmful to the eyes?" "What is meant by 'eye strain'?" "Are there different types of contact lenses?" "Can contact lenses harm the eyes if worn too long?" "How do glasses help straighten a crossed eye?" "Does amblyopia ever come back or recur after it has been corrected?" "Are cataracts hereditary?" "Why would a child have tearing on some days, but not on other days?" "How often should a child be tested for glaucoma?" "What happens to the premature baby's eyes after birth?" He dispels many myths and brings understanding and perception to simple and serious problems. This is not a do-it-yourself book; it emphasizes the roles of the physician and optometrist in the recognition of defects and in the treatment and care of the eyes. It drives home, loudly and firmly, the meaning of a sentence in the Foreword: "A Gift of Sight is a Gift of Life." The authors have done an excellent job of preserving the scholarly aspects of their profession, while providing clarity, simplicity, and insight into the concerns of the parents of children with ocular problems.

I hope that this book becomes readily available in pediatricians' consulting rooms, schools, and clinics where parents and advisors have to deal with the problems of the young. Medical students and residents (and especially those who lean towards general practice and pediatrics) would benefit considerably, not only from Dr. Simmons' experience but also from his approach to the parents of his patients. This book will help to prevent and alleviate the suffering and discomfort of the young patients and their parents. The useful and absorbing information it contains should relieve the anxieties of the latter, and help them to reach sensible decisions, to trust their doctors, and to promote the welfare of their children.

SHIRLEY B. SINGER

SYMPOSIA

Ultrasound in Medicine and Biology. Edited by Toshio Wagai and Ryozo Omoto. Amsterdam, Excerpta Medica, 1980. Hardcover, index, 183 black and white figures. $63.50

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TSUTSUMI, Y., AND ANDOH, Y.: Intraoperative application of the Doppler method on intracranial aneurysm. Clinical experiences and model experiment

Neuro-Ophthalmology 1980. Volume 1. Edited by S. Lessell and J. T. W. Van Dalen. Amsterdam, Excerpta Medica, 1980. Hardcover, 410 pages, introduction, index, 82 black and white figures, $78

WOLFE, R., AND BIRD, A. C.: Fundus

WRAY, S. H.: Optic neuritis

KENNERDELL, J. S.: Tumors of the optic nerve

BEHRENS, M.: Other optic nerve diseases

GLASER, J. S.: The optic chiasm

ELLENBERGER, C., JR.: The retrochiasmal visual pathways

ALEXANDER, M. P., CUMMINGS, J. D., AND ALBERT, M. D.: Higher visual functions

PASIK, T., AND PASIK, P.: Extrageniculostriate vision in primates

KEANE, J. R.: The eyelid

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Glaucoma. Contempory International Concepts. Edited by John G. Bellows. New York, Masson Publishing USA, Inc., 1980. Hardcover, introduction, index, 418 black and white figures. $49.50

WILENSKY, J. T.: **Glaucoma. The scope of the problem**

POLACK, F. M.: **Scanning electron microscopy of the trabecular meshwork in normal and glaucomatous eyes**

SEGAWA, K.: **Electron microscopic changes of the trabecular tissue in primary open angle glaucoma**

LANGHAM, M. E.: **The physiology and pathology of the intraocular pressure**

NEETENS, A.: **The vascular problem in glaucoma**

DRAEGER, J., AND JESSEN, K.: **Tonometry and tonography**

ARMALY, M. F.: **Clinical significance of optic nerve head in glaucoma**

ARMALY, M. F.: **Visual field defects and clinical perimetry in open angle glaucoma**

IWATA, K.: **Retinal nerve fiber layer, optic cupping, and visual field changes in glaucoma**

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FRIEDBURG, D., AND BISCHOF, G.: **The iris in glaucoma**

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OBSTBAUM, S. A., AND GALIN, M. A.: **Primary angle-closure glaucoma**

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SUGAR, H. S.: **Secondary glaucoma**

WOLLENSAK, J.: **Management of malignant glaucoma**

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YANOFF, M., AND EAGLE, R. C.: **Glaucoma secondary to intraocular tumors**

OLSON, R. J., ZIMMERMAN, T. J., AND KAUFMAN, H. E.: **Glaucoma associated with keratoplasty**

ZIMMERMAN, T. J., AND RAWLINGS, E. F.: **Principles of medical therapy of glaucoma—Pharmacology and therapeutics**

WETTRELL, K., AND PANDOLFI, M.: **Beta-adrenoceptor blocking agents in the management of glaucoma**

CAIRNS, J. E.: **Trabeculectomy**

DELLAPORTA, A.: **Trepano-trabeculectomy**

FRONIMOPOULOS, J., AND LAMBROU, N.: **Goniotrepanation (gotrep) with scleral flap**

HOLLWICH, F., AND BUSSEE, H.: **Surgical therapy of secondary glaucoma**

SCUDERI, G., BALESTRAZZI, E., AND RECUPERO, S. M.: **Surgical management of congenital glaucoma**

BECKMAN, H.: **Lasers in the treatment of glaucoma**

ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

American Journal of Human Genetics

GYRATE ATROPHY OF THE CHOROID AND RETINA WITH HYPERORNITHINEMIA. BIOCHEMICAL AND HISTOLOGIC STUDIES AND RESPONSE TO VITAMIN B_6. Kennaway, N. G., Weleber, R. G., and Buist, N. R. M. (Depts. Ophthalmol. and Pediatr., University of Oregon, Portland, Ore.). Am. J. Hum. Genet. 32:529, 1980.

Since 1973 more than 30 patients with gyrate atrophy of the choroid and retina associated with hyperornithinemia have been described. This autosomal recessive disease starts in the first decade at the equatorial retina and progressively involves more and more posterior retina and leads to blindness by the forth or fifth decade. There appears to be a deficient activity of ornithine ketoacid aminotransferase (OKT). Deficient activity of OKT in cultured skin fibroblasts was documented in four patients described here and levels approximately one half of normal were found in obligate heterozygotes. The patients were given vitamin B_6 which is co-factor for OKT. Three of the four patients had a significant reduction of fasting serum ornithine and increase of lysine after vitamin B_6 administration. In vitro response to vitamin B_6 was manifested by increased OKT activity at increased concentrations of pyridoxal phosphate in fibroblasts from the patients. (6 figures, 6 tables, 41 references)—David Shoch

Anesthesiology

A CONTROLLED STUDY OF THE EFFECT OF SUCCINYLCHOLINE SELF-TAMING ON INTRAOCULAR PRESSURE. Meyers, E. F., Singer, P., and Otto, A. (Depts. Anesthesiology and Ophthalmol., Washington University, St. Louis, Mo.). Anesthesiology 53:72, 1980.

It is reasonably accepted that succinylcholine causes an increase in intraocular pressure, probably by causing fasciculation of the extraocular muscles. It has been postulated that a small dose of succinylcholine given first will prevent the later increase when succinylcholine is given during the anesthesia. In this study the authors used tubocurarine in one group of patients and succinylcholine in the second group in small doses to see whether this would prevent the increase in intraocular pressure during intubation. The tubocurarine was successful and the authors found no change in the intraocular pressure from baseline in those patients pretreated with tubocurarine. However, there was a definite increase of 7 to 8 mm Hg in those patients pretreated with succinylcholine. The conclusion is that succinylcholine is not a safe drug to use in patients who are to undergo intraocular surgery. (2 tables, 10 references)—David Shoch

Annals of Neurology

DYSLEXIA. AUTOMATED DIAGNOSIS BY COMPUTERIZED CLASSIFICATION OF BRAIN ELECTRICAL ACTIVITY. Duffy, F. H., Denckla, M. B., Bartels, P. H., Sandini, G., and Kiessling, L. S. (Div. Neurophysiol., and Dept. Neurol., Children's Hosp., Boston, Mass.). Ann. Neurol. 7:421, 1980.

The authors describe a method for the diagnosis of dyslexia based upon a study of electroencephalographic and visual-evoked potential data recorded from 13 normal and 11 dyslexic boys. Measurements were made from topographic maps of brain electrical activity recorded during resting and activated testing conditions. Using a statistically based technique, the authors developed rules for classification that successfully diagnosed 80% to 90% of subjects not used in the initial rule development. The nature of the most useful measurements suggests that aberrant neuro-

physiology in dyslexia involves both hemispheres and is present at rest as well as during complex testing. The method has promise for future diagnosis and research. (3 figures, 3 tables, 23 references)—Authors' abstract

Archives of Disease in Childhood

USE OF INDOMETHACIN AND ITS RELATIONSHIP TO RETINOPATHY OF PREMATURITY IN VERY LOW BIRTHWEIGHT INFANTS. Procianoy, R. S., Garcia-Prats, J. A., Hittner, H. M., Adams, J. M., and Rudolph, A. J. (Depts. Pediatr., and Ophthalmol., Baylor College of Med., Houston, Tex.). Arch. Dis. Child. 55:362, 1980.

The prostaglandin synthetase inhibitor indomethacin has been used for closure of patent ductus arteriosus in low-birthweight infants. There is no information on its effect on other blood vessels and it has been postulated that there is an increase in the incidence of retinopathy of prematurity because of the similarity between the retinal vessels and the ductus arteriosus in their sensitivity to oxygen. To evaluate this problem, 63 preterm infants weighing less than 1,500 g who had been diagnosed as having patent ductus arteriosus were examined at the age of 4 weeks for evidence of retinopathy of prematurity. Of this group of 63 preterm infants, 15 were treated with indomethacin because of severe congestive heart failure. The authors found no difference in the incidence of retinopathy in the treated and untreated patients. Therefore, they conclude that indomethacin does not increase the incidence of retinopathy in very low birthweight infants. (1 table, 15 references)—David Shoch

Archives of Ophthalmology

COMPARATIVE OCULAR PRESSURE ELEVATION BY MEDRYSONE, FLUOROMETHOLONE, AND DEXAMETHASONE PHOSPHATE. Mindel, J. S., Tavitian, H. O., Smith, H., and Walker, E. C. (Ophthalmol. Sec. and Psychiatry Dept., Bronx Veterans Hosp., New York, N.Y.). Arch. Ophthalmol. 98:1577, 1980.

In age-, race-, and sex-matched groups, the relative abilities of medrysone, fluorometholone, and dexamethasone phosphate to increase intraocular pressure were compared in a masked manner. Compliance was controlled by drops given by nursing personnel. The 18 male hospitalized volunteers received commercial preparations of one of the corticosteroids to each eye four times a day for six weeks. The relative ability of medrysone, fluorometholone, and dexamethasone phosphate to increase intraocular pressure was one, four, and 7.6, respectively. (1 figure, 1 table, 6 references)—Authors' abstract

OCULAR LATEROPULSION. A SIGN OF LATERAL MEDULLARY DISEASE. Meyer, K. T., Baloh, R. W., Krohel, G. B., and Hepler, R. S. (Depts. Ophthalmol. and Neurol., UCLA School of Med., Los Angeles, Calif.). Arch. Ophthalmol. 98:1614, 1980.

Four patients with clinically localized lesions in the lateral medulla exhibited a tonic bias of their eyes toward the damaged side despite full extraocular movements. Each reported that his eyes were being pulled toward the involved side. Although the tonic bias was most prominent when fixation was inhibited, it also occurred with fixation and interfered with saccadic and smooth-pursuit eye movements. Saccades were hypometric when directed against the bias, whereas they were hypermetric when directed toward the side of the lesion. Smooth pursuit toward the intact side was severely impaired, whereas pursuit in the direction of the bias was normal or near normal. Lateropulsion of the eyes seems to be a unique sign of disease in the lateral medullary region of the brainstem. (1 figure, 13 references)—Authors' abstract

British Journal of Ophthalmology

THE ACUTE VITREOUS HEMORRHAGE. Lean, J. S., and Gregor, Z. (Moorfields Eye Hosp., City Road, London). Br. J. Ophthalmol. 64:469, 1980.

The authors analyzed 100 cases of acute vitreous hemorrhage in a prospective fashion.

They were able to identify the cause of the hemorrhage in about 80% of the cases. In one half of these the hemorrhage was caused by a retinal tear and only 6% were associated with diabetic retinopathy. (1 table, 6 references)—David Shoch

LATE COMPLICATIONS OF PTERYGIUM TREATMENT. Tarr, K. H., and Constable, I. J. (Univ. Dept. Ophthalmol., Royal Perth Hosp., Perth, Australia). Br. J. Ophthalmol. 64:496, 1980.

The authors report on the complications of pterygium excision and beta irradiation three to 20 years after treatment in a group of 63 eyes. Scleral ulceration was present in 51 eyes and sectorial lens opacities in 19. More serious cataracts reducing the visual acuity occurred in three eyes. Blepharoptosis, symblepharon, and iris atrophy were also seen. *Pseudomonas* endophthalmitis occurred in four patients with scleral ulceration. Beta irradiation to prevent recurrence of pterygia is a significant cause of iatrogenic ocular disease. (8 figures, 6 tables, 25 references)—David Shoch

British Medical Journal

OCULAR DEFECTS IN CHILDREN WITH CEREBRAL PALSY. Black, P. D. (Dept. Ophthalmol., RAF Hosp. Ely, Ely, Cambridge, England). Br. Med. J. 281:487, 1980.

A total of 117 children with cerebral palsy were examined for visual defects. About one-half had refractive errors but, more significantly, there was a comitant squint in 37% and a paralytic squint in 14%. Sixteen percent showed nystagmus and 10% optic nerve atrophy. The authors concluded that all children with cerebral palsy should have an ocular examination soon after the cerebral palsy is first diagnosed because of the very high incidence of ocular defects in these children. (2 figures, 5 references)—David Shoch

Cancer

ORBITAL LYMPHOID NEOPLASMS. A CLINICOPATHOLOGIC STUDY OF 60 PATIENTS. Knowles, D. M., and Jakobiec, F. A. (Depts. Pathol., and Ophthalmol., Edward S. Harkness Eye Inst., Columbia Inst., New York, N.Y.). Cancer 46:576, 1980.

Sixty cases of orbital lymphoid neoplasms originally accessioned as malignant lymphomas were analyzed histopathologically and the follow-up data evaluated separately for each histologic category. Significant clinical differences were not observed among these patients, suggesting that the lesions must be distinguished by histologic rather than clinical criteria. Two of 13 patients (15%) with benign pseudolymphomas, two of seven patients (29%) with atypical lymphoid hyperplasia, two of eight patients (25%) with well differentiated lymphocytic lymphomas, and 22 of 32 patients (68%) with less well differentiated lymphocytic lymphomas either had or later developed systemic lymphomas. Thus, the degree of cytologic differentiation appears to be the single most important factor for determining the prognosis of patients with orbital lymphoid neoplasms. (8 figures, 7 tables, 31 references)—David Shoch

International Journal of Radiation Oncology·Biology·Physics

RADIATION MANAGEMENT OF BILATERAL RETINOBLASTOMA. Gagnon, J. D., Ware, C. M., Moss, W. T., and Stevens, K. R. (Univ. Oregon Health Sci. Ctr., Portland, Ore.). Int. J. Radiation Oncology Biol. Phys. 6:669, 1980.

Thirty-four patients with retinoblastoma were treated from 1944 to 1978. Twenty-five of these patients had bilateral disease. Most patients were treated with megavoltage equipment and received doses of 5,000 rad in five weeks. According to the lifetable method, five-year survival was 67.6%; it increased to 85.7% if patients who were treated with orthovoltage equipment were excluded. Seventy-one percent of evaluable patients had useful vision at five years; an additional two patients had useful vision for two years after radiation therapy. Orthovoltage equipment should not be used to irradiate patients with retinoblastoma. (5 figures, 2 tables, 13 references)—Authors' abstract

Journal Français d'Ophtalmologie

VISUAL COMPLICATIONS OF MITRAL LEAFLET PROLAPSE. Vallat, M., Cuisinier, Y., Renaudin, D., Vallat, J. M., Dumas, M. and Robin, A. (Clinique Ophthalmol., C.H.U. Dupuytren, Limoges Cédex). J. Fr. Ophthalmol. 3:319, 1980.

In the past two years the authors have seen ten cases of prolapse of the mitral valve and two patients of these ten had purely visual symptoms. In one case there was a unilateral loss of vision with retinal edema; in the second case there was involvement of the posterior circulation with bilateral hemianopsia. The patients had no other vascular disease. Echocardiography was done before cardiac cathorization and in both cases this prolapse of mitral valve was diagnosed by this means. This was probably the origin of embolic processes in both patients. Since this is a noninvasive test the authors feel that it should be routinely employed in cases of retinal or cerebral ischemia. (3 figures, 10 references)—David Shoch

Journal of the Neurological Sciences

BINOCULAR INTERACTION IN THE VISUAL EVOKED RESPONSE. TEMPORAL FACTORS. Hoeppner, T. J. (Dept. Neurol. Sci., Rush Med. College, Chicago, Il.). J. Neurol. Sci. 47:49, 1980.

Binocular visual-evoked responses were examined in normal subjects and in patients with prolonged monocular latency. Visual-evoked responses to binocular stimulation are normally similar to the response to monocular stimulation but are somewhat larger. When conduction from one eye is slowed relative to that from its fellow (as often occurs in demyelinating disease), binocular interaction varies widely. When the interocular difference in latency is substantially increased, the binocular response is often that of the fastest pathway with little or no contribution from the slower pathway. These findings suggest a delayed reciprocal inhibition between the inputs from the two eyes which may suppress late-arriving signals. (5 figures, 24 references)—Author's abstract

Journal of Pediatric Ophthalmology and Strabismus

SYSTEMIC CORTICOSTEROIDS IN THE TREATMENT OF TRAUMATIC HYPHEMA. Rynne, M. V., and Romano, P. E. (Div. Ophthalmol., Children's Memorial Hosp., Chicago, Il.). J. Pediatr. Ophthalmol. Strabismus 17:141, 1980.

Eighteen children with traumatic hyphemas were treated with bed rest for five days except for bathroom privileges; they received no occlusion, no eyedrops, no hypotensive agents, and no tonometry. However, they were given prednisone in an oral dose of 0.6 mg/kg/day. A control group of 33 patients were treated in exactly the same fashion except that the oral prednisone was omitted. In the treated group there was only one episode of rebleeding, for an incidence of 5.5%, whereas the control group had a rebleeding rate of 33%. The difference is statistically significant. (1 table, 9 references)—David Shoch

Lancet

PENETRATION OF MERCURY FROM OPHTHALMIC PRESERVATIVES INTO THE HUMAN EYE. Winder, A. F., Sheraidah, G. A. K., Astbury, N. J., and Ruben, M. (Inst. Ophthalmol., and Moorfields Eye Hosp., London). Lancet 2:237, 1980.

Single corneas of nine patients about to undergo full-thickness corneal keratoplasty were exposed to thiomersal by application of a hydrophilic gel contact lens preserved with this mercurial agent. These lenses were removed at operation four hours later, and the lenses, corneal buttons, and samples of aqueous were taken for determination of total mercury by wet digestion and flameless atom-

ic absorption. The contact lenses contained little residual mercury, but mercury was readily detected in the corneal buttons and in the samples of aqueous, ranging from 1 to 60 ng/ml. Five control samples of aqueous obtained without exposure to mercurial preservatives contained less than 1 ng/ml of mercury. Although specific effects on ocular function are not described, the use of these preservatives is associated with penetration of mercury into the aqueous in concentrations similar to those reported with systemic poisoning by organic mercurials. (14 references)—Authors' abstract

TRILATERAL RETINOBLASTOMA. Bader, J. L., Miller, R. W., Meadows, A. T., Zimmerman, L. E., Champion, L. A. A., and Voûte, P. A. (Natl. Cancer Inst., Bethesda, Md.). Lancet 2:582, 1980.

The authors identified four children with heritable retinoblastomas who also had pinealoblastomas. There are apparently six other such cases known to the authors. All ten patients had bilateral lesions and therefore presumably had the retinoblastoma gene. It seems unlikely that the pineal lesions were metastatic since there is no known neuronal or vascular connections between the two organs. Histologically, the retinoblastomas and the pinealoblastomas appear identical. They both arise from the embryonic neuroectoderm. In amphibians and reptiles the pineal contains photoreceptor cells and the common photoreceptor origin of the pineal and retina could account for the susceptibility of both organs to the effects of the retinoblastoma gene. In animals with active photoreceptors in the pineal, the gland is described as the "third eye." Hence, the patients the authors describe may be considered to have "trilateral retinoblastoma." (5 references)—David Shoch

Neurology

OPHTHALMOPLEGIA, PTOSIS, AND MIOSIS IN TEMPORAL ARTERITIS. Dimant, J., Grob, D., and Brunner, N. G. (Dept. Med., Maimonides Med. Ctr., and State Univ. of New York, Brooklyn, N.Y.). Neurology 30:1054, 1980.

Temporal arteritis is frequently associated with fever, headaches, polymyalgia rheumatica, temporal artery tenderness, and increased erythrocyte sedimentation rate. The ocular findings consist primarily of decreased visual acuity, in some cases leading to blindness. The incidence of these visual complications varies from 5% to 57% in different series. The authors here report a series of 14 patients with biopsy-proved temporal arteritis, in seven of whom the ocular findings were primarily related to the extraocular musculature and the pupil Only one patient had diplopia. In six of the seven patients, the pattern of ophthalmoplegia did not conform to the distribution of one of the oculomotor nerves. Impairment of upward gaze was most common. In five patients the ophthalmoplegia was accompanied by blepharoptosis; in five it was accompanied by a miotic pupil. Ophthalmoplegia and blepharoptosis were the major manifestations of temporal arteritis in one patient. Oculomotor signs and symptoms responded to corticosteroid therapy in all seven patients, but in two patients prolonged therapy was necessary. Ophthalmoplegia or blepharoptosis may be early manifestations and may precede visual loss, so that frequent examination is recommended in patients suspected of temporal arteritis, with prompt administration of adequate doses of corticosteroid as soon as any ocular manifestations of temporal arteritis are noted. (4 tables, 30 references)—David Shoch

Ophthalmology

THE SCOPE OF HYALURONIC ACID AS AN EXPERIMENTAL INTRAOCULAR IMPLANT. Hultsch, E. (Dept. Retina Research, Dept. Ophthalmol., Harvard Med. School, Boston, Mass.). Ophthalmol. 87:706, 1980.

The inflammatory potential of hyaluronic acid varies from batch to batch of hyaluronic acid independent of tissue origin, concentration, or amino acid composition. The inflammatory potential can be determined by intravitreous testing only, and not by intracameral testing. Retention of hyaluronic acid in the

eye depends on molecular weight and concentration of hyaluronic acid, and not on the ocular inflammatory response. Concentration, molecular weight, and inflammatory grade of any hyaluronic acid batch used in intraocular surgery should be specified. Monitoring of the postoperative intraocular pressure is recommended. (1 figure, 4 tables, 16 references)—Author's abstract

AN EVALUATION OF ORTHOKERATOLOGY. Binder, P. S., May, C. H., and Grant, S. C. (Sect. Ophthalmol., Veterans Adm. Med. Ctr., University of California, San Diego, Calif.). Ophthalmol. 87:729, 1980.

A group of 23 patients were fitted with hard contact lenses in a manner designed to flatten the cornea. This group was compared to a similar group who wore ordinary hard lenses. In the latter group there was no change in unaided visual acuity or spherical equivalent over the course of the study. The patients fitted with an orthokeratology procedure showed significant changes in uncorrected visual acuity and spherical equivalent but not in the central or peripheral corneal curve. This study indicates that the responses to orthokeratology are unpredictable and uncontrollable. The quality of uncorrected visual acuity is worse than with contacts or spectacles, and the chances of attaining 6/12 (20/40) uncorrected visual acuity are small. Once lenses are removed, the corneal parameters return toward prefit levels. (7 figures, 7 tables, 21 references)—David Shoch

Plastic and Reconstructive Surgery

SUPERIOR OBLIQUE PARESIS AFTER BLEPHAROPLASTY. Wesley, R. E., Pollard, Z. F., and McCord, C. D. (Dept. Ophthalmol., Emory Univ., Atlanta, Ga.). Plast. Reconstr. Surg. 66:283, 1980.

A 40-year-old woman underwent bilateral blepharoplasty for dermatochalasis. Immediately after removal of the surgical drapes she complained of a torsional diplopia. In the next few weeks she developed a marked head tilt with an obvious paresis of the left superior oblique muscle. Review of the surgical notes indicated that a great deal of cautery had been done in the superior nasal area of the eyelid in the course of removing herniated orbital fat. It is presumed that the tendon of the superior oblique muscle was injured in this maneuver. A previous case indicated that a similar problem resulted from incarceration of the superior oblique tendon in a suture used to close the orbital septum. In removing herniated fat during an eyelid procedure, it is probably wise not to suture the orbital septum and to avoid excess cautery in the upper nasal quadrant of the orbit. (3 figures, 11 references)—David Shoch

Correction

In the article, "Biometry of 7,500 cataractous eyes," by Kenneth J. Hoffer (Am. J. Ophthalmol. 90:360, 1980), one line was omitted from Tables 1 and 2. In Table 1, the number of central endothelial cell counts performed was 930 for phakic eyes, 50 for aphakic eyes, and 41 for pseudophakic eyes, totaling 1,021 for the three groups. Also, the mean anterior chamber depth for aphakic eyes should be 3.67 mm rather than 3.76 mm. In Table 2, the number of central endothelial cell counts performed was 475 for hyperopic eyes, 207 for emmetropic eyes, and 339 for myopic eyes. Reprints have been corrected.

NEWS ITEMS

EDITED BY THOMAS CHALKLEY, M.D.

Send News Items double-spaced to News Editor, American Journal of Ophthalmology, Tribune Tower, Suite 1415, 435 N. Michigan Avenue, Chicago, Illinois 60611

For adequate publicity, notices of postgraduate courses, meetings, and lectures must be received at least four months before the date of occurrence.

OPHTHALMIC GENETICS: INTERNATIONAL SYMPOSIUM

An international symposium on ophthalmic genetics will take place in Jerusalem, Israel, Sept. 20–23, 1981. The symposium is sponsored by five organizations: National Foundation-March of Dimes, International Society for Genetic Eye Diseases, Israel Ophthalmological Society, Israel Academy of Sciences, and the Israel-American Ophthalmological Society. For further information, write Donald Bergsma, M.D., Dept. of Ophthalmology, School of Medicine, University of Kentucky, Lexington, KY 40536.

INTERNATIONAL VIENNA COURSE AND WORKSHOP ON STANDARDIZED OPHTHALMIC ECHOGRAPHY

A course and workshop on standardized ophthalmic echography will be held at the University of Vienna, Vienna, Austria, June 2–6, 1981. For further information, write Peter Till II, M.D., Universitats-Augenklinik, Alserstrasse 4, A-1090 Wien, Austria; or Karl C. Ossoinig, M.D., Dept. of Ophthalmology, University of Iowa, Iowa City, IA 52242.

THIRD WORLD CONGRESS OF ERGOPHTHALMOLOGY

The Third World Congress of Ergophthalmology will be held in Istanbul, Turkey, June 14–18, 1981. For further information, write Adj. Gen. Sec. Dr. Ercan Öngör, P.O. Box 29, Tesvikiye-Istanbul, Turkey.

TORONTO CONTACT LENS CONFERENCE

A comprehensive contact lens meeting, including lecture programs and workshops with an international faculty, will be held May 1 and 2, 1981, in Toronto, Canada. An added cataract and intraocular meeting will be held May 3, 1981. For further information, write Dr. Harold Stein, c/o The Park Plaza Hotel, 170 Bloor Street West, Toronto, Ontario, Canada M5S 1T9.

SCHEIE EYE INSTITUTE AND UNIVERSITY OF PENNSYLVANIA: ALUMNI OPHTHALMOLOGICAL ASSOCIATION SYMPOSIUM

A symposium, "The Eye in Systemic Disorders," sponsored by the Scheie Eye Institute and the University of Pennsylvania Alumni Ophthalmological Association, will be held in Philadelphia May 7–9, 1981. Registration is $225 for practicing ophthalmologists and $200 for University of Pennsylvania ophthalmological alumni. For further information, write David M. Kozart, M.D., Scheie Eye Institute, 51 North 39th St., Philadelphia, PA 19104.

ASPEN CORNEAL SOCIETY: FIRST ANNUAL MEETING

The First Annual Meeting of the Aspen Corneal Society will be held at Snowmass, Colorado, Feb. 1–8, 1981. Tuition is $250. For further information, write Aspen Corneal Society, Room 315-S, Francis I. Proctor Foundation, University of California, San Francisco, CA 94143.

PRESBYTERIAN HOSPITAL, NEW YORK: POSTGRADUATE COURSE IN NEURORADIOLOGY

A Postgraduate Course in Neuroradiology and Radiology of the Orbit, Head, Neck, and Spine will be held May 9–13, 1981, at the Essex House in New York City. The fee is $360. A course on head and neck radiology will be held May 9 and 10; the fee is $160. A course on neuroradiology will be held May 11–13, 1981; the fee is $240. For further information, write Sadek K. Hilal, M.D., Director of Neuroradiology, Neurological Institute, Box 63, 710 W. 168th St., New York, NY 10032.

Third Annual Strabismus Workshop

The Third Annual Strabismus Workshop will be held in Steamboat Springs, Colorado, March 7–14, 1981. For further information, write Western Colorado Ophthalmological Society, 790 Wellington, Grand Junction, CO 81501.

Mayo Clinic: Continuing Education Program—Ophthalmic Reviews

The continuing education program, "Ophthalmic Reviews," will be held May 8 and 9, 1981, at the Mayo Clinic. The topic will be "Glaucoma syndromes." For further information, write Mr. Carl Rider, Mayo Clinic, Rochester, MN 55901.

Joint Meeting of American Society of Contemporary Ophthalmology and Fifth International Glaucoma Congress

A joint meeting of the American Society of Contemporary Ophthalmology and the Fifth International Glaucoma Congress will be held March 15–21, 1981, in Orlando, Florida. For further information write, John G. Bellows, M.D., 6 N. Michigan Ave., Chicago, IL 60602.

National Eye Institute and Epistat Associates: Course on Clinical Vision Research

A course on Clinical Vision Research: Epidemiologic and Biostatistical Approaches will be presented by the National Eye Institute and Epistat Associates, April 23–25, 1981, in Sarasota, Florida. For further information, write Ms. Catherine M. Beinhauer, Conference Consultant, Epistat Associates, Box 214, Norwich, VT 05055.

Annual Iowa Eye Association Meeting

The Annual Iowa Eye Association Meeting will be held May 7–9, 1981, in Iowa City, Iowa. For further information, write Thomas A. Weingeist, M.D., Dept. of Ophthalmology, University Hospitals and Clinics, Iowa City, IA 52242.

Modern Concepts in Refraction: Corneal Contact Lenses and Refractive Keratoplasty Seminar

A seminar on Modern Concepts in Refraction: Corneal Contact Lenses and Refractive Keratoplasty will be held in San Francisco, California, May 8 and 9, 1981. For further information, write Ms. Jacquie Williams, Education Committee, Narcissus Medical Foundation, 1800 Sullivan Ave., Suite 506, Daly City, CA 94015.

Georgetown University Center for Sight: Training Program

The Georgetown University Center for Sight is offering a two-year training program of ophthalmic technicians and technologists, June 29, 1981, to June 30, 1983. The tuition is $1,800 per year, The application deadline is May 1, 1981. For further information, write Phyllis L. Finebert, C.O.T., Director, Ophthalmic Medical Assistant Program, Georgetown University Center for Sight, 3800 Reservoir Rd., N.W., Washington, DC 20007.

Strabismus Update 1981

The 1981 Western/Mid-Western regional meeting of the American Association of Certified Orthoptists will be held in Boulder, Colorado, April 2–4, 1981. For further information, write The Center for Conferences and Management/Technical Programs, 970 Aurora, Boulder, CO 80302.

Intraocular Lens Implantation Courses

Professor Jan Worst will conduct two-day instructional courses on intraocular lens implantation April 12 and 13, June 14 and 15, and Oct. 11 and 12, 1981, in Haren, Holland. For further information, write Mrs. Josine L. van der Valk, Secretary, Julianalaan 11, 9751 BM Haren, Holland.

Albany Medical College: Seminar/Teaching Days

Albany Medical College, Department of Ophthalmology, is sponsoring postgraduate teaching days on the first Thursday of each month beginning in January 1981 and ending in June. For further information, write Laurie Hauser, Dept. of Ophthalmology, Albany Medical College, Albany, NY 12208.

Pittsburgh Ophthalmology Society and University of Pittsburgh School of Medicine: 17th Annual Spring Meeting

The Pittsburgh Ophthalmology Society and the Department of Ophthalmology, University of Pittsburgh School of Medicine, will present the 17th Annual Spring Meeting March 27 and 28, 1981. The guest of honor, A. Edward Maumenee, will present the First Annual Harvey E. Thorpe Lecture. There will be an all-day symposium for nurses and allied ophthalmic personnel on Saturday, March 28. For further information, write Frank Cignetti, M.D., 2545 Mosside Blvd., Monroeville, PA 15146.

Medical College of Georgia-Department of Ophthalmology

The Department of Ophthalmology at the Medical College of Georgia is sponsoring a three-day continuing education program entitled "Selected Topics in Pediatric Ophthalmology," in Savannah, Georgia, March 23–25, 1981. For further information, write Division of Continuing Education, Medical College of Georgia, Augusta, GA 30912.

Utah Ophthalmological Society: Annual Winter Meeting

The Utah Ophthalmological Society will hold its annual winter meeting, Feb. 22–27, 1981, in Park City, Utah. For further information, write A. Thomas Williams, M.D., Utah Ophthalmological Society, 715 East 3900 South, #205, Salt Lake City, UT 84107.

Tulane Medical Center: Vitrectomy Workshop

Tulane Medical Center will hold a vitrectomy workshop, March 27 and 28, 1981. The registration fee is $450. For further information, write Office of Continuing Medical Education, Tulane Medical Center, 1430 Tulane Ave., New Orleans, LA 70112.

The New York Branch of the Orton Society, Inc.: Eighth Annual Conference

The New York Branch of the Orton Society, Inc. will present its Eighth Annual Conference, "The Challenge of the 80s: Effective Education for the Language and Learning Disabled," March 26–28, 1981, at the New Grand Hyatt Hotel in New York City. For further information, write New York Branch of the Orton Society, Inc., 80 Fifth Ave., New York, NY 10011.

Oregon Academy of Ophthalmology: 40th Postgraduate Convention

The Oregon Academy of Ophthalmology will sponsor the 40th Postgraduate Convention, March 5–7, 1981, in Portland, Oregon. For further information, write Ms. Joan Buck, Oregon Academy of Ophthalmology, Executive Office, P.O. Box 12945, Salem, OR 97309.

Association of University Professors Announces New Appointments

The Association of University Professors of Ophthalmology announced the following appointments: Peter R. Egbert, Acting Head, Division of Ophthalmology, Stanford University Medical Center; James P. Ganley, Chairman, Louisiana State University Medical Center; Jean E. Holt, Assistant Professor and Acting Chairman, Department of Ophthalmology, University of Texas Health Science Center; Theodore Lawwill, Professor and Chairman, Department of Ophthalmology, University of Kansas Medical Center; and George W. Weinstein, Professor and Chairman, Department of Ophthalmology, West Virginia University Medical Center.

Personals

Robert Machemer

Robert Machemer received the 1980 Award of Merit in Retina Research from the Retina Research Foundation in recognition of his many accomplishments in the basic studies leading to the introduction of the pars plana vitrectomy. The award was made Oct. 10, 1980.

Frank W. Newell

Frank W. Newell presented the Fourth James E. McDonald Lecture, Oct. 1, 1980, at Loyola Medical Center, Maywood, Illinois. The topic of his lecture was "Ocular changes in lysosomal disorders."

Alfred Sommer

Alfred Sommer was honored Oct. 14, 1980, by Helen Keller International for his outstanding contributions to the prevention of childhood blindness.

The special award to Dr. Sommer, which was made in observance of the 100th anniversary of the birth of Helen Keller this year, was presented in Jakarta, Indonesia, by Susan T. Pettiss, Director of Blindness Prevention of Helen Keller International.

Children's Eye Care Foundation: Research Grants, Fellowship Training Grants, and Institutional Training Grants, 1980–1981

The Children's Eye Care Foundation of Washington, D.C., which established a Research Support Program in 1975 to promote and encourage research in pediatric ophthalmology, has announced the recipients of its 1980 grant awards. The Program will aid the following four new research projects, with grants aggregating $20,000:

Project Title	Chief Investigator
1. Technique for Screening Visual Resolution in 6-Month to 3-Year-Old Children	Velma Dobson, Ph.D. University of Washington
2. Long-Term Effects of Strabismus Procedures on Anterior Segment Physiology	Lloyd M. Wilcox, Jr., M.D. New England Medical Center Hospital
3. Quantitative Measurement of Ocular Motility for Strabismus Management	Henry S. Metz, M.D. University of Rochester, School of Medicine and Dentistry
4. Anatomical Study of Human Retinal Development	Richard M. Robb, M.D. The Children's Hospital Medical Center

In addition, the Foundation hopes to promote better eye care for young children throughout North America via its fellowship support program. This year the Foundation has awarded a total of $30,000 in stipends to the following fellows receiving post-residency training in pediatric ophthalmology or strabismus: Jane D. Kivlin, M.D., University of Miami School of Medicine; Rebecca J. Adams, M.D., Wills Eye Hospital; John W. Simon, M.D., Wills Eye Hospital; Robert W. Lingua, M.D., Children's Hospital of Los Angeles; and Vincent J. Sutton, M.D., University of Iowa Hospitals and Clinics.

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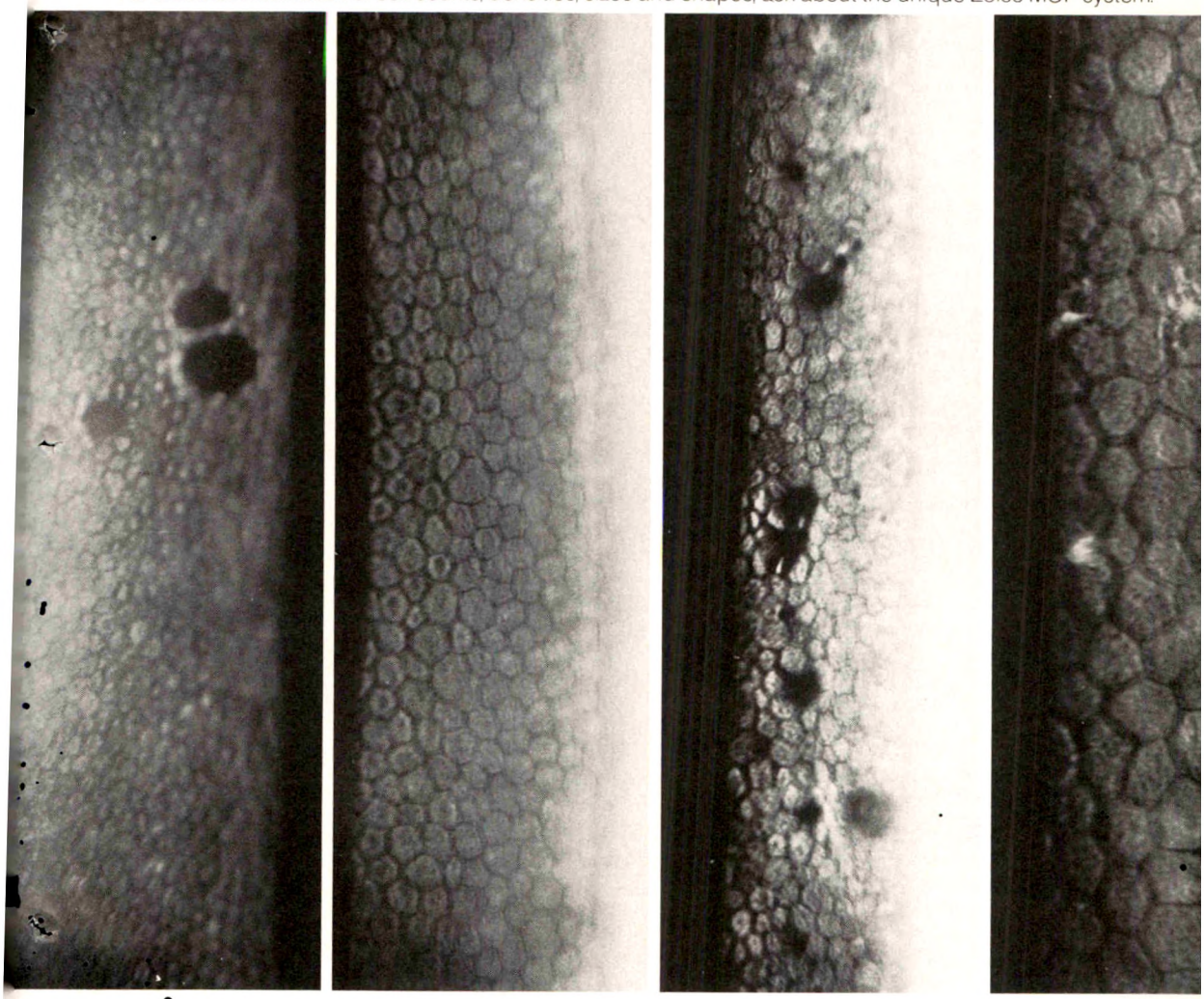
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Joseph Baldone, M.D., New Orleans, LA.
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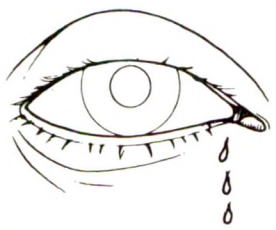
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American Journal of Ophthalmology	0 0 0 2 9 3 9 4	Sept. 25, 1980
3. FREQUENCY OF ISSUE	A. NO. OF ISSUES PUBLISHED ANNUALLY	B. ANNUAL SUBSCRIPTION PRICE
Monthly	12	$21.00

4. LOCATION OF KNOWN OFFICE OF PUBLICATION (Street, City, County, State and ZIP Code) (Not printers)
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5. LOCATION OF THE HEADQUARTERS OR GENERAL BUSINESS OFFICES OF THE PUBLISHERS (Not printers)
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6. NAMES AND COMPLETE ADDRESSES OF PUBLISHER, EDITOR, AND MANAGING EDITOR

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Ophthalmic Publishing Company, 435 N. Michigan Ave., Suite 1415, Chicago IL 60611

EDITOR (Name and Address)
Frank W. Newell, M.D., 435 N. Michigan Ave., Suite 1415, Chicago IL 60611

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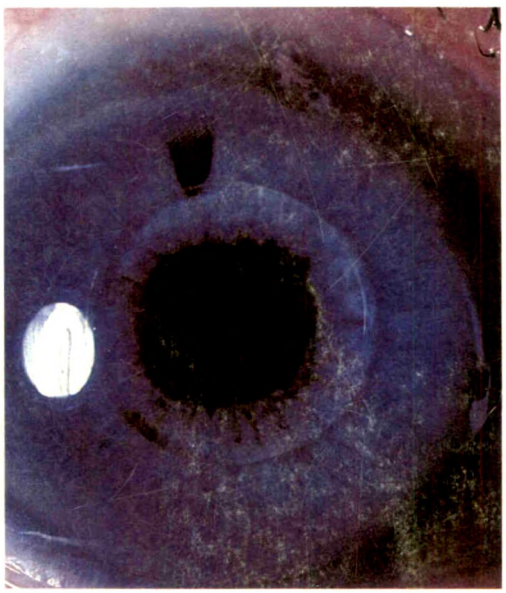
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1. Nelson, EL. Ophthalmic Steroids in Ocular Anti-Inflammatory Therapy. HB Kaufman, Editor, Springfield, ILL. Thomas, 1970.
2. Hull, FW, Jr. Epstein, WA. Comparison of the intraocular pressure effects of topical dexamethasone and fluorometholone. Allergan Report Series No. 74, 1972.

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